Principles and Practice of Stress Management

FOURTH EDITION

edited by Paul M. Lehrer
Robert L. Woolfolk

Guilford Press
e-book

PRINCIPLES AND PRACTICE OF STRESS MANAGEMENT

Also Available

The Value of Psychotherapy:
The Talking Cure in an Age of Clinical Science
Robert L. Woolfolk

Treating Somatization:
A Cognitive-Behavioral Approach
Robert L. Woolfolk and Lesley A. Allen

Principles and Practice of Stress Management

FOURTH EDITION

Edited by
Paul M. Lehrer
Robert L. Woolfolk

Foreword by Omer Van den Bergh

THE GUILFORD PRESS
New York London

A Division of Guilford Publications, Inc.
370 Seventh Avenue, Suite 1200, New York, NY 10001
www.guilford.com

Printed in the United States of America

This book is printed on acid-free paper.

Last digit is print number: 9 8 7 6 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data

Names: Lehrer, Paul M., editor. | Woolfolk, Robert L., editor.
Title: Principles and practice of stress management / edited by Paul M. Lehrer, Robert L. Woolfolk; foreword by Omer Van den Bergh.
Description: Fourth edition. | New York : The Guilford Press, 2021. | Includes bibliographical references and index.
Identifiers: LCCN 2020053567 | ISBN 9781462545100 (paperback) | ISBN 9781462545117 (hardcover)
Subjects: LCSH: Stress management. | Stress (Psychology)—Prevention. | Stress (Psychology)—Treatment.
Classification: LCC RA785 .P75 2021 | DDC 155.9/042—dc23
LC record available at *https://lccn.loc.gov/2020053567*

About the Editors

Paul M. Lehrer, PhD, BCB, is Professor Emeritus of Psychiatry at Rutgers Robert Wood Johnson Medical School. Dr. Lehrer's research interests include the mechanisms and applications of heart rate variability biofeedback; mind–body medicine; respiratory psychophysiology; and the clinical and physiological effects of relaxation therapies. His publications include 200 journal articles and book chapters. He is Associate Editor of the journal *Applied Psychophysiology and Biofeedback*, an Honorary Fellow of the Association for Applied Psychophysiology and Biofeedback, and a Fellow of the American Psychological Association and the Association for Behavioral and Cognitive Therapies. Dr. Lehrer is a recipient of the Lifetime Achievement Award, Outstanding Service Award, and Distinguished Scientist Award from the Association for Applied Psychophysiology and Biofeedback.

Robert L. Woolfolk, PhD, is Professor of Psychology and Philosophy at Rutgers, The State University of New Jersey. He has served on the faculties of Princeton University and the University of Texas at Austin. His research interests include psychopathology and psychotherapy, the philosophical foundations of psychology, and the treatment of somatoform disorders, and he has published widely in these areas. Dr. Woolfolk has maintained an active clinical practice since the 1970s. Drawing from a variety of therapeutic approaches, he has conducted therapy with individuals, couples, families, and groups. He is a past editor of the *Journal of Theoretical and Philosophical Psychology*; the coauthor of *Stress, Sanity, and Survival* and *Treating Somatization: A Cognitive-Behavioral Approach*; and the author of *The Cure of Souls* and *The Value of Psychotherapy*.

Contributors

Acharya Balkrishna, Patanjali Research Foundation, Haridwar, India

Patricia Carrington, PhD (deceased), Department of Psychiatry, University of Medicine and Dentistry of New Jersey–Robert Wood Johnson Medical School, Piscataway, New Jersey

Kevin W Chen, PhD, MPH, Faculty of Physical Education, Shenzhen University, Shenzhen, China

Jonathan S. Comer, PhD, Department of Psychology and Center for Children and Families, Florida International University, Miami, Florida

Laura Nelson Darling, MA, Center for Anxiety and Related Disorders, Boston University, Boston, Massachusetts

Cristina del Busto, PhD, Department of Psychology, Florida International University, Miami, Florida

Richard N. Gevirtz, PhD, California School of Professional Psychology, Alliant International University, San Diego, California

Ram Kumar Gupta, MSc, Patanjali Research Foundation, Haridwar, India

Ingrid Hammarlund, MA, Department of Music Education, Royal College of Music, Stockholm, Sweden

Niranjan Kala, MA, Patanjali Research Foundation, Haridwar, India

Robert A. Karlin, PhD, Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, New Jersey

Jean L. Kristeller, PhD, Department of Psychology, Indiana State University, Terre Haute, Indiana

Alexander W. Kusnecov, PhD, Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, New Jersey

Deany Laliotis, LICSW, The Center for Excellence in EMDR Therapy, Inc., Washington, DC

Robert L. Leahy, PhD, American Institute for Cognitive Therapy, New York, New York

Paul M. Lehrer, PhD (emeritus), Department of Psychiatry, Rutgers Robert Wood Johnson Medical School, Piscataway, New Jersey

Wolfgang Linden, PhD, Department of Psychology, University of British Columbia, Vancouver, British Columbia, Canada

F. J. McGuigan, PhD (deceased), Institute for Stress Management, U.S. International University, San Diego, California

Alicia E. Meuret, PhD, Department of Psychology, Southern Methodist University, Dallas, Texas

Erica D. Musser, PhD, Department of Psychology and Center for Children and Families, Florida International University, Miami, Florida

Marialaina Nissenbaum, MA, Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, New Jersey

Sara Norton, PhD, Department of Psychology, Washington University, St. Louis, Missouri

Cristy Phillips, PT, EdD, Department of Physical Therapy, Arkansas State University, Jonesboro, Arkansas

Donna B. Pincus, PhD, Center for Anxiety and Related Disorders, Boston University, Boston, Massachusetts

Thomas Ritz, PhD, Department of Psychology, Southern Methodist University, Dallas, Texas

Francine Shapiro, PhD (deceased), EMDR Institute, Watsonville, California; founder and originator of EMDR therapy

Wesley E. Sime, PhD, Department of Health and Human Performance, University of Nebraska–Lincoln, Lincoln, Nebraska

Jonathan C. Smith, PhD, Department of Psychology, Roosevelt University, Chicago, Illinois

Roger Solomon, PhD, EMDR Institute, Arlington, Virginia

Shirley Telles, PhD, Patanjali Research Foundation, Haridwar, India

Töres Theorell, MD, PhD (emeritus), Department of International Health, Karolinska Institute; Department of Music Education, Royal College of Music; and Department of Psychology, University of Stockholm, Stockholm, Sweden

Lynda Thompson, PhD, ADD Centre and Biofeedback Institute of Toronto, Toronto, Ontario, Canada

Michael Thompson, MD, ADD Centre and Biofeedback Institute of Toronto, Toronto, Ontario, Canada

Jan van Dixhoorn, MD, PhD, The Center for Breathing Therapy, Amersfoort, The Netherlands; Kennemer Hospital, Haarlem, The Netherlands

Scott Woodruff, PhD, American Institute for Cognitive Therapy, New York, New York

Robert L. Woolfolk, PhD, Department of Psychology, Rutgers, The State University of New Jersey, Piscataway, New Jersey

Foreword

There are few scientific words that have made their way into the language of everyday life with a stronger appeal to laypeople and the media than the word *stress*. In this respect, it is ironic that few scientific words were more ambiguous from the beginning. It entered science through the world of engineering, where it referred to an external force imposed on an object, whereas in medicine it was used only occasionally and in a metaphorical way by Walter Cannon, physiologist and founding father of stress research in the beginning of the 20th century (Cannon, 1914). When Hans Selye, the other founding father, introduced his general adaptation syndrome in 1936 in his "Letter to the Editor" in *Nature* and in further elaborations (Selye, 1950), *stress* gradually became a word that indicated a response of a challenged body. However, when he investigated the effects of, for example, large doses of insulin, it remained unclear whether the term *stress* referred to a cause or to an effect. His former PhD students Szabo, Tache, and Somogyi (2012) describe how, in the 1940s, Hans Selye remained greatly concerned by criticisms about this, leading him to distinguish between *stressor* and *stress*. They also describe that, at the end of his life, he "complained that people used the term 'stress' almost indiscriminately, commenting that 'it was hard to get acceptance in the 1940s—now it is almost a household term' " (Szabo et al., 2012, p. 475). Now, four decades later (Selye died in 1982), *stress is* a household term. There is work stress, marital stress, parenting stress, caregiver stress, environmental stress, posttraumatic stress, choice stress, vacation stress, eustress and distress, and so on. Everybody has experienced some form of stress, intuitively "knows" what it is, and believes that it is generally bad for one's health. As a result, stress management trainings of all kinds and forms are abundantly offered by trainers with different backgrounds and often inspired by idiosyncratic beliefs.

The importance of the fourth edition of *Principles and Practice of Stress Management*, edited by Paul Lehrer and Robert Woolfolk, can hardly be overestimated in this context. There is a strong need to close the gap between, on the one hand, specialized cutting-edge research on stress and, on the other hand, solid, comprehensive, up-to-date knowledge and its practical implications for everyday life and health care. Lehrer and Woolfolk's book does exactly that. Basic and applied research on stress has generated and still does generate new empirical data at a tremendous pace, requiring an update of its implications for evidence-based stress management at least every decade. After the first edition of this book in 1984, a second in 1993, and a third in 2007, this fourth edition in 2021 was urgently needed. I am writing this foreword only a few days after receiving the obituary, issued by Rockefeller University, of Bruce S. McEwen, who died on January 2, 2020. It appears that McEwen's career is deeply intertwined with much of the progress in the field during the last decades. One of his latest papers (McEwen, 2018) eloquently summarizes much of this progress. The concept of allostatic load, first coined in 1993, has become the dominant integrative framework for guiding stress-related research and interpreting how it affects health. Groundbreaking findings have, for example, demonstrated the impact of repeated or chronic stressful experiences on epigenetic mechanisms and how they remodel neural connections in the brain. They have shown how early adverse experiences may permanently alter the brain and neuroendocrine interactions of a developing child and how stress-related lifestyles and environmental factors mediate between social inequality and health disparity through a cascade of mechanisms at cellular and organ-specific levels.

If anything, the last two decades have witnessed growing evidence showing how stress creeps into the body through a multitude of mechanisms at the level of genetics, neurobiology, immunology, endocrinology, and gastroenterology in interaction with behavioral and social factors, illustrating that stress refers to a domain of study that is interdisciplinary par excellence. It should be noted in passing that the *load* part in *allostatic load* emphasizes the temporal rather than the intensity dimension as the toxic element of stress. This means that there is an interesting potential discrepancy between what *feels* bad and what *is* bad. The toughness model (Dienstbier, 2014) and findings by Seery and colleagues (Seery, Leo, Lupien, Kondrak, & Almonte, 2013) are examples suggesting that intermittent activation of stress responses (that may feel bad) may promote mental and somatic health and well-being (that may actually be good). Overall, stress research in the last decade has further matured and revealed ever more complexities, making the challenge to edit a book on stress and stress management for students, professionals, and the stress management "consumer" bigger with every new edition.

This fourth edition of *Principles and Practice of Stress Management* critically discusses individual ways to reduce stress and to improve hardiness and resilience. The five opening chapters in Part I lay the ground for 16 chapters in the second part that present a wide array of techniques and their implied cognitive, affective, and psychophysiological mechanisms. These first five chapters reveal the position of the editors on conceptual issues about stress, on the importance of the societal and cultural context, and on the balance between the needs for both an empirical, evidence-based approach and a clinical, idiographic one. In addition, they place stress and its management in a broader picture. Lehrer (Chapter 2) discusses stress from a cybernetic system regulation perspective and describes its management as a form of preventing and controlling dysregulation and improving allostatic capacity. Smith (Chapter 3) presents psychological relaxation theory as a comprehensive way of viewing a diverse array of approaches to self-relaxation, meditation, and mindfulness. Kusnecov, Norton, and Nissenbaum (Chapter 4) provide the neuroscience framework for understanding stress, describing brain–body interactions involving neural, immunological, and endocrine mechanisms. A final chapter (Comer, Darling, del Busto, Musser, & Pincus, Chapter 5) specifically focuses on stress in children and adolescents.

Part II describes the state of the art of a large array of different methods to handle stress and improve resilience, grouped into psychophysiological (seven chapters), meditative (four chapters), psychotherapeutic (one chapter), and hypnotic (two chapters) methods. Two other chapters focus on exercise and music. Each of the chapters is written to meet the needs of the clinician. Besides historical and/or theoretical foundations, sufficient information describes how the technique should be applied, for which kinds of conditions, and, if available, which results can be expected. Often, chapters include case material.

The broad range of methods covered in juxtaposition may leave the impression of the dodo bird's verdict in *Alice's Adventures in Wonderland,* well known in psychotherapy research: "Everybody has won and all must have prizes." However, the integrative chapter on psychological relaxation theory (Smith, Chapter 3), discussing relaxation, meditation, and mindfulness, "presents a map of an empirically derived set of states not based on any particular tradition. Such a universal lexicon invites us to look beyond our islands of favored techniques and consider the larger world of relaxation, meditation, and mindfulness" (p. 55). The volume's final chapter (Chapter 22), on clinical considerations, such as the match between the patient's expectations/preferences and the characteristics of the method, is very helpful in guiding the clinician to select a specific method for a specific problem.

This book makes a clear choice: It addresses methods that focus on individual stress responses and how to manage them. Thus it excludes other factors that may be of paramount importance as well for stress management, namely the capacity to assess and manage the stimuli and the environmental conditions that cause individual stress. Nor does it pay much attention to societal stress. On the other hand, most clinicians see patients who suffer and often have little control over the stressful stimuli and the contextual factors. In addition, individuals often want to develop skills to prevent stress and stress-related risk factors, and/or they may need to first regain strength and develop resilience before they are able to influence and change detrimental environmental conditions. This book is thus an invaluable source of scientific and practical knowledge for every student and professional interested or involved in mental health care; it is also for the stress management consumer. And aren't we all consumers at some moment in life?

Omer Van den Bergh, PhD
University of Leuven, Belgium

REFERENCES

Cannon, W. B. (1914). The emergency function of the adrenal medulla in pain and the major emotions. *American Journal of Physiology–Legacy Content*, *33*(2), 356–372.

Dienstbier, R. (2014). *Building resistance to stress and aging: The toughness model.* Cham, Switzerland: Springer.

McEwen, B. S. (2018). Redefining neuroendocrinology: Epigenetics of brain–body communication over the life course. *Frontiers in Neuroendocrinology, 49*, 8–30.

Seery, M. D., Leo, R. J., Lupien, S. P., Kondrak, C. L., & Almonte, J. L. (2013). An upside to adversity? Moderate cumulative lifetime adversity is associated with resilient responses in the face of controlled stressors. *Psychological Science, 24*(7), 1181–1189.

Selye H. (1950). *The physiology and pathology of exposure to stress, a treatise based on the concepts of the general-adaptation syndrome and the diseases of adaptation.* Montreal: ACTA.

Szabo, S., Tache, Y., & Somogyi, A. (2012). The legacy of Hans Selye and the origins of stress research: A retrospective 75 years after his landmark brief "letter" to the editor of *Nature*. *Stress*, *15*(5), 472–478.

Preface

Although we have written this book primarily for students, trainees, and professionals interested in learning about empirically supported stress management methods, we have found that previous editions of our book have also been read and appreciated by stress management consumers. Indeed, we all should consider ourselves to be stress management consumers, because stress is an inescapable component of modern life for all of us. We personally use most of the methods described here. We chose these methods on the basis of our knowledge of empirical research methods and invited the most authoritative experts we knew to author each of the chapters—people who, in addition to having great expertise in particular areas, are both consummate clinicians and well-respected researchers.

All of us are or should be concerned about whether the approaches we use for managing stress really work; hence our own reliance on empirically supported methods. We applied that standard to choosing chapters for this book. We would *not* argue that methods not in this book are ineffective, but only that we are not aware of controlled studies demonstrating their effects. Indeed, there are many stress management methods that we have decided not to include in the book that may be effective but that have simply not been evaluated in controlled research.

Although this criterion may sound formidable, it is actually less rigorous than it seems. Many control conditions used in clinical trials are simply "treatment as usual." This kind of research design obviously does not control for elements of the placebo effect, including expectancies, or the relationship with a supportive therapist or health care institution. Also, statistical findings of efficacy say little about the effects of a technique on a particular individual. Furthermore, we have found little statistical evidence for differential beneficial effects of the various methods we review here. People have individual preferences based on a number of factors, including personality, culture, and target symptomatology. Probably all of these factors contribute to effectiveness. Perhaps the most important factor is the personal appeal of each method. This contributes to regular adherence to the method, and any method that is unused is ipso facto ineffective. Thus, although we use science to increase the chances of our selecting a method that works, the practitioner or consumer using these methods is really an artist, choosing the methods that best fit an incalculably broad range of needs and preferences.

That said, why is it necessary to provide a new edition of a book about methods, most of which have been available for many years? In previous editions, as in the current one, we have invited chapters from the foremost clinicians and researchers in the world who are identified with each method. However, research in this area has exploded in the past decade, and several new methods have appeared. All chapters on particular methods now include a list of applications with significant findings from controlled clinical trials, which were much less plentiful for previous editions of this book. A brief summary of this research is now included. A new chapter has been added on end-tidal carbon dioxide biofeedback, an important innovation given the widespread symptomatology caused by hyperventilation and dysfunctional breathing. Also, more is known about the role of inflammation in stress symptoms and about the interplay among exercise, the autonomic nervous system, immunity, inflammation, cognitions, and stress. The neuroscience of stress and stress management is also now better understood, and several chapters include detailed expositions on the neural and neuroendocrine mechanisms of stress and neural underpinnings of stress management methods. There also has been a great increase in number and quality of empirical validation studies of all the methods described in this book, including pathways by which they work and clinical efficacy. The chapters in this new edition reflect that deepened evidence base.

Indeed, the role that all of the methods described in this book play in health care and psychological well-being is now incontrovertible. On the other hand, although we are more confident about the general usefulness of these methods than we were a decade ago, we are somewhat less confident about specific targeted effects of the various methods for treating particular problems. Although a fine-grained evaluation of relative effect sizes for particular problems might be a worthwhile undertaking, there is tremendous evidence for overlapping effects. This is not to say that the various methods are equivalent. Many people prefer one method over another, find some more pleasant and easier to learn than others, or are sensitive to the particular side effects of some methods. Side effects, as well as beneficial effects, differ among the methods, and particular skills, problems, or constitutions may make some of the methods easier to learn and more beneficial than others. The relative appeal of various methods among various cultural groups and personality types remains mostly unexplored but may play a large role in the willingness of people to practice and use a particular method. These issues are discussed in detail in Chapter 22.

We should add that many of the methods discussed in the book share important components. Changes in breathing dynamics figure prominently not just in whole-body relaxed breathing and end-tidal carbon dioxide biofeedback but also in heart rate variability biofeedback and yoga, Qigong, and various meditative and hypnotic approaches. Attention to muscle relaxation also cuts across methods, and there are both cognitive and meditative effects of engaging in any of the methods. All involve changes in mental set from thinking about everyday stressors, and most provide a particular alternative focus of attention. In their application to daily life, most approaches encourage the practitioner to use the method in stressful situations or when experiencing uncomfortable feelings, thus providing encouragement to expose themselves to these situations and feelings and thereby allow extinction of maladaptive stress-inducing conditional reflexes. Awareness of and comfort with body sensations are targeted in many of the methods. Also, to the extent that all of the methods involve an interaction with a therapist, the large effects of engagement with a warm and supportive instructor cannot be underestimated. Indeed, this may be a crucial component for all of the methods. Finally, the effects of expectancy and suggestion cannot be ruled out for any of the methods. Although behavioral placebos have been designed to evaluate most of the methods, these are rarely completely neutral. Some such controls, such as false biofeedback, can be frustrating and therefore may create greater stress, whereas others, such as paced breathing at faster rates, may have a relaxing and meditative effect, thus minimizing the effects of the "real" method.

With these caveats in mind, this volume offers a state-of-the-art description of empirically supported stress management methods, each described by a well-recognized expert on the method. We hope students, teachers, professional caregivers, and consumers continue to find this new edition an important and useful resource.

Contents

PART I. OVERVIEW OF STRESS AND STRESS MANAGEMENT

1. Conceptual Issues Underlying Stress Management 3
Robert L. Woolfolk and Paul M. Lehrer

2. Psychophysiological and Systems Perspectives on Stress and Stress Management 16
Paul M. Lehrer

3. The Psychology of Relaxation, Meditation, and Mindfulness: An Introduction to RMM Theory, Practice, and Assessment 39
Jonathan C. Smith

4. Stress, Inflammatory Cytokines, and the Brain: An Overview 58
Alexander W. Kusnecov, Sara Norton, and Marialaina Nissenbaum

5. Stress and Stress Management in Children and Adolescents 93
Jonathan S. Comer, Laura Nelson Darling, Cristina del Busto, Erica D. Musser, and Donna B. Pincus

PART II. STRESS MANAGEMENT METHODS

SECTION A. Psychophysiological Methods

6. Psychophysiological Interventions for Stress-Related, Medically Unexplained, and Anxiety Disorders 131
Richard N. Gevirtz

7. Progressive Relaxation: Origins, Principles, and Clinical Applications 151
F. J. McGuigan and Paul M. Lehrer

8. Eye Movement Desensitization and Reprocessing Therapy for Stress Management 193
Roger Solomon, Deany Laliotis, and Francine Shapiro

9. Neurofeedback with Biofeedback for Stress Management 214
Michael Thompson and Lynda Thompson

10. Biofeedback Training to Increase Heart Rate Variability 264
Paul M. Lehrer

11. Capnometry-Assisted Respiratory Training: Principles and Findings 303
Alicia E. Meuret and Thomas Ritz

12. Whole-Body Breathing: A Systems Perspective on Respiratory Retraining 327
Jan van Dixhoorn

SECTION B. **Meditative Methods**

13. Modern Forms of Mantra Meditation for Stress Management 377
Patricia Carrington and Paul M. Lehrer

14. Mindfulness Meditation for Stress Management 412
Jean L. Kristeller

15. Qigong Therapy for Stress Management 450
Kevin W Chen

16. Yoga for Stress Management: History, Research, and Practical Details 472
Shirley Telles, Niranjan Kala, Ram Kumar Gupta, and Acharya Balkrishna

SECTION C. **Psychotherapeutic Methods**

17. Cognitive Therapy for Stress 489
Robert L. Leahy and Scott Woodruff

SECTION D. **Hypnotic Methods**

18. The Autogenic Training Method of J. H. Schultz 527
Wolfgang Linden

19. The Treatment of Pain and Stress with Hypnosis: A Series of Emerging Literatures 553
Robert A. Karlin

SECTION E. **Other Methods**

20. Stress Concepts in Relation to Music 591
Töres Theorell and Ingrid Hammarlund

21. Walk–Talk Exercise, Stress Resilience, and Mental Health 606
Cristy Phillips and Wesley E. Sime

22. Wearing the Clinical Hat 649
Paul M. Lehrer and Robert L. Woolfolk

Index 657

PART I

OVERVIEW OF STRESS AND STRESS MANAGEMENT

CHAPTER 1

Conceptual Issues Underlying Stress Management

Robert L. Woolfolk
Paul M. Lehrer

THE CONCEPT OF STRESS

The word *stress* dates back to the 14th century and its origins to that time when the English language developed from an intermingling of Norman French and Anglo-Saxon ("Stress," 1989). *Stress* derives from the Middle French word *destresse* ("distress"), which in turn derives from the Latin *strictus* ("compressed"). Various forms of the word originally denoted hardship or adversity. By the 16th century, the word was employed to indicate subjecting an entity "(a material thing, a bodily organ, a mental faculty) to stress or strain; to overwork, fatigue" ("Stress," 1989).

Long before it was adopted as a scientific term, *stress* was part of the everyday English vernacular and was used to denote either an external disturbing event or the perturbation resulting from it. In the 19th century, *stress* became a precise scientific term employed within physics, used to refer to force applied to objects that could potentially result in deformation or strain. The prevailing use of the concept of stress in biological anthropology before the 1980s was to refer to an environmental condition placing "strain" on the organism (Iscan, 1983). For example, Siegel, Doyle, and Kelley (1977) describe external events as "heat stress" in experimental skeletal biology and in Stini's (1969) use of the concept of "nutritional stress" in studies of adaptation to chronic undernutrition.

The great physiologist Walter Cannon (1939), though he used the term *stress* infrequently, originated our modern biomedical concept of stress as involving a perturbation of somatic homeostasis by external threats that induce a mobilization of bodily resources to contend with the circumstances. Cannon coined the term *fight-or-flight response* to describe a mobilization of the organism that prepares it more effectively to aggress or to flee. Cannon located the genesis of the response in the limbic system and presented to us the ironic conception of the modern human being, located in a complex contemporary world in which physical danger is minimal, yet equipped with an antiquated reptilian response system disposed to mobilize the organism for fight or flight, even though, for most contemporary threats, neither fleeing nor combat is a viable option.

The next great figure in the history of stress was Hans Selye (1956), the man who promulgated the concept of stress within medicine and biology and made it a household word among the general public. Selye expanded upon Cannon's work and described three stages of the stress response: the alarm stage, the adaptive–resistance stage, and the exhaustion stage. The alarm stage is equivalent to Cannon's fight-or-flight response, the adrenomedullary response that prepares the individual to respond to an emergency or threat. During the second stage, adaptive–resistance, homeostatic processes cause the body to return to its state prior to arousal, when the stressor is no longer present. The exhaustion stage is sometimes called "burnout" and results from extended excessive metabolic demands of a protracted alarm stage. The resulting depletion of bodily resources makes the organism prone to infirmity, or even death. Under the influence of Selye's work, the biosocial conception of stress has shifted more and more to that of the organismic response to some external or internal stimulus.

Whereas Cannon tended to view stress as primarily in terms of a disruption of homeostasis during the elicitation of the fight-or-flight response, recent, more complex formulations of the response to stress have suggested that stress may activate bodily mechanisms whose function is to maintain stability in the face of a changing internal or external environment. The term *allostasis,* which literally means the maintenance of stability through change, was conceived by Sterling and Eyer (1988) in characterizing the functioning of the cardiovascular system in its adjustment to various levels of bodily activity.

The concept of allostasis has been incorporated into a model of stress by McEwan and colleagues (Goldstein & McEwen, 2002; McEwen, 1998) that emphasizes the complex and multifarious functions of the glucocorticoids and the catecholamines. A related concept, allostatic load, is used to refer to the "wear and tear" on the organism that results from multiple cycles of allostasis, as well as from less than optimal initiation and cessation of allostatic cycles. A classic example of animals manifesting allostatic load is male cynomolgus monkeys striving for status in an unstable dominance hierarchy who encounter repeated and continuous confrontations and conflicts with other animals. These monkeys exhibit accelerations in atherosclerotic plaque formation that are associated with rises in blood pressure and raised catecholamine levels (Manuck, Kaplan, Adams, & Clarkson, 1995). Another example would be the study showing that serum cholesterol levels in accountants rose and blood-clotting time was reduced during the peak workload of tax season (Friedman, Rosenman, & Carroll, 1958).

One Stress Response or Many?

Although the stress response is most often formulated in terms of the individual organism defending itself against threat by fighting or fleeing, a number of kinds of responses to stress have been observed. The antithesis of behavioral activation, a "freeze–hide" response, also has been widely observed. Evolutionary biologists sometimes refer to the tendency to produce an active versus a passive response to stress as a distinction between "hawks" and "doves" and between "hawk" versus "dove" strategies for acquiring resources and enhancing inclusive fitness (Kortea, Jaap, Koolhaasb, Wingfield, &

McEwen, 2005). Hawks are proactive and bold, whereas doves favor a response style that emphasizes passivity, reactivity, nonaggression, and caution. Each style has its evolutionary advantages. A recent paper has suggested that hawks exhibit Cannon's fight-or-flight response, the sympathetic adrenomedullary response that produces elevated epinephrine in the bloodstream, increased levels of synaptic norepinephrine, and, during fight, the activation of a hypothalamic–pituitary–gonadal response that produces an increase in plasma testosterone. In response to stress, doves manifest the activation of the hypothalamic–pituitary–adrenal axis and the concomitant production of corticotropin-releasing factor that stimulates the pituitary gland to secrete adrenocorticotropic hormone into the blood. As a result, the adrenal cortex releases cortisol. Although much of the research underlying the hawk–dove formulation is based on studies of infrahumans, some research on humans has been predicated on the view that each style can provide evolutionary advantages (Davies, Sturge-Apple, & Cicchetti, 2011). The possibility that the stress response may be more differentiated than originally assumed is an intriguing possibility for future research and theory and is further supported by findings suggesting possible gender-based differences.

Tend-and-Befriend

In a series of papers, Taylor and her colleagues (Taylor et al., 2000; Taylor et al., 2002; Taylor et al., 2010) have argued that males and females may respond to stress differently. In particular, they described a response to stress that is posited to be an alternative to fight or flight. This response is labeled *tend and befriend* and is thought to be characteristic of the female of various species. According to the model, the tend-and-befriend response is selected for by evolution and reflects the proclivity of females toward affiliation, cooperation, and caretaking: "Tending involves nurturant activities designed to protect the self and offspring that promote safety and reduce distress; befriending is the creation and maintenance of social networks that may aid in this process" (Taylor et al., 2000, p. 411).

The tend-and-befriend response putatively derives from females' attachment and caregiving propensities. The biological underpinnings of the response, adduced from human and animal studies, appear to involve oxytocin operating in concert with female reproductive hormones and endogenous opioid peptide mechanisms. Aggression in males seems to be mediated by arousal of the sympathetic nervous system, whereas in females aggression has not been reliably linked to sympathetic arousal, suggesting that, in females, aggression may not be a component of the adrenomedullary "initial alarm" stage of the response to stress. The possibility that the stress response may be "gendered" further complicates our picture of the stress response.

Parasympathetic Activity, Tend and Befriend, and Stress Management

The affiliative nature of the tend-and-befriend pattern has inherent stress management benefits. Whether among men or women, social support is a powerful buffer against the ravages of high external stress in many contexts (e.g., recent contributions to a huge literature include: Chang, Wang, Chang, Yu, & Lee, 2018; Coburn, Gonzales, Luecken, & Crnic, 2016; Faw, 2018; Fingerhut, 2018; Leon-Perez, Wallston, Goggins, Poppendeck, & Kripalani, 2016; Mackin, Perlman, Davila, Kotov, & Klein, 2017; Pow, King, Stephenson, & DeLongis, 2017; Stein & Smith, 2015; Wiesmaierova et al., 2019; Zhang, 2017).

Social support also has parasympathetic correlates that characterize relaxation and may buffer stress on a physiological level. Porges (2011) has observed that people (and animals) with a greater affiliative pattern tend to have greater parasympathetic tone, as evidenced in respiratory sinus arrhythmia (see Lehrer, Chapter 10, this volume). For example, dogs and horses, like people, have high respiratory sinus arrhythmia, whereas cats and nonaffiliative rodents do not. It is high in good marriages, low in bad ones. It is low in autism (Patriquin, Scarpa, Friedman, & Porges, 2013) and in marital conflict (Smith et al., 2009). The parasympathetic response is homeostatic in nature, as is the corticosteroid response described for doves (see Lehrer, Chapter 2; Kusnecov, Norton, & Nissenbaum, Chapter 4; and Lehrer, Chapter 10, this volume), and is an important component in stress resiliency, except when this modulatory response itself becomes dysregulated and causes problems (see Lehrer, Chapter 2, this volume). The physiological profile of the doves described above parallels that of the tend-and-befriend pattern.

Contemporary Revisions of the Stress Concept

In recent years, the concept of psychosocial stress has proliferated and become widespread in both scientific and popular usage, not only in the United States but also around the world. Public awareness of the adverse effects of stress has never been higher. In fact, one could argue that the awareness has become a preoccupation. Many magazine covers, including that of *Time,* have highlighted stories about stress, and countless self-help books on the topic have been printed. So great is the importance attributed to stress in the etiology of disease that laypersons occasionally regard it as more central to health than it may actually be. For example, in surveys, stress reliably emerges as the risk factor the public considers most important in the etiology of coronary heart disease, more important than smoking or hypertension (French, Senior, Weinman, & Marteau, 2001). And, indeed, stress is listed more frequently than tobacco consumption as a cause of cancer (Maskarinec, Gotay, Tatsumura, Shumay, & Kakai, 2001).

The concept of stress has been criticized for its lack of precision and questioned as to what exactly it refers. Indeed, as indicated earlier, *stress* is sometimes used to refer to what in psychology is called *external stimulation*. Under this usage, stress is in the environment, part of the external world. Another usage posits stress to be not the stimulus itself but the perceived or processed stimulus. This is the stance of those such as Richard Lazarus (1994) and other subscribers to a cognitive-appraisal model of stress. Still others conceive of stress as the response rather than the stimulus. Stress can be chronic or episodic, positive or negative, a problem or a challenge.

What is one to do with such a plurality of meanings? *Stress,* in fact, is probably best thought of as a generic, nontechnical term analogous to the terms *disease, trauma,* or *addiction*. At times, the term *stress* may function as little more than a rather crude metaphor. But stress is a useful umbrella concept in that it helps identify and categorize a multiplicity of diverse phenomena, although without supplying much in the way of theoretical guides to a deeper understanding of the phenomena. It is part of an idiom that allows laypersons to describe life's perturbations without using such potentially undesirable terms as *anxiety* or *depression*. It is also an umbrella term that fosters communication and heuristically beneficial frameworks for scientific collaboration (Woolfolk, 2001). The intellectual functions of the concept of stress often seem to be analogous to those of other biopsychosocial concepts occurring at similar levels of abstraction, such as disorder, trauma, and addiction, that organize objects of scientific research conveniently but without clear theoretical utility. For example, the stress label seems to work as a concept for communication among scientists and clinicians. Consequently, the notion of stress management, which is the common denominator of this volume, does not denote a narrow set of techniques with straightforward and consistent defining characteristics. Yet those of us who operate within the subculture of stress management recognize the affinities, or what Wittgenstein called "family resemblances," among the different endeavors that we pursue. The concept of stress may eventually be replaced by a set of more precise terms, such as *allostasis*. Until that time, however, *stress* is a term just nebulous enough to facilitate communication among diverse practitioners and to provide a rubric that has allowed a valuable set of scientific and clinical efforts to develop and to flourish.

STRESS MANAGEMENT

In the midst of the burgeoning public concern with stress and its deleterious consequences, scientists have systematically investigated the effects of numerous stress reduction techniques and validated the efficacy and effectiveness of various methods of managing stress. During the second half of the 20th century and the first two decades of the 21st, research has shown that a variety of such methods can now be warranted as "evidence-based." The clinician who turns to the research literature to learn about these efficacious methods, however, finds little of the art required to apply stress management techniques to real-world patients.

In the empirical research literature evaluating the efficacy of stress management techniques, the descriptions of treatment methods are cursory and terse; only those already intimately familiar with the interventions studied can understand clearly what procedures were involved. Treatments typically are not adapted to individual cases but are relatively uniform across all study participants. Frequently, the "clinical version" of the treatment undergoes some modification so that what is investigated is a standardized, sometimes abbreviated form suitable for replicable empirical studies. Moreover, stress reduction techniques often are streamlined to make them easier to teach, easier to learn, and consistent with control or comparison conditions on such dimensions as length of training or amount of therapist contact.

Our aims for the present volume are the same as for the editions that preceded it: Given the plethora of methods and claims on their behalf circulating through the mental health culture and society at large, clinicians and students interested in the treatment of stress problems need to learn not only which techniques have efficacy but also the clinical tradecraft that is an essential aspect of the application of those techniques. This book is, in some sense, designed to fill the gap between the academic research lab and the everyday world of clinical practice. We have sought to provide a handbook that is a kind of manual to inform the application of interventions that reduce stress. The present volume provides not only empirical evidence but also extensive descriptions of clinical methods, descriptions sufficient to provide a primer on clinical application. The book is designed to serve both the practitioner and the researcher. Although we have asked our contributors to refrain from providing comprehensive and exhaustive surveys of the empirical literature, we have asked for empirical validation for each of the methods described. All of our contributors are scientists as well as clinicians and we have chosen to include only chapters on methods that have a firm empirical base demonstrating efficacy in controlled trials. The chapters include state-of-the-art descriptions of the various techniques, clinical uses, and overall current understanding of the research literature.

We also chose contributors who are sophisticated and experienced clinicians, who provide procedural manuals and case examples based on their rich clinical experience.

They are consummate artists of their crafts, each a master of his or her respective area. We have commissioned them to make personal statements based on their clinical experience. Our charge to them has been to communicate to their fellow clinicians those aspects of clinical acumen, artistry, and sagacity that so often are missing from descriptions of stress management methods. We have asked our contributors to become teachers—not only to convey the readily specifiable, technical aspects of their crafts but also to explicate those seemingly ineffable therapeutic nuances and intuitive rules of thumb that are the hallmarks of clinical virtuosos. We have asked them also to describe pertinent research findings regarding both therapeutic and adverse consequences of their techniques and to show how they themselves utilize research findings to guide their practice.

All of the contributions to this volume address the basic clinical questions that cut across techniques: assessment, selection of appropriate techniques, the client–therapist relationship, the limitations of stress management technology, potential impediments to therapeutic success, client cooperation, resistance, and adherence to therapeutic regimens. The clinical experience of our contributors represents a unique and invaluable repository of knowledge—the kind of practical knowledge and clinical rules of thumb that can only be acquired through experience.

LIMITS OF THE RESEARCH LITERATURE: THE NEED FOR CLINICAL EXPERTISE

Due to the presence of experimental controls necessary for the preservation of internal validity, treatment outcome studies of efficacy may not always provide a veridical picture of the application of stress management methods in the real world. Furthermore, because of the methodological requirements of research designs seeking to isolate causal influences, a given group of participants often is administered a single stress management technique. Overly recalcitrant or disturbed participants may be excluded from studies at the outset, or they may become casualties of experimental attrition, resulting in their data being removed or statistically imputed in the final data analysis. The exigencies of research often dictate the random assignment of participants to treatment conditions, making it impossible to observe the interactions among individual differences and factors related to treatments. Because stress-related problems often are intertwined in complicated ways with other pathological life circumstances and personal characteristics, the standard factorial design is not well suited to an examination of these complexities.

Applications of general knowledge to unique, concrete human situations, however, are always problematic. In the treatment of stress-related disorders, bridging the gap between laboratory and clinic presents numerous challenges to which the research literature is an insufficient guide. In behavioral science experiments, the emphasis on statistical significance, or even an effect size, as an index of treatment success creates a different emphasis from that in the everyday world of clinical application. It is, of course, necessary to demonstrate that differences between treatment and control conditions are unlikely to have occurred as a result of chance factors. Efficacy versus a control condition in a laboratory setting is a necessary but insufficient basis for a technique to be inferred to be clinically effective in "real world" settings. Efficacy versus a control condition in a well-controlled study demonstrates that a technique was found superior to a control in a study with high internal validity. In the real-world utility of a therapeutic method, on the other hand, effectiveness requires that the impact of therapeutic techniques be demonstrated in a study with high external validity. Effectiveness research, exemplified by the recent generation of "services research" studies, attempts to examine the impact of clinical interventions in everyday real-world contexts. In such research, the external validity of the research may be accorded more importance than considerations of internal validity, resulting in the use of systematic naturalistic methods or quasi-experimental designs. Out in the real world of clinical services, a therapy technique not only must be better than nothing; it must also be clinically powerful enough to justify its use. It must provide sufficient relief of suffering and enhanced ability to function to make it worth the time and effort to invoke it. It must be sufficiently acceptable to the client population to generate a high rate of treatment adherence. It must also be cost-effective.

The distinction between efficacy and effectiveness research is paralleled in the pharmaceutical literature by the distinction between phase 2 and phase 3 drug trials. Phase 2 trials are designed to show efficacy under carefully controlled conditions. Phase 3 trials are designed to show effects in the wider population, including among people who may have various comorbid disorders, in maximally diverse populations, and administered by providers who might be expected to administer the drugs in standard practice under conditions in which they would ordinarily do so. Phase 3 trials must be large because of the large variability in effect expected in the general population and because they are considered definitive. They ordinarily are carried out on thousands or tens of thousands of individuals. Because of the expense and difficulty in doing controlled behavioral research of such magnitude, we know of no phase 3 trials of any behavioral intervention for any problem. Although meta-analyses of numerous smaller studies can be used to approximate the effect of a phase 3 trial, the many methodological and population differences among studies do not reach the definitive rigor of a well-designed phase 3 trial. Thus, in order to find symptoms and populations most appropriate for any particular intervention, additional large studies are needed in order to obtain definitive results from mediational analyses.

Thus empirical research cannot provide answers to all the questions that arise in the course of clinical practice. Applications of scientific knowledge to the clinical arena will inevitably contain elements of art and of pedagogy. A mistake made often by newcomers to this field is to assume that the extensive scientific foundation of self-regulation technology obviates the necessity for clinical sensitivity, perspicacity, and wisdom. Although stress reduction techniques are more standardized and explicit than some other therapeutic methods, their success is no less dependent on the tacit skills and know-how that experienced and effective clinicians develop as they face the intricate and thorny problems clients present to them.

THE IDIOGRAPHIC NATURE OF STRESS MANAGEMENT

Statistical results can never provide complete certainty for choosing the best treatment method of a particular individual. An important distinction that must be drawn at the outset is that between the nomothetic and the idiographic. Nomothetic knowledge is general; idiographic knowledge is specific to a particular case. Much knowledge in psychiatry and psychology is generic. Most knowledge that the therapist possesses is knowledge of generalities and is derived from theory, research, and personal experience. Psychosocial treatment is an application of this general knowledge to a specific case that is always, in some respects, unique. Therapeutic interventions, interpretations, and prescriptions always are directed toward an idiograph: an individual person, couple, family, or group. The therapist is always in the position of asking her- or himself, "What is going on here, with this particular case I presently am treating?" The challenge in treating any given case is determining which generalities, to what degree, apply to that particular case. Such determinations always involve some uncertainty and may require some trial and error. Also, in case studies, we are always prone to the fallacy of *post hoc ergo propter hoc*.[1]

Even in applied fields derived from such systematic and unimpeachable disciplines as physics and chemistry, application to specific cases is not entirely straightforward. Mishaps occur frequently, and some practical knowledge about the particular arena of application often is required to effect a successful translation from theory to practice. Newly created airplanes may not perform aerodynamically as expected, climate control systems may not produce the temperatures intended, tunnels may collapse, winds may cause buildings to sway unacceptably, and torpedoes may bounce off the hulls of enemy ships without detonating.

Biological and psychological phenomena are more complex and variable than those of the physical sciences. They are more difficult to classify and measure. The truth about them is harder to discover and more difficult to confirm. Most research results are subject to conflicting interpretations, and debate on the clinical significance of almost any research finding often seems endless. When we do find an unchallenged truth, it commonly comes in the form of a stochastic or probabilistic generalization, for example, that smokers are twice as likely to develop heart disease or that Treatment A is effective 60% of the time whereas Treatment B works 45% of the time. The external and ecological validities of clinical research are rarely established for the clinician. Can the treatments be implemented? Was the sample representative? Will the results generalize? These elementary questions often have no clear answers. Now, this is not to say nomothetic research findings are without import. Often they are the best we can do in the absence of knowledge about the particular case with which we are concerned.

But treatments are always applied to unique individuals. Helping an individual manage his or her stress is best achieved through a thorough and comprehensive understanding of the person being treated. Stress management is an active intervention, analogous to physical therapy. The patient's cooperation and active participation are a requirement of effective treatment. Also, in whatever methods the clinician is expert, it is always valuable to be able to determine the source of an individual's stress. This last endeavor is by no means always straightforward and may require considerable clinical skill and practical judgment and, at the very least, the development of an open relationship with the patient.

PHILOSOPHICAL AND SOCIOCULTURAL ASPECTS OF STRESS MANAGEMENT

Despite the impressive array of clinical wisdom and empirical data accumulated to date about modifying stress responses in individuals, it is likely that some issues related to stress management can only be comprehended at the levels of history, society, and culture. The stressors, diets, toxins, and activity patterns of industrialized urban life take their toll on our minds and on our bodies. Diseases such as coronary heart disease, hypertension, and cancer, which are rare among primitive peoples, are our chief causes of death and disability. A major reason for the rise of the degenerative physical diseases, as well as the high levels of psychological distress in contemporary Western societies, may well be the stress of modern life, as well as changes in diet and decreases in exercise. This stress seems inescapable; no matter what we do, we cannot avoid it entirely.

Why should stress be such a problem in the contemporary world? Were there not terrible happenings and awful circumstances that troubled our ancestors, just as our current frustrations and tribulations beset us? Clearly, premodern life was (and still is, in contemporary developing nations) very difficult. Wars, pestilence, and dangers from the elements have undoubtedly produced emotional arousal throughout human history. And, indeed, life often has been viewed as too much for human beings, almost too difficult to bear. The first noble truth of Buddhism asserts that life is *dukkha* (usually translated as "suffering," but occasionally translated as "stress"). Many centuries ago, the Stoics developed a philosophy based on the view that extraordinary training was necessary for one not to be undone by the ubiquitous perturbations of this existence.

Yet there are key differences in the sociocultural environment of the modern world that can foster a kind of unremitting tension in modern individuals—at levels that are chronic and multilayered, as opposed to the less complex stressors that were (and are) more characteristic of less complicated societies. The modern world is fundamentally and qualitatively different from that of the past. These differences provide some important clues to the capacity of contemporary Western society to create great distress and tension in its citizens.

Among the distinctive features of modernization are the ordering of life by the clock and a large increase in time-pressured work. Caplan, Cobb, French, Harrison, and Pinneau (1975) found that machine-paced assembly workers reported more somatic complaints and anxiety than did assemblers who were not machine paced. Levi and his colleagues (Levi, 1972) reported that piecework pressure produced increases in noradrenaline levels and in blood pressure. As mentioned earlier, tax accountants were found to show substantial increases in serum cholesterol and reductions in blood-clotting latency when working under the pressure of deadlines (Friedman et al., 1958).

As Alvin Toffler (1970) and various other observers have pointed out, not only is modern society an ever-changing panorama, but also the rate of that change is ever-increasing. Many of the implications of this condition are rather straightforward. An individual is constantly required to make adjustments to a varying sociocultural matrix. Such social disruptions can elevate levels of stress. In the areas of job skills, interpersonal relationships, management of personal finances, and sex roles, to name just a few, such rapid and fundamental changes have occurred that old assumptions are in continual need of revision.

Three hundred years ago, it was possible for one individual, the German philosopher Gottfried Wilhelm von Leibniz, to assimilate the entire scope of human knowledge. Eighty-five years ago, an individual could know the entire fledgling field of psychology. But today the knowledge explosion makes it impossible for one person to possess a significant fraction of the pertinent information in even a small domain of that field, or any other. And the pool of knowledge is growing every day. Professionals who are not inclined toward voracious reading within their specialties are soon out of date, their knowledge obsolete. Life is a little like this for all of us: We must keep changing, ceaselessly adapting to a world that transforms itself more rapidly each day. We must move faster and faster just to maintain our places.

One effect of industrialization and modernization is almost universally regarded as beneficial—that is, the enhancement of the freedom and material well-being of individuals. The removal of many of the economic and social barriers to personal growth and self-expression presents the modern individual with a dazzling array of choices, as well as an awesome set of responsibilities. Today we choose our own careers, our circles of friends, our places of residence, and, perhaps most importantly, our own values. We must make decisions on a number of issues that would have been given or fixed in prior eras. We decide everything from whether to alter our appearances through dieting, exercise, or cosmetic surgery to what religion (if any) to follow. We are able to create for ourselves the rules that will govern relations with both intimates and acquaintances. The plasticity of the human being is such that even alteration of gender is now possible. Very little is set, fixed, or taken for granted. In the absence of some generally agreed-upon set of values or beliefs that provides practical wisdom to guide the making of choices, too many options can be difficult to bear. The great French sociologist Emile Durkheim (1897/1958), writing at the turn of the century, called this lack of values and norms "anomie"; he demonstrated that, under anomic conditions, suicide rates rise dramatically.

In the contemporary global economy, individuals continually are thrown back upon themselves and their own psychic resources. The German sociologist and philosopher Arnold Gehlen (1980) has written that one of the functions of society is to protect the individual from the burden of excessive choice. If this be so, contemporary industrialized societies are less than completely successful in this function. Never before has such a large percentage of the population been without durable and dependable social support.

The loss of community is related to the lack of meaning that is so often described by sociologists and existential philosophers as a concomitant of contemporary life. The premodern worldview was communal and spiritual, as contrasted with the individualistic and materialistic consciousness of contemporary times. Premoderns felt themselves to be useful and necessary elements of a cosmological order that had inherent purpose and meaning (Taylor, 1975). But the replacement of religion by science as the ultimate source of epistemic authority, the desacralization of nature, the ascendency of technology and its associated rationality and means–ends perspective, and the relativization of values have disrupted the human security that emanated from a sense of belonging and the confidence that each individual life had some larger purpose and meaning (Richardson, Fowers, & Guignon, 1999). Existential writers such as Albert Camus have described this shift in human self-perception in compelling terms: "In a universe suddenly divested of illusions and lights, man feels an alien, a stranger. His exile is without remedy since he is deprived of the memory of a lost home or the hope of a promised land" (Camus, 1960, p. 5).

The advent of computer technology and the Internet and widespread use of cell phones and texting as a primary method of interaction have both added to and subtracted from stress, social cohesiveness, and the sense of social support. Social media, blogs, videos, and so forth provide an almost unlimited amount of information on ways people can connect with each other and manage stress, but they also provide an avenue for Internet bullying and fear mongering and may sometimes substitute for the face-to-face human contact and touching that play a strong role in human bonding.

There is much evidence that people who find some purpose in existence, who believe that their activities are meaningful, and who view life as possessing coherence and lawfulness are less likely to manifest stress-related reactions (Antonovsky, 1987; Kobasa, 1979; Shepperd & Kashani, 1991). This evidence brings to mind the writings of the great sociologist Max Weber and his concept of "theodicy." Theodicies are elements of a cultural worldview that explain and confer meaning on experiences of suffering and wrongfulness. Berger, Berger, and Kellner (1973) have pointed out that despite the great changes that have accompanied modernization, the "finitude, fragility, and mortality" of the human condition is essentially unchanged; at the same time, those previous definitions of reality that made life easier to bear have been seriously weakened by modernization.

Psychotherapy and stress management (especially the cognitive aspects) may very well be in the business of supplying secular theodicies to people who look to science and scientifically grounded professionals to alleviate the discomforts of life (Woolfolk, 1998). The worldviews we proffer typically are justified on the basis of some instrumental criterion: They reduce stress, promote health, enhance happiness, or the like. Determining the beliefs that reduce the stress of life is, however, not equivalent to establishing the truth or legitimacy of those beliefs. Any worldview that is advanced solely on the basis of its instrumental benefits to health and happiness will be subject to various forms of criticism. One such criticism is the ethical rejoinder suggesting that many beliefs may be worth being stressed for—indeed, worth dying for. A worldview whose ultimate claim to authority is pragmatic may be inherently self-limiting and self-defeating if an "inherent" sense of purpose is what provides people with the capacity to withstand the corrosive aspects and vicissitudes of living.

The activities of stress management professionals are orchestrated within the sociocultural matrix and also—along with the ubiquitous mental health and self-improvement industry of which they are part—serve to constitute this matrix. It would seem important for practitioners to have a grasp of the moral and sociohistorical aspects of their role. With respect to existential issues, they may not possess any ultimate answers, but they should at least know what the questions are.

NOTE

1. The Latin expression is translated "After that, therefore because of that" and refers to the logical error of inferring that event A causes event B simply because A occurs before B. *Post hoc ergo propter hoc* is a special case of the fallacy of the "false cause" (Copi, 1961). A related instance of the false-cause fallacy familiar to behavioral scientists is that of inferring causation from mere correlation.

REFERENCES

Antonovsky, A. (1987). The salutogenic perspective: Toward a new view of health and sickness. *Advances, 4,* 47–55.

Berger, P. L., Berger, B., & Kellner, H. (1973). *The homeless mind.* New York: Random House.

Camus, A. (1960). *The myth of Sisyphus and other essays.* New York: Vintage.

Cannon, W. B. (1939). *The wisdom of the body.* New York: Norton.

Caplan, R. D., Cobb, S., French, J. R. P., Jr., Harrison, R. V., & Pinneau, S. R., Jr. (1975). *Job demands and worker health* (DHEW Publication No. NIOSH 75-160). Washington, DC: U.S. Government Printing Office.

Chang, P.-Y., Wang, H.-P., Chang, T.-H., Yu, J.-M., & Lee, S.-Y. (2018). Stress, stress-related symptoms and social support among Taiwanese primary family caregivers in intensive care units. *Intensive and Critical Care Nursing, 49,* 37–43.

Coburn, S. S., Gonzales, N. A., Luecken, L. J., & Crnic, K. A. (2016). Multiple domains of stress predict postpartum depressive symptoms in low-income Mexican American women: The moderating effect of social support. *Archives of Women's Mental Health, 19*(6), 1009–1018.

Copi, I. M. (1961). *Introduction to logic.* New York: Macmillan.

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D. (2011). Interparental aggression and children's adrenocortical reactivity: Testing an evolutionary model of allostatic load. *Development and Psychopathology, 23,* 801–814.

Durkheim, E. (1958). *Suicide*. Glencoe, IL: Free Press. (Original work published 1897)

Faw, M. H. (2018). Supporting the supporter: Social support and physiological stress among caregivers of children with severe disabilities. *Journal of Social and Personal Relationships, 35*(2), 202–223.

Fingerhut, A. W. (2018). The role of social support and gay identity in the stress processes of a sample of Caucasian gay men. *Psychology of Sexual Orientation and Gender Diversity, 5*(3), 294–302.

French, D., Senior, V., Weinman, J., & Marteau, T. (2001). Causal attributions for heart disease: A systematic review. *Psychology and Health, 16,* 77–98.

Friedman, M., Rosenman, R. H., & Carroll, V. (1958). Changes in serum cholesterol and blood clotting time in men subjected to cyclic variation of occupational stress. *Circulation, 17,* 852–861.

Gehlen, A. (1980). *Man in the age of technology*. New York: Columbia University Press.

Goldstein, D. S., & McEwen, B. (2002). Allostasis, homeostasis, and the nature of stress. *Stress, 5,* 55–58.

Iscan, M. Y. (1983). *A topical guide to the American Journal of Physical Anthropology, Volumes 22–53 (1964–1980)*. New York: Liss.

Kobasa, S. (1979). Stressful life events, personality, and health: An inquiry into hardiness. *Journal of Personality and Social Psychology, 37,* 1–11.

Kortea, S. M., Jaap, M., Koolhaasb, J. M., Wingfield, J. C., & McEwen, B. S. (2005). The Darwinian concept of stress: Benefits of allostasis and costs of allostatic load and the trade-offs in health and disease. *Neuroscience and Biobehavioral Reviews, 29,* 3–38.

Lazarus, R. S. (1994). *Emotion and adaptation*. New York: Oxford University Press.

Leon-Perez, G., Wallston, K. A., Goggins, K. M., Poppendeck, H. M., & Kripalani, S. (2016). Effects of stress, health competence, and social support on depressive symptoms after cardiac hospitalization. *Journal of Behavioral Medicine, 39*(3), 441–452.

Levi, L. (1972). *Stress and distress in response to psychosocial stimuli*. Elmsford, NY: Pergamon Press.

Mackin, D. M., Perlman, G., Davila, J., Kotov, R., & Klein, D. N. (2017). Social support buffers the effect of interpersonal life stress on suicidal ideation and self-injury during adolescence. *Psychological Medicine, 47*(6), 1149–1161.

Manuck, S. B., Kaplan, J. R., Adams, M. R., & Clarkson, T. B. (1995). Studies of psychosocial influences on coronary artery atherosclerosis in cynomolgus monkeys. *Health Psychology, 7,* 113–124.

Maskarinec, G., Gotay, C. C., Tatsumura Y., Shumay, D. M., & Kakai, H. (2001). Perceived cancer causes: Use of complementary and alternative therapy. *Cancer Practice, 9,* 183–190.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840,* 33–44.

Patriquin, M. A., Scarpa, A., Friedman, B. H., & Porges, S. W. (2013). Respiratory sinus arrhythmia: A marker for positive social functioning and receptive language skills in children with autism spectrum disorders. *Developmental Psychobiology, 55*(2), 101–112.

Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation*. New York: Norton.

Pow, J., King, D. B., Stephenson, E., & DeLongis, A. (2017). Does social support buffer the effects of occupational stress on sleep quality among paramedics?: A daily diary study. *Journal of Occupational Health Psychology, 22*(1), 71–85.

Richardson, F. C., Fowers, B. J., & Guignon, C. B. (1999). *Re-envisioning psychology: Moral dimensions of theory and practice*. San Francisco: Jossey-Bass.

Selye, H. (1956). *The stress of life*. Toronto, Ontario, Canada: McGraw-Hill.

Shepperd, J. A., & Kashani, J. V. (1991). The relationship of hardiness, gender, and stress to health outcomes in adolescents. *Journal of Personality, 59,* 747–768.

Siegel, M. I., Doyle, W. J., & Kelley, C. (1977). Heat stress, fluctuating asymmetry and prenatal selection in the laboratory rat. *American Journal of Physical Anthropology, 46,* 121–126.

Smith, T. W., Uchino, B. N., Berg, C. A., Florsheim, P., Pearce, G., Hawkins, M., et al. (2009). Conflict and collaboration in middle-aged and older couples: II. Cardiovascular reactivity during marital interaction. *Psychology and Aging, 24*(2), 274–286.

Stein, E. R., & Smith, B. W. (2015). Social support attenuates the harmful effects of stress in healthy adult women. *Social Science and Medicine, 146,* 129–136.

Sterling, P., & Eyer, J. (1998). Allostasis: A new paradigm to explain arousal pathology. In J. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition and health* (pp. 629–649). New York: Wiley.

Stini, W. A. (1969). Nutritional stress and growth: Sex differences in adaptive response. *American Journal of Physical Anthropology, 37,* 417–426.

Stress. (1989). In J. A. Simpson & E. S. C. Weiner (Eds.), *Oxford English dictionary* (2nd ed.). Oxford, UK: Oxford University Press.

Taylor, C. (1975). *Hegel.* Cambridge, UK: Cambridge University Press.

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A. R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107,* 411–429.

Taylor, S. E., Lewis, B. P., Gruenewald, T. L., Gurung, R. A. R., Updegraff, J. A., & Klein, L. C. (2002). Sex differences in biobehavioral responses to threat: Reply to Geary and Flinn. *Psychological Review, 109,* 751–753.

Taylor, S. E., Seeman, T. E., Eisenberger, N. I., Kozanian, T. A., Moore, A. N., & Moons, W. G. (2010). Effects of a supportive or unsupportive audience on biological and psychological responses to stress. *Journal of Personality and Social Psychology, 98,* 47–56.

Toffler, A. (1970). *Future shock.* New York: Random House.

Wiesmaierova, S., Petrova, D., Arrebola Moreno, A., Catena, A., Ramirez Hernandez, J. A., & Garcia-Retamero, R. (2019). Social support buffers the negative effects of stress in cardiac patients: A cross-sectional study with acute coronary syndrome patients. *Journal of Behavioral Medicine, 42*(3), 469–479.

Woolfolk, R. L. (1998). *The cure of souls: Science, values, and psychotherapy.* San Francisco: Jossey-Bass.

Woolfolk, R. L. (2001). The concept of mental illness: An analysis of four pivotal issues. *Journal of Mind and Behavior, 22,* 161–178.

Zhang, R. (2017). The stress-buffering effect of self-disclosure on Facebook: An examination of stressful life events, social support, and mental health among college students. *Computers in Human Behavior, 75, 527–537.*

CHAPTER 2

Psychophysiological and Systems Perspectives on Stress and Stress Management

Paul M. Lehrer

THE NATURE OF STRESS

What is stress? It is both a stimulus and a response. As a stimulus, it involves a cue or series of cues signaling a need to prepare for danger or for action. We may think of these cues and the responses to them as simple Pavlovian conditioning effects, in which the "conditional response" is the body's preparation to respond to an anticipated physical need to think faster and/or to act. By themselves, these "calls to action" may not be sufficient to be considered "stressful," unless they are accompanied by unpleasant symptoms, elevated risk for illness, or impairment in function. Indeed, they are part of everyday life and are responsible for our ability to work, perform, and think more productively when needed. The responses may include increased blood flow to the brain, nervous system, and muscles that increases neuromuscular efficiency and strength, improves thought processes to prepare strategies for coping with danger, increases inflammatory activity to fight invasion of microbes, and increases the efficiency of various modulatory responses that prevent stress reactions from hurting the body. Such modulatory reflexes act to limit the mobilization and inflammatory responses to prevent self-injury. Poor and dysfunctional regulation of stress responses may occur when stress stimuli are too severe or prolonged, or when coping behaviors and reflexes are insufficient to manage the stress stimuli and the body's responses to it. Stress-induced dysregulation is responsible for a large number of both physical and psychological ailments (see Table 2.1). This book is a compilation of empirically validated methods that help strengthen cognitive and psychophysiological processes involved in coping with stress.

In modern society, many stress stimuli are social. Social stressors usually do not require the type of physical mobilization required by our ancestors who were dealing

TABLE 2.1. Conditions Exacerbated by Stress

Mental/emotional conditions	Physical conditions

• Anxiety disorders
• Depression
• Schizophrenia
• Eating disorders
• Behavioral disorders
• Personality disorder
• Anger disorders
• Somatization/hypochondriasis
• Tourette syndrome
• Insomnia
	• Asthma
	• Arthritis
	• Hypertension
	• Various pain syndromes
	  ◦ Headache, TMJ
	• Irritable and inflammatory bowel
	• Raynaud's disease
	• Cancer
	  ◦ Chemotherapy side effects
	  ◦ Disease progression
	• Stress of mechanical ventilation
	• Diabetes
	• CPAP adherence
	• Postsurgery wound healing
	• Parkinson's disease
	• Infectious disease
	• Allergy
	• Atherosclerosis and heart disease
	• Unexplained symptoms
	• Pain and somatic problems in general

Note. Data from citations in PsycInfo (American Psychological Association, 2020) and Medline (National Library of Medicine, 2020).

with saber-toothed tigers, so the physical mobilization produced by stress responses often is not particularly useful to us, but it occurs anyway as a vestige of our ancient past as a species. The sociological and social psychological literature describes stress symptoms associated with low social status, including such factors as poverty, crime, racial or ethnic discrimination, and so forth (Hollingshead & Redlich, 1958; Merton, 1938; Pearlin, 1969; Srole, Langner, Michael, Oplear, & Rennie, 1962). Lower rank in organizational hierarchies also increases vulnerability to stress (Luceno-Moreno, Garcia-Albuerne, Talavera-Velasco, & Martin-Garcia, 2016; Martins & Lopes, 2012; Shirom & Mayer, 1993). Although having a lower institutional rank may cause stress through lower levels of control of one's work environment, there is some evidence that an individual's *perception* of low social status may contribute to stress, whether or not this corresponds to more objective measures (Fales, 2018; Han, 2014; President, 2017; Sabik, Falat, & Magagnos, 2020; Scott et al., 2014). Minority groups and lower-socioeconomic status groups are particularly vulnerable to stress effects in the absence of protective family and community conditions. Individual psychological, social, and environmental factors also play a role in stress vulnerability. These could include isolation, poor family coherence, changing role expectations (e.g., changing sex- and age-role expectations over time, culture clashes for both immigrants and hosts), effects of economic swings and risks of unemployment, effects of natural disasters and social upheavals, poor or inadequate diet, deprivation of daylight, family mental illness and abuse, and so forth.

A survey by the American Psychological Association (2017) found that concerns about health care and the economy topped the list of stress sources, along with some other items related to current political debates in a turbulent political time in the United States. (See Table 2.2.) Presumably the concerns for 2020 will relate to the stresses induced by the COVID-19 pandemic, including fear of disease, economic dislocation, social isolation, exacerbation of household relationship problems, switch from live to

TABLE 2.2. Sources of Stress

Source of Stress	% of Responders
Health care	43
The economy	35
Trust in government	32
Crime and hate crimes	31
Terrorist attacks	30
High taxes	28
Unemployment/low wages	22
Climate change/environment	21

Note. Data from *www.apa.org/news/press/releases/stress/2017/state-nation.pdf*.

online methods for working, shopping, and socializing, and other stress sources yet to be identified.

There also is considerable evidence that exposure to severe stress early in life creates more vulnerability to stress symptoms in adulthood (Favaro, Tenconi, Degortes, Manara, & Santonastaso, 2015; van den Bosch, Dijk, Tibboel, & de Graaff, 2017), perhaps because the body becomes attuned to expect conditions that require psychobiological readiness to adapt to stressful conditions. It may come to expect a need for greater alertness, sensitivity to cues for danger, and physiological preparation for behavioral mobilization as well as the need to cope physiologically with injury. It is also possible that these characteristics can be transmitted intergenerationally, either from in utero learning or epigenetic changes (Fogelman & Canli, 2019; Guillen-Burgos & Gutierrez-Ruiz, 2018; Voisey, Young, Lawford, & Morris, 2014). There is evidence that in utero exposure to violence and other stressors experienced by the mother can create greater incidence of allergy and asthma in the offspring (Lee et al., 2018; Magnus et al., 2018; Wright et al., 2004). It is known that inflammation and elevated immune reactions are characteristic of the stress response, leading to such autoimmune symptoms when the body's coping mechanisms are overwhelmed, as described in Chapter 4 of this volume by Kusnecov, Norton, and Nissenbaum. The need to adapt to severe stress in subsequent generations could contribute to species survival in stressful environments and would be consistent with possible epigenetic changes caused by prenatal exposure to stress.

HARDINESS

The correlation between exposure to stressors and occurrence of stress symptoms is far from perfect. Although various conditions lead to higher rates of illness and feelings of discomfort, some people seem to remain unscathed. A concept used to explain this discrepancy is "hardiness." Some people appear to take exposure to stressors more in stride than do others (Maddi, 2017; Maddi et al., 2017; Pitts, Safer, Russell, & Castro-Chapman, 2016; Stoppelbein, McRae, & Greening, 2017). Although psychological research on hardiness has tended to focus mainly on such social buffers as cohesiveness and support (Kuzmin & Konopak, 2016; Zeer, Yugova, Karpova, & Trubetskaya, 2016), biological factors also play a role (McVicar, Ravalier, & Greenwood, 2014; Oken, Chamine,

& Wakeland, 2015; Parkash, Archana, & Kumar, 2017). Longitudinal developmental research has shown that some infants show greater levels of autonomic and emotional reactivity than others and that these correlate, however imperfectly, with greater emotional reactivity later in life (Berry, Blair, Willoughby, & Granger, 2012; Cohen, 1989; Raby, 2016; Wagner et al., 2017). Some people are more resilient, perhaps showing transient effects of strain but bouncing back to relative equanimity most of the time.

SPECIFIC TREATMENTS FOR PARTICULAR SYMPTOMS OF STRESS

The kinds of stress-related symptoms that people experience can differ. An interesting experiment by Peter Lang in the 1960s provides a useful way of classifying some of these differences. In a study of people who were afraid of snakes, participants were told to approach a caged harmless snake, to pick it up, and to play with it. He measured their physiological arousal as reflected in heart rate and skin conductance, their expressed fear, and how close they actually came to the snake. He found these measures not to be highly correlated with each other. He concluded that three dimensions of fear are partially independent of each other: physiological, cognitive, and behavioral. He advised that each of these dimensions be given specific attention in treatment for stress-related problems (Lang, 1968, 1979), and that some people may need more attention paid to one dimension than to another. Some people are constitutionally more physiologically reactive or have had illnesses (Menard, Pfau, Hodes, & Russo, 2017; Porcelli, Laera, Mastrangelo, & Di Masi, 2012) or life experiences (Engert et al., 2010; Goodman & Brand, 2009; Gunnar & Vazquez, 2006; Uchida et al., 2010) that make them more physiologically sensitive. Some people tend to think of the world and their life situations as more threatening than do others and have less "self-efficacy" for managing their problems (Chung, AlQarni, Al Muhairi, & Mitchell, 2017; Delahaij & Van Dam, 2017; Lavenda & Kestler-Peleg, 2017; Troesch & Bauer, 2017). Some people are less skillful than others in managing their problems or managing particular job or social demands (Fadirepo, 2013; Faul, Jim, Williams, Loftus, & Jacobsen, 2010; Harvey, Harris, Harris, & Wheeler, 2007; Jordet, Hartman, Visscher, & Lemmink, 2007; Zhang et al., 2014). When looking at diagnostic classifications of anxiety disorders by the psychiatric profession (American Psychiatric Association, 2013), these three dimensions neatly correspond to specific anxiety diagnoses: panic disorder on the physiological dimension, characterized by the physiological symptoms of panic, generalized anxiety disorder on the cognitive dimension, characterized by exaggerated and pervasive worry, and phobic disorders on the behavioral dimension, characterized by behavioral avoidance.

More highly specific treatment tailored to the particular system displaying the problem can sometimes produce larger beneficial effects. Although cognitive and behavioral interventions can produce psychophysiological relaxation (Abelson, Neese, Weg, & Curtis, 1996; Hofmann, 1999; Lundgren, Carlsson, & Berggren, 2006), these effects are not consistently reported (Michelson et al., 1990), suggesting that in some cases a more psychophysiologically focused treatment may be more powerful for treating psychophysiological symptoms of stress. Even biofeedback procedures to train one specific muscle group (usually the frontalis muscle) often do not generalize to greater relaxation in other muscles (Thompson, Haber, & Tearnan, 1981), so a single psychophysiological approach may not even be the most beneficial approach for all psychophysiological problems. Similarly, cognitive interventions may be expected to have specific effects on thoughts, behavioral interventions on coping efficacy.

Although there are few head-to-head comparison studies of these modalities on specific outcomes and considerable evidence for cross-modality improvements, there is some evidence for symptom-treatment specificity. In a meta-analysis of breathing therapies for anxiety symptoms, we recently found a large effect size for panic symptoms, a moderate effect size for general anxiety symptoms, and a small effect size for phobias and posttraumatic stress disorder (PTSD) symptoms (Lehrer et al., 2017). Exposure therapies have been found to have greater effects than relaxation or cognitive therapies for treating phobic disorders, although the latter do have some therapeutic effect (Da Costa, Sardinha, & Nardi, 2008; Gilroy, Kirkby, Daniels, Menzies, & Montgomery, 2000; Otto, Hearon, & Safren, 2010). For generalized anxiety disorder, cognitive-behavioral therapy has been found to have a small and inconsistently greater effect than relaxation therapy (Donegan & Dugas, 2012; Kushner et al., 2013; Norton, 2012).

FOCUS OF THIS VOLUME: PSYCHOPHYSIOLOGICAL STRESS TREATMENTS

In this volume, we focus primarily on interventions with a strong psychophysiological focus, although we also include chapters describing cognitive and behavioral interventions. We do this because the psychophysiological dimension is one that often is shortchanged in both training and practice of modern psychotherapists. This volume contains chapters for each modality written by leading practitioners of those modalities.

For better or worse, modern treatment of stress-related conditions often is relegated to providers associated with medical care, where treatment most often is pharmacological. This volume deals with nonpharmacological alternatives. However, even independent nonmedical psychotherapists who eschew the "medical model" of emotional disorders still function in a medically dominated system, at least to the extent that their incomes are often dependent on medical insurance and the classification of emotional problems as forms of illness. Although the growth of the "life coach" industry may be an exception to this tendency, and although teachers, coaches, and counselors have long contributed their skills to building stress resilience, the ways in which we treat stress-related problems have long been influenced by medical conceptualizations, from the time of Hippocrates and the ancient Greeks (Chrousos, Loriaux, & Gold, 1988) through psychoanalysis (Cleghorn, 1965; Hruby, Hasto, & Minarik, 2011; Kimball, 1983) and the current reliance on the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) system for conceptualizing "mental diseases" (Padmanabhan, 2017).

Although in earlier days many of the medical approaches overlapped the psychophysiological approaches described in this volume, the advent of modern psychopharmacology has accentuated chemical treatment of emotional problems, with psychophysiological interventions playing a minor and often insignificant role. The advent of psychoanalysis produced a temporary change in this pattern, so helping people to understand unconscious conflicts and wishes became seen as a medical specialty. Nevertheless, advocates of psychoanalysis and some of the other methods described in this book, particularly autogenic training, advised that practice of these methods be restricted to physicians. However, from the early papers by Freud on "lay analysis" (Freud & Eitingon, 1927), nonmedical practitioners of psychoanalysis played increasingly important roles in the practice and development of this and associated methods, and although psychoanalytical treatments are no longer considered the exclusive province of psychiatrists, the practice of psychotherapy retains a medical stamp, and people who treat stress-related problems almost always justify insurance reimbursement by referring to the American Psychiatric

Association's taxonomy of emotional problems in the DSM manuals (e.g., American Psychiatric Association, 2013).

The behavior therapy movement was a reaction among psychologists to this medical approach and sought to use the science of human behavior rather than, as in psychoanalysis, clinical experience to guide practice. The later development of cognitive-behavior therapy (CBT) imported from psychoanalytic therapy some of the understanding of thought patterns in emotional problems (Beck, 2004; Ellis, 2005) and targeted them more directly in a behavior modification framework. However, in many circles, "cognitive-behavioral therapy" has become almost exclusively a form only of *cognitive* therapy, with psychophysiological and even behavioral dimensions falling by the wayside. More recently, behavioral techniques have gained some resurgence, such as in the practice of exposure therapy for anxiety disorders and behavior activation for depression (Ahs, Gingnell, Furmark, & Fredrikson, 2017; Braun, Gregor, & Tran, 2013; Collins & Coles, 2017; Hofmann, Mundy, & Curtiss, 2015; Hopko & Lejuez, 2007; Jayasinghe et al., 2014; McGuire et al., 2012; Ramnero, Folke, & Kanter, 2016; Williams, Crozier, & Powers, 2011). However, psychophysiological approaches still play only a minor role, with many CBT practitioners having little or no training in these methods (cf. current texts for training in behavior therapy; e.g., Spiegler & Guevremont, 2016). The emphasis in this book on psychophysiological approaches is an attempt to restore the balance among techniques for treating cognitive, behavioral, and psychophysiological aspects of emotional problems by providing a number of chapters in the various empirically validated methods of psychophysiological interventions.

So, then, what do these psychophysiological interventions target? The targets are improving regulation and resilience.

REGULATION

Popularly, stress-related problems are often measured as *too much* or *too little* of some psychophysiological dimension: high or low levels of blood pressure, elevated muscle tension, exaggerated startle reactivity, and so forth. These can be manifested in such physical conditions as hypertension, muscular aches and pains, insomnia, constipation, low sexual desire or impaired performance, loss of appetite, various anxiety conditions, and more. They are all manifestations of the well-known "fight or flight" reaction, in which the sympathetic branch of the autonomic nervous system predominates and inhibits the opposing influences of the parasympathetic nervous system, which has been characterized as the "rest and digest" system and which lowers heart rate and blood pressure, fosters feelings of relaxation, increases gastrointestinal activity, facilitates sexual arousal, and other effects. Because sympathetic overarousal is often one of the predominant symptoms of stress, many stress management methods specifically target decreasing sympathetic activity, sometimes accompanied by ways of increasing parasympathetic function (Bali & Jaggi, 2015).

However, the body's system of autonomic control is much more complicated than this. Some stress-related symptoms are parasympathetic, not sympathetic. These include symptoms of fatigue, fainting, gastric hyperacidity, diarrhea, asthma, hypersexuality, and overeating. In some cases, the predominant stress response is parasympathetic. For example, some people with blood phobias faint when exposed to the sight of blood (Engel, 1978), although there is some evidence that this is an exaggerated parasympathetic rebound reaction after initial sympathetic arousal (Dahllöf & Öst, 1998; Ritz, Meuret,

& Simon, 2013). Most organisms, including people, freeze or faint when exposed to severe, life-threatening, and unavoidable stress, although it also has been hypothesized that such freezing occurs when a threat is at a distance and alertness to external stimuli is needed, as well as in individuals who have been traumatized, perhaps having experienced unavoidable severe stress (Niermann, Figner, & Roelofs, 2017). Some people tend to be more parasympathetically tuned, such that they more readily show a parasympathetic rather than a sympathetic response when exposed even to more minor stressors. This is consistent with observations of the "orienting reflex" (Graham & Clifton, 1966), in which deceleration in heart rate is associated with greater perceptual acuity when the organism is stimulated to "take in" environmental information. There is some evidence that this reflex is characteristic of people with some parasympathetically mediated or activated disorders. We have found this in our own research on asthma, in which constriction of the smooth muscles in the bronchi is mediated by parasympathetic activity and in which exposure to some laboratory stressors produces a parasympathetic response along with bronchoconstriction, whereas other people tend to respond with sympathetic arousal and/or inhibition of parasympathetic activity (Feldman, Lehrer, Hochron, & Schwartz, 2002).

Adding to the complexity of parasympathetic involvement in the stress response is the phenomenon of parasympathetic rebound. As the great physiologist Ernst Gellhorn observed in his studies of the hypothalamus, the activating components of autonomic activity, which he termed *ergotropic,* tended to activate *reactivity* in the opposing system, which he called *trophotropic,* at the same time as the two systems tended to inhibit effects of the other (Gellhorn, 1959, 1967, 1968). Thus, although sympathetic arousal may inhibit some parasympathetic functions (digestion, relaxation, etc.), parasympathetic functions become more hair-triggered. Thus, after sympathetic arousal subsides, parasympathetic symptoms often break out. These can include such common events as postexamination fatigue in students, nocturnal asthma or gastrointestinal symptoms, hunger, and increased sexual desire after sympathetic activity has suddenly decreased. We have found that following a period of muscle relaxation, parasympathetic activity increases in asthma patients, including a tendency to parasympathetic bronchoconstriction (Lehrer et al., 1997), suggesting that muscle relaxation might not be a good recommendation during an acute asthma attack. Sometimes the reactivity is so powerful that it overwhelms the inhibitory effects of sympathetic arousal, such that both sympathetically and parasympathetically mediated events may occur simultaneously, such as diarrhea, hunger, asthma, fatigue, or even sexual arousal occurring during a period of intense stress (DeGood & Williams, 1982; Harrison, Jones, Hughes, & LeFevre, 2013; Lazarus & Mayne, 1990; Lee et al., 2016; Mandell, 2017; Manto, 1969; Overmier, Murison, Ursin, & Skoglund, 1987). In some cases, the parasympathetic component in the stress response may facilitate the "play dead" response to overwhelming stress described above.

Just as parasympathetic arousal is not always "good" in the context of stress management, sympathetic arousal is not always "bad." It is important for producing feelings of vigor and energy after waking up, for maintaining muscle tone, and for maintaining sufficient blood pressure and proper function of all organs that the sympathetic system innervates. Thus to think of stress management as directed at reducing sympathetic arousal and/or increasing parasympathetic arousal is an oversimplification. The important concepts here, then, are *regulation* and its opposite, *dysregulation.* The body is composed of multiple systems that maintain a proper balance, allow us to respond to various external demands, and return to a healthy and asymptomatic state. These systems regulate the body. When symptoms occur, the body is *dysregulated.* The target, then, of stress management is dysregulation: how to prevent and remedy it.

THE CYBERNETICS OF REGULATION AND DYSREGULATION

In discussing regulation and dysregulation, it is useful to draw on some cybernetic concepts, often more in the province of systems engineers than of psychologists and psychophysiologists. Here we review concepts of *control systems*, positive and negative *feedback loops*, and both *open* and *closed* system control (Lehrer & Eddie, 2013).

A *system* is defined as an entity that has its own characteristics, independently of its component parts. Thus the cardiovascular system is more than cardiac output and blood pressure fluctuations, just as the norms by which family members relate to each other are built of more than the sum of personality types of individual family members, and as cell behavior differs from component chemical processes that guide cellular behavior.

A *control system* is a system that contains various internal processes that keep the system operating properly, even when various environmental perturbations may act to change or destroy system function. Thus various guidance systems have mechanisms to maintain a predetermined course of an aircraft, and gasoline supply to modern motor engines is regulated based on the engine's need for fuel. The body has hundreds of control systems. The reciprocal relationship between the sympathetic and parasympathetic systems is just one of them. Others control almost all aspects of psychological and physiological function—for example, sleep–wake cycles (Fisher, Foster, & Peirson, 2013); respiration (Aittokallio, Gyllenberg, Polo, & Virkki, 2007; Guz, 1997; Krimsky & Leiter, 2005); hunger cycles (Blundell et al., 2012; Cheung, Ko, Chow, & Kong, 2018; Mithieux, 2013; Read, 1992); immune system fluctuations (Hirayama & Okita, 2000; Wong & Germain, 2017); cardiovascular system control (Batzel & Bachar, 2010; Mainardi, Bianchi, & Cerutti, 2002); and even social system control (Bhatti & Channabasavanna, 1979; Daniels, Krakauer, & Flack, 2017; Geist, 1986; Michener, 1987; Wright & Meyer, 1978). The healthy individual shows sympathetic or parasympathetic dominance where appropriate: hunger and satiety, elevated or depressed immune system function, variations in types of social interactions among friends, associates, and lovers.

Heart rate variability is a quintessential example of a multiplicity of control systems in the body. Chaos in the pattern of heart rate variability is a strong correlate of resilience (Karavirta et al., 2013; Lefebvre, Goodings, Kamath, & Fallen, 1993; Li & Yuan, 2008; Poon, 1999; Wayne et al., 2013). This is understandable if we think of *chaos* not as random variability, but as reflecting the action of many control systems in the cardiovascular system, all acting simultaneously with differing frequency characteristics. With more control systems as "backups" for each other, the greater the resilience should be for the cardiovascular system as a whole.

Control systems are usually made up of multiple *negative feedback loops*. A negative feedback loop exists when one process modulates variability in another. The body has hundreds of them. One example is the baroreflex. This reflex controls blood pressure (Eckberg & Sleight, 1992) and, indirectly, through brainstem projections to the limbic system, emotional reactivity (Henderson et al., 2004; Mather & Thayer, 2018). The baroreflex acts through stretch receptors in the aorta and carotid arteries. When blood pressure rises, the baroreflex acts to decrease heart rate and expand the blood vessels. The opposite occurs when blood pressure falls. The two loops (heart rate and vascular tone) function with different frequency characteristics (Vaschillo, Lehrer, Rishe, & Konstantinov, 2002; Vaschillo, Vaschillo, Buckman, Pandina, & Bates, 2012), thus contributing to the chaotic nature of heart rate variability. Both, however, help maintain blood pressure at a healthy level. Stimulation and strengthening of the baroreflex is the primary mechanism underlying the effects of heart rate variability biofeedback (Chapter 10 of this volume by Lehrer). Such negative feedback loops occur from the cellular level to the social systems level, helping to provide stability and modulating change in such a way that the systems do not disintegrate. They maintain body temperature, mood, various personality characteristics, marriages, and even whole societies.

This description of negative feedback loops assumes that the system is entirely self-maintaining and does not need external help to maintain stability. A *closed loop* system does not depend on external influences in order to work. In cases of dysregulation, in which physiology, emotions, behavior, and so forth start acting maladaptively, closed loops do not appear to be adequate to maintain system stability. That is when some outside help is needed. Some of this help can come from various sources of social and material support or various forms of environmental stimulation. For example the baroreflex is exercised by the presence of gravity and effects of normal exercise on the baroreflex system. Advice, insight, and social support can help people to act more adaptively and correct fluctuations in mood or anxiety. Sometimes special stress management techniques are needed. This book describes many ways in which various empirically validated stress management methods, from relaxation exercises to cognitive restructuring, can help to restore stability. Systems that habitually rely on such external influences for stability are called *open loop* systems. Most of human behavior is regulated by open loop as well as closed loop systems, which work synchronously to adapt to environmental demands while preserving internal integrity (Lehrer & Eddie, 2013). All of us, therefore, can make good use of the methods described in this book.

Positive feedback loops also have their place in maintaining stability (Avendano, Leidy, & Pedraza, 2013; Cinquin & Demongeot, 2002; Lehrer & Eddie, 2013). In these loops, activity in the system generates more of the same activity, rather than limiting it. Thus, when we exercise or are under threat, the sympathetic nervous system activates, and various parts of the system (e.g., muscle tension) act to further increase this arousal. When we are anxious about one thing, we often start worrying about other things as well, thus increasing our level of alertness—an adaptive strategy in times of danger. But when dysregulation occurs, these positive feedback loops can lead to tension-related physical problems and emotional disorders. Relaxation can have a modulatory effect through an open loop process. Thus the rationale for "progressive" muscle relaxation is that generalization sometimes does occur from relaxation in one muscle area to others (a positive feedback loop), and general muscle relaxation lowers sympathetic arousal (see McGuigan and Lehrer, Chapter 7, this volume). On the other hand, prolonged lack of exercise, as often happens in illness, can lead to fatigue, disability, and atrophy of reflexes needed for healthy physiological and emotional regulation.

A systems perspective thus lends some perspective to the role of stress in health and illness. Although too much stress can overload the body's ability to adapt, a certain amount of stress may be necessary to promote healthy adaptation. Only by exposure to difficulties do we develop skills to manage them. Only by responding to stressors do the reflexes that modulate stress become exercised and tuned. Positive feedback loops may exaggerate various stress responses, but they also exercise negative feedback loops that control these responses. Cannon's early description of homeostasis appeared in a book entitled *The Wisdom of the Body* (1932). True wisdom requires an endless capacity for complexity.

ALLOSTASIS AND ALLOSTATIC OVERLOAD

Most theories of stress and stress management have been framed by Cannon's concept of homeostasis. The various control systems in the body are designed to keep the body in a constant state. When stressors occur, negative feedback loops act to restore functioning to a constant resting level. This concept was simultaneously introduced by the physiologist Claude Bernard as the *milieu intérieur* (Bernard, 1974). The problem with the notion of homeostasis is that it does not describe healthy functioning. If heart rate did not rise when we were undergoing a period of prolonged exercise, we would not function efficiently and might not even survive. Decreased heart rate variability is associated with a variety of somatic and emotional diseases. In very impaired individuals or in the at-risk fetus is a negative predictor of survival (see Lehrer, Chapter 10, this volume). Heart rate must be able to change in response to constantly changing environmental demand. Even in personality structure, we would consider constancy in emotional state to be a liability, as a sign of rigidity. Healthy people are sometimes happy and sometimes sad, sometimes suspicious and sometimes trusting, sometimes angry and sometimes calm. An individual whose baseline level is only one of these could easily fit into one of the DSM categories of mental illness.

Add to this a cardinal characteristic of negative feedback loops: oscillation (Cinquin & Demongeot, 2002). When sympathetic arousal gets too high, parasympathetic arousal brings it down. When parasympathetic activity is too active, sympathetic activity emerges. This has a certain rhythm. Each of these autonomic branches also has its own multiple internal rhythms, including diurnal rhythms, monthly rhythms, seasonal rhythms, and so forth (Haim, Downs, & Raman, 2001; Lo et al., 2017; Varga & Heck, 2017). Heart rate and brain functions oscillate in intervals varying from milliseconds to minutes. So do moods and rhythms in relationships, with varying mathematical characteristics (Gottman, Murray, Swanson, Tyson, & Swanson, 2002).

This characteristic has been widely studied for heart rate. Heart rate variability is depressed in almost all physical and emotional illnesses, as well as in older age (Corino, Matteucci, & Mainardi, 2007; Mahinrad et al., 2016), in young infants (Eyre, Duncan, Birch, & Fisher, 2014; Jewell, Suk, & Luecken, 2018; Samper Villagrasa, Ventura Faci, Fabre Gonzalez, Bescos Pison, & Perez Gonzalez, 1989; Vigo, Guinjoan, Scaramal, Siri, & Cardinali, 2005), and in other conditions, such as illness, in which diminished resilience might be expected (Buchman, Stein, & Goldstein, 2002). It is elevated in people who are aerobically more fit (Alderman & Olson, 2014; Kaikkonen et al., 2014). Recent research has found that people are able to increase their heart rate variability through various voluntary control exercises, as in heart rate variability biofeedback (see Lehrer, Chapter 10, this volume). Heart rate variability also has been found to increase after CBT (Carney et al., 2000; Jang, Hwang, Padhye, & Meininger, 2017; Kim, Lim, Chung, & Woo, 2009), after various relaxation strategies described in this book (Huang, Hsieh, & Lai, 2016; McKenna, Gallagher, Forbes, & Ibeziako, 2015; Nijjar et al., 2014; Pal, Ganesh, Karthik, Nanda, & Pal, 2014; Wang, Dong, & Li, 2014), and after increased aerobic exercise (Castello et al., 2011; Dougherty, Glenny, & Kudenchuk, 2008; Marquis et al., 2008; Pigozzi et al., 2001; Raimundo et al., 2013; Shen & Wen, 2013), particularly where improvement in symptoms also occurs.

So, then, healthy stability is more characterized by organized *variability* rather than constancy, both in the resting state and in natural everyday life. This is the nature of allostasis (Karatsoreos & McEwen, 2011; McEwen, 2004, 2017). *Allostasis* connotes "stability through variability." Allostatic control requires adequate functioning of the various reflexes that mediate the many negative and positive feedback loops in the body. Although a moderate amount of exercise of these reflexes may help maintain their tone (thus illustrating how a moderate amount of stress might actually be good for us), too much strain on them can fatigue them and decrease their effectiveness. McEwen uses this concept in describing *allostatic overload,* in which the individual is faced with stress that is too great or too prolonged for the various negative control loop reflexes to function properly (Karatsoreos & McEwen, 2011; McEwen & Wingfield, 2003). These are the conditions in which various symptoms of stress appear, including disorders in mood, behavior, autonomic stability, and inflammation. As almost all body and emotional systems are controlled by regulatory processes, the failure of regulation that occurs with allostatic overload can be considered "dysregulation." Dysregulation, or allostatic overload, can occur because of severe and prolonged stress but also, in combination with these, by other impediments to effective regulation. These can include almost any form of physical disease, a physical or emotional predisposition to experience certain symptoms in response to minor stressors or challenges, or lack of resources or skills to manage the challenges that do occur. By definition, then, when dysregulation occurs, the individual experiences signs or symptoms of illness, impairment in functioning, or impaired quality of life due to discomfort, weakness, and other problems. Dysregulation is the hallmark of functional disorders, and stress is often implicated in producing it.

A variety of somatic diseases involve components of autonomic dysregulation. In the lungs, asthma (airway constriction); in the gut, irritable bowel syndrome (constipation or diarrhea) or chronic hyperacidity; in the cardiovascular system (hypertension or hypotension, fainting, vascular inflammation, heart attack, and more). When one branch of the autonomic nervous system becomes overactive and responses to stimulation are not sufficiently modulated, then symptoms occur. Healthy autonomic regulation is therefore demonstrated in a complex pattern of variability, reflecting the operation of multiple negative and positive feedback loops, often leading to a chaotic pattern of fluctuations. As mentioned above, *chaotic* does not mean "random"—randomness is a sign of disorganization, illness, and dysregulation. A chaotic pattern is a deterministic pattern governed by a complicated set of rules, which often can be described only by complicated combinations of nonlinear statistics, often characterized as "entropy" measures. For example, high degree of heart rate entropy (a measure of chaos in heart rate) predicts fetal survival in high-risk pregnancies (Ahearne, Boylan, & Murray, 2016; Frusca, Todros, Lees, Bilardo, & TRUFFLE Investigators, 2018), as well as survival in serious disease (Arzeno, Kearney, Eckberg, Nolan, & Poon, 2007; Halberg et al., 2000). A very simple pattern of heart rate variability, conversely, also indicates pathology, when one or two control reflexes predominate but others are inoperable.

Immune Dysregulation and Autoimmune Processes

The most influential 20th-century view of stress effects was proposed by Hans Selye, who likened the stress response to the body's response against invasive microbes (Selye, 1978). Stress is thus considered as a conditional reflex, designed to protect the body in anticipation of violation, perhaps in combat. This would be characterized by a heightened immune and inflammatory response, in which the body marshals resources to neutralize an invader (cf. Kusnecov and colleagues, Chapter 4, this volume), as well as, perhaps, a corticosteroid response that may be activated to modulate the inflammatory response when the threat is not great or prolonged, a reaction that can *depress* the immune reaction. Thus immune system dysregulation can be in the form of either a depressed or an overactive immune response. Cohen, Tyrrell, and Smith (1991) found that people experiencing psychological stress were more likely to catch cold when experimentally exposed to a rhinovirus than those reporting less stress. An overly active immune system could trigger an autoimmune response, as in allergy and asthma (Li et al., 2013; Miyasaka, Dobashi-Okuyama, Takahashi, Takayanagi, & Ohno, 2018; Miyasaka et al., 2016; Ohno, 2017). Interestingly, some epigenetic research has found a higher incidence of allergies and asthma among children whose mothers experienced severe environmental stress during pregnancy, showing that these tendencies may be carried on to future generations (Trump et al., 2016; Wright, 2007), where the bodies of offspring exhibit the preparatory response to threat of injury.

Social Systems Dysregulation

Social systems also tend to have regular and stable ways of acting. Much of social behavior reflects the influence of social norms that keep people behaving in expected ways. Sometimes normative regulations inflict some discomfort in individuals, as in family systems that include acceptance of abuse and societal systems that tolerate inequality and poverty. Disruptive behavior or severe stressors can make this discomfort intolerable and lead people to challenge social norms. Then the structure of the social system may change or even fall apart, as in divorce, internal warfare, or separatism. Patterns of interaction in marriage have been modeled mathematically (Gottman et al., 2002), and marital therapists are advised to help find alternative patterns that are already in a couple's repertoire and normative structure (Gottman & Gottman, 2008). With sufficient love and desire to maintain the marriage, some disagreements and discomforts can be overlooked in order to keep the marriage intact (Gottman & Gottman, 2017). Other social forces can even prevent therapeutic changes in social structures. When one person changes, as, for example, when one person in the family becomes less emotionally reactive or more assertive, others may get upset and sometimes act to restore the previous way of behaving. As in the famous *All in the Family* television show, attempts by Edith or the grown children to take on new, more assertive, and less conformative roles can be met with strong resistance from Archie, whose rampages are sometimes, but not always, successful in moderating some of the changes. Sometimes one person's becoming more relaxed and less reactive to stress may meet resistance in some quarters and often must be addressed in family therapy. Of course individuals' behavior and norms within the family and the culture at large do change, as demonstrated in the changing roles of women and minorities over the generations in most Western countries. This usually requires great and persistent effort on the part of social activists, or therapists. Although this volume does not include a chapter on social systems intervention, practitioners are wise to keep social control forces in mind as they implement other stress management methods.

Respiratory Dysregulation

A common form of stress-induced physiological dysregulation is in the respiratory system. The respiratory system is extraordinarily sensitive to demands on the body, both physical and emotional, actual or anticipated. When a real or anticipated demand for increased physical exercise or mental activity occurs, respiratory drive increases (Houtveen, Rietveld, & de Geus, 2002). This can be measured as the force of inhalation with each breath, as well as diaphragmatic muscle activity during inhalation (Garland, Doshi, & Turcanu, 2015; Kay, 1979; MacBean, Hughes, Nicol, Reilly, & Rafferty, 2016; Reilly et al., 2013; Reiterer & Muller, 2003). When increases in ventilation are matched with increased metabolic rate, then the oxygen–carbon dioxide balance in the body is maintained. When these are mismatched, various symptoms of respiratory dysregulation occur often in the form of hyperventilation.

The term *dysfunctional breathing* has been used to describe such patterns of dysfunctional breathing. Dysfunctional breathing can include a pattern of thoracic breathing and general excess muscle tension involved in respiration (Barker & Everard, 2015; Prys-Picard & Niven, 2008), often accompanied by decreased partial pressure of carbon dioxide in the blood. Through several well-known reflexes, this can produce decreased oxygen availability to tissues, which, in turn, can cause dysfunction throughout the body (see Meuret & Ritz, Chapter 11, and van Dixhoorn, Chapter 12, this volume). Although thoracic breathing and blood levels of carbon dioxide often do not correlate well with each other (Courtney, Greenwood, & Cohen, 2011), they both are related to a variety of physical and psychological symptoms. This may reflect an episodic nature for some hyperventilation problems, which may not be present when measured in the laboratory. We know that tension in the muscles of the abdomen, lower back, and pelvis can impede action of the diaphragm in breathing. In natural breathing, the diaphragm moves down toward the lower abdomen, thus producing a partial vacuum in the lung, acting as a passive "balloon" and filling with air when the diaphragm moves down. However, muscle tension in the lower torso can impede movement of the diaphragm. When this happens, "accessory" muscles in the chest and shoulders must be used to create a vacuum in the lung during inhalation. Not only does this increase the work of breathing and general muscle tension, but also, because the skeletal muscles are part of the sympathetic nervous system, the excess muscle tension in the trunk of the body from tension in the lower trunk and compensatory muscular activity in the upper trunk may help a positive feedback loop cascade that increases general sympathetic arousal. In a vicious cycle, increased sympathetic arousal can increase ventilation and the level of hypocapnia that can be promoted by thoracic breathing.

Sighing

We all sigh every few minutes. Several studies have found that sighing may be necessary for proper respiratory regulation (Ramirez, 2014; Vlemincx et al., 2013; Vlemincx, Van Diest, Lehrer, Aubert, & Van den Bergh, 2010). Vlemincx and colleagues found that usually there is a high autocorrelation looking at each breath time compared with the immediately previous one. However, over time this correlation decreases, to the point where the pattern approaches randomness. This appears to trigger a sigh, after which the autocorrelation is restored (Vlemincx et al., 2013; Vlemincx et al., 2010). Perhaps, as in heart rate variability (HRV), the autocorrelation represents the operation of various negative feedback loops controlling the period of breathing. It is known that, in many oscillatory feedback systems, a completely constant oscillation, such as may occur during paced breathing, may deprive the system of information that may occur at other frequencies. Perhaps the sigh provides the necessary periodic variability for respiratory regulation systems to operate efficiently. It also may help reinflate alveoli where air does not sufficiently reach them in usual tidal breathing.

Sighing also contributes to emotional control (Vlemincx, Meulders, & Abelson, 2017; Vlemincx, Van Diest, & Van den Bergh, 2016). Since sighing is often accompanied by a long slow outbreath, it is possible that sighing also may occur as a modulatory maneuver to decrease anxiety. During exhalation, the vagus nerve produces a decrease in heart rate, with general parasympathetic stimulation. We often are told to "take a deep breath" in order to reduce stress. However, a sigh also increases ventilation and may lead to hyperventilation. Increases in sighing occur with sympathetic arousal (Zanella et al., 2014), where the body anticipates a need for increased oxygen intake, and in anxiety disorders (Roth, 2005), in which hyperventilation often occurs.

CONCLUSION

In approaching this volume, we suggest that readers keep in mind the complexity of factors contributing to strain, vulnerability, hardiness, and resilience. Some may be constitutional. Some may result from early upbringing experiences that may make people more or less reactive to various stressors. Buffering factors such as wealth and socioeconomic status (Jewell, Luecken, Gress-Smith, Crnic, & Gonzales, 2015; Johnson, Cavallaro, & Leon, 2017), family and social support (Gleeson, Hsieh, & Cryer-Coupet, 2016; Gradus, Smith, & Vogt, 2015; Leshem, Haj-Yahia, & Guterman, 2016; Levens, Elrahal, & Sagui, 2016; Mansour & Tremblay, 2016), protection from social and natural disasters (Lai, Lewis, Livings, La Greca, & Esnard, 2017; Lee et al., 2017; Raveis, VanDevanter, Kovner, & Gershon, 2017; Rosellini, Dussaillant, Zubizarreta, Kessler, & Rose, 2018), and social skill (Chua & Pachana, 2016; Cote et al., 2017; Patnaik, 2014; Rosen & Perrewé, 2017; Treadway, Campion, & Williams, 2017; Zhang, Liu, Jiang, Wu, & Tian, 2014) all play important roles. However, stress is a universal experience, and almost everyone experiences some symptoms, disabilities, or body dysfunctions because of it at some point in life. Therefore, the methods described here may have some usefulness for everyone.

This book is about ways to improve hardiness, prevent and control dysregulation, and improve allostatic capacity. The various methods described here each work on specific aspects of regulatory systems. In a way, all methods of psychotherapy, health promotion, diet, medical intervention, habit control, and so forth have this aim. Although exposure to certain amounts of stress is part of the human condition and may be necessary to tune various allostatic reflexes, excessive stress causes dysregulation. Here we focus on particular empirically validated physical, psychophysiological, and cognitive interventions that prevent and treat dysregulation. Although the various chapters refer to particular outcomes that have received empirical study, the reader should keep in mind that all body and psychological systems are interrelated and affect each other. Resilience and hardiness are strengthened by practicing effective stress management methods. This book describes the foremost empirically tested stress management methods now in use.

REFERENCES

Abelson, J. L., Neese, R. M., Weg, J. G., & Curtis, G. C. (1996). Respiratory psychophysiology and anxiety: Cognitive intervention in the doxapram model of panic. *Psychosomatic Medicine, 58*(4), 302–313.

Ahearne, C. E., Boylan, G. B., & Murray, D. M. (2016). Short- and long-term prognosis in perinatal asphyxia: An update. *World Journal of Clinical Pediatrics, 5*(1), 67–74.

Ahs, F., Gingnell, M., Furmark, T., & Fredrikson, M. (2017). Within-session effect of repeated stress exposure on extinction circuitry function in social anxiety disorder. *Psychiatry Research, 261,* 85–90.

Aittokallio, T., Gyllenberg, M., Polo, O., & Virkki, A. (2007). Parameter estimation of a respiratory control model from noninvasive carbon dioxide measurements during sleep. *Mathematical Medicine and Biology, 24*(2), 225–249.

Alderman, B. L., & Olson, R. L. (2014). The relation of aerobic fitness to cognitive control and heart rate variability: A neurovisceral integration study. *Biological Psychology, 99,* 26–33.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

American Psychological Association. (2017). Stress in America: The state of our nation. Retrieved from *www.apa.org/news/press/releases/stress/2017/state-nation.pdf.*

American Psychological Association. (2020). *PsycInfo*. Retrieved from *www.apa.org/pubs/databases/psycinfo*.

Arzeno, N. M., Kearney, M. T., Eckberg, D. L., Nolan, J., & Poon, C. S. (2007). Heart rate chaos as a mortality predictor in mild to moderate heart failure. *Twenty-ninth Annual International Conference of the IEEE Engineering in Medicine and Biology Society* (pp. 5051–5054). Piscataway, NJ: IEEE.

Avendano, M. S., Leidy, C., & Pedraza, J. M. (2013). Tuning the range and stability of multiple phenotypic states with coupled positive–negative feedback loops. *Nature Communications, 4,* 2605.

Bali, A., & Jaggi, A. S. (2015). Clinical experimental stress studies: Methods and assessment. *Reviews in the Neurosciences, 26*(5), 555–579.

Barker, N., & Everard, M. L. (2015). Getting to grips with "dysfunctional breathing." *Paediatric Respiratory Reviews, 16*(1), 53–61.

Batzel, J. J., & Bachar, M. (2010). Modeling the cardiovascular–respiratory control system: Data, model analysis, and parameter estimation. *Acta Biotheoretica, 58*(4), 369–380.

Beck, A. T. (2004). Cognitive therapy, behavior therapy, psychoanalysis, and pharmacotherapy: A cognitive continuum. In A. Freeman, M. J. Mahoney, P. Devito, & D. Martin (Eds.), *Cognition and psychotherapy* (2nd ed., pp. 197–220). New York: Springer.

Bernard, C. (1974). *Lectures on the phenomena common to animals and plants* (H. E. Hoff, R. Guillemin, & L. Guillemin, Trans.). Springfield, IL: Charles C Thomas.

Berry, D., Blair, C., Willoughby, M., & Granger, D. A. (2012). Salivary alpha-amylase and cortisol in infancy and toddlerhood: Direct and indirect relations with executive functioning and academic ability in childhood. *Psychoneuroendocrinology, 37*(10), 1700–1711.

Bhatti, R. S., & Channabasavanna, S. (1979). Social system approach to understand marital disharmony. *Indian Journal of Social Work, 40*(1), 79–88.

Blundell, J. E., Caudwell, P., Gibbons, C., Hopkins, M., Naslund, E., King, N., et al. (2012). Role of resting metabolic rate and energy expenditure in hunger and appetite control: A new formulation. *Disease Models and Mechanisms, 5*(5), 608–613.

Braun, S. R., Gregor, B., & Tran, U. S. (2013). Comparing bona fide psychotherapies of depression in adults with two meta-analytical approaches. *PLOS ONE, 8*(6), e68135. Retrieved from *https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0068135*.

Buchman, T. G., Stein, P. K., & Goldstein, B. (2002). Heart rate variability in critical illness and critical care. *Current Opinion in Critical Care, 8*(4), 311–315.

Cannon, W. (1932). *The wisdom of the body.* New York: Norton.

Carney, R. M., Freedland, K. E., Stein, P. K., Skala, J. A., Hoffman, P., & Jaffe, A. S. (2000). Change in heart rate and heart rate variability during treatment for depression in patients with coronary heart disease. *Psychosomatic Medicine, 62*(5), 639–647.

Castello, V., Simoes, R. P., Bassi, D., Catai, A. M., Arena, R., & Borghi-Silva, A. (2011). Impact of aerobic exercise training on heart rate variability and functional capacity in obese women after gastric bypass surgery. *Obesity Surgery, 21*(11), 1739–1749.

Cheung, L. T. F., Ko, G. T. C., Chow, F. C. C., & Kong, A. P. S. (2018). Association between hedonic hunger and glycemic control in non-obese and obese patients with type 2 diabetes. *Journal of Diabetes Investigation, 12,* 12.

Chrousos, G. P., Loriaux, D. L., & Gold, P. W. (1988). Introduction: The concept of stress and its historical development. In G. P. Chrousos, D. L. Loriaux, & P. W. Gold (Eds.), *Mechanisms of physical and emotional stress* (pp. 3–7). Boston: Springer.

Chua, J., & Pachana, N. A. (2016). Use of a psychoeducational skill training DVD program to reduce stress in Chinese Australian and Singaporean dementia caregivers: A pilot study. *Clinical Gerontologist, 39*(1), 3–14.

Chung, M. C., AlQarni, N., Al Muhairi, S., & Mitchell, B. (2017). The relationship between trauma centrality, self-efficacy, posttraumatic stress and psychiatric co-morbidity among Syrian refugees: Is gender a moderator? *Journal of Psychiatric Research, 94,* 107–115.

Cinquin, O., & Demongeot, J. (2002). Roles of positive and negative feedback in biological systems. *Comptes Rendus Biologies, 325*(11), 1085–1095.

Cleghorn, R. A. (1965). Psychosomatic principles. *Canadian Medical Association Journal, 92,* 441–447.

Cohen, B. (1989). Relations among autonomic responding, infant and adult emotions, temperament and personality. *Dissertation Abstracts International, 49*(12-B, Pt. 1), 5540.

Cohen, S., Tyrrell, D. A., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *New England Journal of Medicine, 325*(9), 606–612.

Collins, L. M., & Coles, M. E. (2017). Sudden gains in exposure therapy for obsessive–

compulsive disorder. *Behaviour Research and Therapy, 93,* 1–5.

Corino, V. D., Matteucci, M., & Mainardi, L. T. (2007). Analysis of heart rate variability to predict patient age in a healthy population. *Methods of Information in Medicine, 46*(2), 191–195.

Cote, S. M., Larose, M.-P., Geoffroy, M. C., Laurin, J., Vitaro, F., Tremblay, R. E., et al. (2017). Testing the impact of a social skill training versus waiting list control group for the reduction of disruptive behaviors and stress among preschool children in child care: The study protocol for a cluster randomized trial. *BMC Psychology, 5*(29). Retrieved from *https://bmcpsychology.biomedcentral.com/articles/10.1186/s40359-017-0197-9.*

Courtney, R., Greenwood, K. M., & Cohen, M. (2011). Relationships between measures of dysfunctional breathing in a population with concerns about their breathing. *Journal of Bodywork and Movement Therapies, 15*(1), 24–34.

Da Costa, R. T., Sardinha, A., & Nardi, A. E. (2008). Virtual reality exposure in the treatment of fear of flying. *Aviation, Space, and Environmental Medicine, 79*(9), 899–903.

Dahllöf, O., & Öst, L.-G. (1998). The diphasic reaction in blood phobic situations: Individually or stimulus bound? *Scandinavian Journal of Behaviour Therapy, 27*(3), 97–104.

Daniels, B. C., Krakauer, D. C., & Flack, J. C. (2017). Control of finite critical behaviour in a small-scale social system. *Nature Communications, 8,* 14301.

DeGood, D. E., & Williams, E. M. (1982). Parasympathetic rebound following EMG biofeedback training: A case study. *Biofeedback and Self-Regulation, 7*(4), 461–465.

Delahaij, R., & Van Dam, K. (2017). Coping with acute stress in the military: The influence of coping style, coping self-efficacy and appraisal emotions. *Personality and Individual Differences, 119,* 13–18.

Donegan, E., & Dugas, M. J. (2012). Generalized anxiety disorder: A comparison of symptom change in adults receiving cognitive-behavioral therapy or applied relaxation. *Journal of Consulting and Clinical Psychology, 80*(3), 490–496.

Dougherty, C. M., Glenny, R., & Kudenchuk, P. J. (2008). Aerobic exercise improves fitness and heart rate variability after an implantable cardioverter defibrillator. *Journal of Cardiopulmonary Rehabilitation and Prevention, 28*(5), 307–311.

Eckberg, D. L., & Sleight, P. (1992). *Human baroreflexes in health and disease.* Oxford, UK: Clarendon Press.

Ellis, A. (2005). Why I (really) became a therapist. *Journal of Clinical Psychology, 61*(8), 945–948.

Engel, G. L. (1978). Psychological stress, vasodepressor (vasovagal) syncope, and sudden death. *Annals of Internal Medicine, 89,* 403–412.

Engert, V., Buss, C., Khalili-Mahani, N., Wadiwalla, M., Dedovic, K., & Pruessner, J. C. (2010). Investigating the association between early life parental care and stress responsivity in adulthood. *Developmental Neuropsychology, 35*(5), 570–581.

Eyre, E. L., Duncan, M. J., Birch, S. L., & Fisher, J. P. (2014). The influence of age and weight status on cardiac autonomic control in healthy children: A review. *Autonomic Neuroscience: Basic and Clinical, 186,* 8–21.

Fadirepo, A. A. (2013). Acting skill: A panacea to stress management technique. *IFE Psychologia, 21*(3), 161–166.

Fales, M. (2018). Subjective social status and health: Environmental antecedents and molecular mechanisms. Retrieved from *https://escholarship.org/uc/item/4mh1d6t2.*

Faul, L. A., Jim, H. S., Williams, C., Loftus, L., & Jacobsen, P. B. (2010). Relationship of stress management skill to psychological distress and quality of life in adults with cancer. *Psycho-Oncology, 19*(1), 102–109.

Favaro, A., Tenconi, E., Degortes, D., Manara, R., & Santonastaso, P. (2015). Neural correlates of prenatal stress in young women. *Psychological Medicine, 45*(12), 2533–2543.

Feldman, J. M., Lehrer, P. M., Hochron, S. M., & Schwartz, G. E. (2002). Defensiveness and individual response stereotypy in asthma. *Psychosomatic Medicine, 64*(2), 294–301.

Fisher, S. P., Foster, R. G., & Peirson, S. N. (2013). The circadian control of sleep. In A. Kramer & M. Merrow (Eds.), *Handbook of experimental pharmacology: Vol. 217. Circadian clocks* (pp. 157–183). Heidelberg, Germany: Springer.

Fogelman, N., & Canli, T. (2019). Early life stress, physiology, and genetics: A review. *Frontiers in Psychology, 10,* 1668.

Freud, S., & Eitingon, M. (1927). Concluding remarks on the question of lay analysis. *International Journal of Psychoanalysis, 8,* 392–401.

Frusca, T., Todros, T., Lees, C., Bilardo, C. M., & TRUFFLE Investigators. (2018). Outcome in early-onset fetal growth restriction is best combining computerized fetal heart rate analysis with ductus venosus Doppler: Insights from the trial of umbilical and fetal flow in Europe. *American Journal of Obstetrics and Gynecology, 218*(2S), S783–S789.

Garland, A. J., Doshi, A., & Turcanu, V. (2015). Neural respiratory drive measurement for COPD assessment and monitoring. *Pneumologia, 64*(1), 14–17.

Geist, P. J. (1986). Modeling control in the hospital organization: A social system in transition. Retrieved from *https://docs.lib.purdue.edu/dissertations/AAI8520008*.

Gellhorn, E. (1959). Sympathetic and parasympathetic summations. *Acta Neurovegetativa, 20,* 181–194.

Gellhorn, E. (1967). The tuning of the nervous system: Physiological foundations and implications for behavior. *Perspectives in Biology and Medicine, 10*(4), 559–591.

Gellhorn, E. (1968). Central nervous system tuning and its implications for neuropsychiatry. *Journal of Nervous and Mental Disease, 147*(2), 148–162.

Gilroy, L. J., Kirkby, K. C., Daniels, B. A., Menzies, R. G., & Montgomery, I. M. (2000). Controlled comparison of computer-aided vicarious exposure versus live exposure in the treatment of spider phobia. *Behavior Therapy, 31*(4), 733–744.

Gleeson, J. P., Hsieh, C.-M., & Cryer-Coupet, Q. (2016). Social support, family competence, and informal kinship caregiver parenting stress: The mediating and moderating effects of family resources. *Children and Youth Services Review, 67,* 32–42.

Goodman, S. H., & Brand, S. R. (2009). Depression and early adverse experiences. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 249–274). New York: Guilford Press.

Gottman, J. M., & Gottman, J. S. (2008). Gottman method couple therapy. In A. S. Gurman (Ed.), *Clinical handbook of couple therapy* (4th ed., pp. 138–164). New York: Guilford Press.

Gottman, J., & Gottman, J. (2017). The natural principles of love. *Journal of Family Theory and Review, 9*(1), 7–26.

Gottman, J. M., Murray, J. D., Swanson, C. C., Tyson, R., & Swanson, K. R. (2002). *The mathematics of marriage: Dynamic nonlinear models*. Cambridge, MA: MIT Press.

Gradus, J. L., Smith, B. N., & Vogt, D. (2015). Family support, family stress, and suicidal ideation in a combat-exposed sample of Operation Enduring Freedom/Operation Iraqi Freedom veterans. *Anxiety, Stress and Coping, 28*(6), 706–715.

Graham, F. K., & Clifton, R. K. (1966). Heart rate change as a component of the orienting response. *Psychological Bulletin, 65,* 305–320.

Guillen-Burgos, H. F., & Gutierrez-Ruiz, K. (2018). Genetic advances in post-traumatic stress disorder. *Revista Colombiana de Psiquiatria, 47*(2), 108–118.

Gunnar, M. R., & Vazquez, D. (2006). Stress neurobiology and developmental psychopathology. *Developmental Psychopathology: Developmental Neuroscience* (2nd ed., Vol. 2, pp. 533–577). Hoboken, NJ: Wiley.

Guz, A. (1997). Brain, breathing and breathlessness. *Respiration Physiology, 109*(3), 197–204.

Haim, A., Downs, C. T., & Raman, J. (2001). Effects of adrenergic blockade on the daily rhythms of body temperature and oxygen consumption of the black-tailed tree rat (*Thallomys nigricauda*) maintained under different photoperiods. *Journal of Thermal Biology, 26*(3), 171–177.

Halberg, F., Cornelissen, G., Katinas, G., Watanabe, Y., Otsuka, K., Maggioni, C., et al. (2000). Feedsidewards: Intermodulation (strictly) among time structures, chronomes, in and around us, and cosmo-vasculo-neuroimmunity. About ten-yearly changes: What Galileo missed and Schwabe found. *Annals of the New York Academy of Sciences, 917,* 348–375.

Han, C. (2014). Health implications of socioeconomic characteristics, subjective social status, and perceptions of inequality: An empirical study of China. *Social Indicators Research, 119*(2), 495–514.

Harrison, M. A., Jones, K., Hughes, S. M., & LeFevre, A. J. (2013). Self-reports of nipple erection in emotional and somatic contexts. *Psychological Record, 63*(3), 489–499.

Harvey, P., Harris, R. B., Harris, K. J., & Wheeler, A. R. (2007). Attenuating the effects of social stress: The impact of political skill. *Journal of Occupational Health Psychology, 12*(2), 105–115.

Henderson, L. A., Richard, C. A., Macey, P. M., Runquist, M. L., Yu, P. L., Galons, J. P., et al. (2004). Functional magnetic resonance signal changes in neural structures to baroreceptor reflex activation. *Journal of Applied Physiology, 96*(2), 693–703.

Hirayama, H., & Okita, Y. (2000). A mathematical method for investigating dynamic behavior of an idiotype network of the immune system:

The time minimum optimal control theory. *Pathophysiology, 7*(3), 215–229.

Hofmann, S. G. (1999). The value of psychophysiological data for cognitive behavioral treatment of panic disorder. *Cognitive and Behavioral Practice, 6*(3), 244–248.

Hofmann, S. G., Mundy, E. A., & Curtiss, J. (2015). Neuroenhancement of exposure therapy in anxiety disorders. *AIMS Neuroscience, 2*(3), 123–138.

Hollingshead, A. B., & Redlich, F. C. (1958). *Social class and mental illness*. New York: Wiley.

Hopko, D. R., & Lejuez, C. W. (2007). *A cancer patient's guide to overcoming depression and anxiety: Getting through treatment and getting back to your life*. Oakland, CA: New Harbinger.

Houtveen, J. H., Rietveld, S., & de Geus, E. J. (2002). Contribution of tonic vagal modulation of heart rate, central respiratory drive, respiratory depth, and respiratory frequency to respiratory sinus arrhythmia during mental stress and physical exercise. *Psychophysiology, 39*(4), 427–436.

Hruby, R., Hasto, J., & Minarik, P. (2011). Attachment in integrative neuroscientific perspective. *Neuroendocrinology Letters, 32*(2), 111–120.

Huang, C. Y., Hsieh, Y. M., & Lai, H. L. (2016). Effect of stimulative and sedative music videos on depressive symptoms and physiological relaxation in older adults: A pilot study. *Research in Gerontological Nursing, 9*(5), 233–242.

Jang, A., Hwang, S. K., Padhye, N. S., & Meininger, J. C. (2017). Effects of cognitive behavior therapy on heart rate variability in young females with constipation-predominant irritable bowel syndrome: A parallel-group trial. *Journal of Neurogastroenterology and Motility, 23*(3), 435–445.

Jayasinghe, N., Sparks, M. A., Kato, K., Wilbur, K., Ganz, S. B., Chiaramonte, G. R., et al. (2014). Exposure-based CBT for older adults after fall injury: Description of a manualized, time-limited intervention for anxiety. *Cognitive and Behavioral Practice, 21*(4), 432–445.

Jewell, S. L., Luecken, L. J., Gress-Smith, J., Crnic, K. A., & Gonzales, N. A. (2015). Economic stress and cortisol among postpartum low-income Mexican American women: Buffering influence of family support. *Behavioral Medicine, 41*(3), 138–144.

Jewell, S. L., Suk, H. W., & Luecken, L. J. (2018). Respiratory sinus arrhythmia: Modeling longitudinal change from 6 weeks to 2 years of age among low-income Mexican Americans. *Developmental Psychobiology, 60*, 232–238.

Johnson, S. C., Cavallaro, F. L., & Leon, D. A. (2017). A systematic review of allostatic load in relation to socioeconomic position: Poor fidelity and major inconsistencies in biomarkers employed. *Social Science and Medicine, 192*, 66–73.

Jordet, G., Hartman, E., Visscher, C., & Lemmink, K. A. (2007). Kicks from the penalty mark in soccer: The roles of stress, skill, and fatigue for kick outcomes. *Journal of Sports Sciences, 25*(2), 121–129.

Kaikkonen, K. M., Korpelainen, R. I., Tulppo, M. P., Kaikkonen, H. S., Vanhala, M. L., Kallio, M. A., et al. (2014). Physical activity and aerobic fitness are positively associated with heart rate variability in obese adults. *Journal of Physical Activity and Health, 11*(8), 1614–1621.

Karatsoreos, I. N., & McEwen, B. S. (2011). Psychobiological allostasis: Resistance, resilience and vulnerability. *Trends in Cognitive Sciences, 15*(12), 576–584.

Karavirta, L., Costa, M. D., Goldberger, A. L., Tulppo, M. P., Laaksonen, D. E., Nyman, K., et al. (2013). Heart rate dynamics after combined strength and endurance training in middle-aged women: Heterogeneity of responses. *PLOS ONE, 8*(8), e72664. Retrieved from *https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0072664*.

Kay, B. (1979). The measurement of occlusion pressure during anaesthesia: A comparison of the depression of respiratory drive by methohexitone and etomidate. *Anaesthesia, 34*(6), 543–548.

Kim, W., Lim, S. K., Chung, E. J., & Woo, J. M. (2009). The effect of cognitive behavior therapy-based psychotherapy applied in a forest environment on physiological changes and remission of major depressive disorder. *Psychiatry Investigation, 6*(4), 245–254.

Kimball, C. P. (1983). The biopsychosocial approach: Liaison medicine and its models. *Psychotherapy and Psychosomatics, 40*(1–4), 48–65.

Krimsky, W. R., & Leiter, J. C. (2005). Physiology of breathing and respiratory control during sleep. *Seminars in Respiratory and Critical Care Medicine, 26*(1), 5–12.

Kushner, M. G., Maurer, E. W., Thuras, P., Donahue, C., Frye, B., Menary, K. R., et al. (2013). Hybrid cognitive behavioral therapy versus relaxation training for co-occurring anxiety and alcohol disorder: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 81*(3), 429–442.

Kuzmin, M. Y., & Konopak, I. A. (2016). Distinctive features of adolescent hardiness in families of different composition. *Psychology in Russia: State of the Art, 9*(3), 95–102.

Lai, B. S., Lewis, R., Livings, M. S., La Greca, A. M., & Esnard, A.-M. (2017). Posttraumatic stress symptom trajectories among children after disaster exposure: A review. *Journal of Traumatic Stress, 30*(6), 571–581.

Lang, P. J. (1968). Fear reduction and fear behavior: Problems in treating a construct. In J. M. Schlien (Ed.), *Research in psychotherapy* (pp. 90–102). Washington, DC: American Psychological Association.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16*(6), 495–512.

Lavenda, O., & Kestler-Peleg, M. (2017). Parental self-efficacy mitigates the association between low spousal support and stress. *Psychiatry Research, 256,* 228–230.

Lazarus, A. A., & Mayne, T. J. (1990). Relaxation: Some limitations, side effects, and proposed solutions. *Psychotherapy: Theory, Research, Practice, Training, 27*(2), 261–266.

Lee, A., Hsu, H. H. L., Chiu, Y. H. M., Bose, S., Rosa, M. J., Kloog, I., et al. (2018). Prenatal fine particulate exposure and early childhood asthma: Effect of maternal stress and fetal sex. *Journal of Allergy and Clinical Immunology, 141*(5), 1880–1886.

Lee, E. M., Klement, K. R., Ambler, J. K., Loewald, T., Comber, E. M., Hanson, S. A., et al. (2016). Altered states of consciousness during an extreme ritual. *PLOS ONE, 11*(5), Retrieved from *https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0153126*.

Lee, J.-Y., Kim, S.-W., Bae, K.-Y., Kim, J.-M., Shin, I.-S., & Yoon, J.-S. (2017). Factors associated with post-traumatic stress symptoms among adolescents exposed to the Sewol ferry disaster in Korea. *Psychiatry Research, 256,* 391–395.

Lefebvre, J. H., Goodings, D. A., Kamath, M. V., & Fallen, E. L. (1993). Predictability of normal heart rhythms and deterministic chaos. *Chaos, 3*(2), 267–276.

Lehrer, P. M., & Eddie, D. (2013). Dynamic processes in regulation and some implications for biofeedback and biobehavioral interventions. *Applied Psychophysiology and Biofeedback, 38*(2), 143–155.

Lehrer, P. M., Hochron, S. M., Mayne, T., Isenberg, S., Lasoski, A. M., Carlson, V., et al. (1997). Relationship between changes in EMG and respiratory sinus arrhythmia in a study of relaxation therapy for asthma. *Applied Psychophysiology and Biofeedback, 22*(3), 183–191.

Lehrer, P. M., Kaur, K., Sgobba, P., Versella, M., Yang, M.-J., Sharma, A., et al. (2017, March). *Meta-analytic review of mind–body interventions.* Paper presented at the annual meeting of the Association for Applied Psychophysiology and Biofeedback, Chicago, IL.

Leshem, B., Haj-Yahia, M. M., & Guterman, N. B. (2016). The role of family and teacher support in post-traumatic stress symptoms among Palestinian adolescents exposed to community violence. *Journal of Child and Family Studies, 25*(2), 488–502.

Levens, S. M., Elrahal, F., & Sagui, S. J. (2016). The role of family support and perceived stress reactivity in predicting depression in college freshman. *Journal of Social and Clinical Psychology, 35*(4), 342–355.

Li, B., Duan, X. H., Wu, J. F., Liu, B. J., Luo, Q. L., Jin, H. L., et al. (2013). Impact of psychosocial stress on airway inflammation and its mechanism in a murine model of allergic asthma. *Chinese Medical Journal, 126*(2), 325–334.

Li, B. B., & Yuan, Z. F. (2008). Non-linear and chaos characteristics of heart sound time series. *Proceedings of the Institution of Mechanical Engineers: Part H. Journal of Engineering in Medicine, 222*(3), 265–272.

Lo, M. T., Bandin, C., Yang, H. W., Scheer, F., Hu, K., & Garaulet, M. (2017). CLOCK 3111T/C genetic variant influences the daily rhythm of autonomic nervous function: Relevance to body weight control. *International Journal of Obesity, 24,* 24.

Luceno-Moreno, L., Garcia-Albuerne, Y., Talavera-Velasco, B., & Martin-Garcia, J. (2016). Stress in Spanish police force depending on occupational rank, sex, age and work-shift. *Psicothema, 28*(4), 389–393.

Lundgren, J., Carlsson, S. G., & Berggren, U. (2006). Relaxation versus cognitive therapies for dental fear: A psychophysiological approach. *Health Psychology, 25*(3), 267–273.

MacBean, V., Hughes, C., Nicol, G., Reilly, C. C., & Rafferty, G. F. (2016). Measurement of neural respiratory drive via parasternal intercostal electromyography in healthy adult subjects. *Physiological Measurement, 37*(11), 2050–2063.

Maddi, S. R. (2017). Hardiness as a pathway to resilience under stress. In U. Kumar (Ed.), *The Routledge international handbook of psychosocial resilience* (pp. 104–110). New York: Routledge/Taylor & Francis.

Maddi, S. R., Matthews, M. D., Kelly, D. R., Villarreal, B. J., Gundersen, K. K., & Savino, S. C. (2017). The continuing role of hardiness and grit on performance and retention in West Point cadets. *Military Psychology, 29*(5), 355–358.

Magnus, M. C., Wright, R. J., Roysamb, E., Parr, C. L., Karlstad, O., Page, C. M., et al. (2018). Association of maternal psychosocial stress with increased risk of asthma development in offspring. *American Journal of Epidemiology, 187*(6), 1199–1209.

Mahinrad, S., Jukema, J. W., van Heemst, D., Macfarlane, P. W., Clark, E. N., de Craen, A. J., et al. (2016). 10-second heart rate variability and cognitive function in old age. *Neurology, 86*(12), 1120–1127.

Mainardi, L. T., Bianchi, A. M., & Cerutti, S. (2002). Time-frequency and time-varying analysis for assessing the dynamic responses of cardiovascular control. *Critical Reviews in Biomedical Engineering, 30*(1–3), 175–217.

Mandell, D. (2017). Autonomic stress recovery and habituation in migraine. Retrieved from *https://search.proquest.com/openview/c4baada0a44fa7c301bacf3d7ac2d5c3/1?pq-origsite=gscholar&cbl=18750&diss=y*.

Mansour, S., & Tremblay, D.-G. (2016). Workload, generic and work–family specific social supports and job stress: Mediating role of work–family and family–work conflict. *International Journal of Contemporary Hospitality Management, 28*(8), 1778–1804.

Manto, P. G., Jr. (1969). An investigation of feedback mechanisms in stress-induced changes of autonomic balance. *Dissertation Abstracts International, 29*(7-B), 2637.

Marquis, K., Maltais, F., Lacasse, Y., Lacourciere, Y., Fortin, C., & Poirier, P. (2008). Effects of aerobic exercise training and irbesartan on blood pressure and heart rate variability in patients with chronic obstructive pulmonary disease. *Canadian Respiratory Journal, 15*(7), 355–360.

Martins, L., & Lopes, C. (2012). Military hierarchy, job stress and mental health in peacetime. *Occupational Medicine, 62*(3), 182–187.

Mather, M., & Thayer, J. (2018). How heart rate variability affects emotion regulation brain networks. *Current Opinion in Behavioral Sciences, 19*, 98–104.

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: Allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences, 1032*, 1–7.

McEwen, B. S. (2017). Allostasis and the epigenetics of brain and body health over the life course: The brain on stress. *JAMA Psychiatry, 74*(6), 551–552.

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior, 43*(1), 2–15.

McGuire, J. F., Lewin, A. B., Geller, D. A., Brown, A., Ramsey, K., Mutch, J., et al. (2012). Advances in the treatment of pediatric obsessive–compulsive d-cycloserine with exposure and response prevention. *Neuropsychiatry, 2*(4).

McKenna, K., Gallagher, K. A., Forbes, P. W., & Ibeziako, P. (2015). Ready, set, relax: Biofeedback-assisted relaxation training (BART) in a pediatric psychiatry consultation service. *Psychosomatics, 56*(4), 381–389.

McVicar, A., Ravalier, J. M., & Greenwood, C. (2014). Biology of stress revisited: Intracellular mechanisms and the conceptualization of stress. *Stress and Health, 30*(4), 272–279.

Menard, C., Pfau, M. L., Hodes, G. E., & Russo, S. J. (2017). Immune and neuroendocrine mechanisms of stress vulnerability and resilience. *Neuropsychopharmacology, 42*(1), 62–80.

Merton, R. (1938). Social structure and anomie. *American Sociological Review, 3*(5), 672–682.

Michelson, L., Mavissakalian, M. R., Marchione, K., Ulrich, R. F., Marchione, N., & Testa, S. (1990). Psychophysiological outcome of cognitive, behavioral and psychophysiologically-based treatments of agoraphobia. *Behaviour Research and Therapy, 28*(2), 127–139.

Michener, H. A. (1987). Control, conflict, and crisis management in the Space Station's social system (year 2000). In T. B. Sheridan, D. S. Kruser, & S. Deutsch (Eds.), *Human factors in automated and robotic space systems: Proceedings of a symposium* (pp. 356–389). Washington, DC: National Research Council.

Mithieux, G. (2013). Nutrient control of hunger by extrinsic gastrointestinal neurons. *Trends in Endocrinology and Metabolism, 24*(8), 378–384.

Miyasaka, T., Okuyama-Dobashi, K., Masuda, C., Iwami, S., Sato, M., Mizoguchi, H., et al. (2016). The involvement of central nervous system histamine receptors in psychological stress-induced exacerbation of allergic airway inflammation in mice. *Allergology International, 65* (Suppl.), S38–S44.

Miyasaka, T., Dobashi-Okuyama, K., Takahashi, T., Takayanagi, M., & Ohno, I. (2018).

The interplay between neuroendocrine activity and psychological stress-induced exacerbation of allergic asthma. *Allergology International, 67*(1), 32–42.

National Library of Medicine. (2020). *Medline*. Retrieved from *www.nlm.nih.gov/bsd/medline.html*.

Niermann, H. C., Figner, B., & Roelofs, K. (2017). Individual differences in defensive stress-responses: The potential relevance for psychopathology. *Current Opinion in Behavioral Sciences, 14,* 94–101.

Nijjar, P. S., Puppala, V. K., Dickinson, O., Duval, S., Duprez, D., Kreitzer, M. J., et al. (2014). Modulation of the autonomic nervous system assessed through heart rate variability by a mindfulness-based stress reduction program. *International Journal of Cardiology, 177*(2), 557–559.

Norton, P. J. (2012). A randomized clinical trial of transdiagnostic cognitive-behavioral treatments for anxiety disorder by comparison to relaxation training. *Behavior Therapy, 43*(3), 506–517.

Ohno, I. (2017). Neuropsychiatry phenotype in asthma: Psychological stress-induced alterations of the neuroendocrine–immune system in allergic airway inflammation. *Allergology International, 66*(Suppl.), S2–S8.

Oken, B. S., Chamine, I., & Wakeland, W. (2015). A systems approach to stress, stressors and resilience in humans. *Behavioural Brain Research, 282,* 144–154.

Otto, M. W., Hearon, B. A., & Safren, S. A. (2010). Mechanisms of action in the treatment of social anxiety disorder. In S. G. Hofmann & P. M. DiBartolo (Eds.), *Social anxiety: Clinical, developmental, and social perspectives* (2nd ed., pp. 577–598). San Diego, CA: Elsevier.

Overmier, J., Murison, R., Ursin, H., & Skoglund, E. J. (1987). Quality of poststressor rest influences the ulcerative process. *Behavioral Neuroscience, 101*(2), 246–253.

Padmanabhan, D. (2017). From distress to disease: A critique of the medicalisation of possession in DSM-5. *Anthropology and Medicine, 24*(3), 261–275.

Pal, G. K., Ganesh, V., Karthik, S., Nanda, N., & Pal, P. (2014). The effects of short-term relaxation therapy on indices of heart rate variability and blood pressure in young adults. *American Journal of Health Promotion, 29*(1), 23–28.

Parkash, V., Archana, & Kumar, U. (2017). Role of genetics and temperament in resilience. In U. Kumar (Ed.), *The Routledge international handbook of psychosocial resilience* (pp. 75–87). New York: Routledge/Taylor & Francis.

Patnaik, G. (2014). Life skill enhancement strategies to minimize stress. *Social Science International, 30*(2), 281–289.

Pearlin, L. I. (1969). The sociological study of stress. *Journal of Health and Social Behaviors, 30*(3), 241–256.

Pigozzi, F., Alabiso, A., Parisi, A., Di Salvo, V., Di Luigi, L., Spataro, A., et al. (2001). Effects of aerobic exercise training on 24-hr profile of heart rate variability in female athletes. *Journal of Sports Medicine and Physical Fitness, 41*(1), 101–107.

Pitts, B. L., Safer, M. A., Russell, D. W., & Castro-Chapman, P. L. (2016). Effects of hardiness and years of military service on post-traumatic stress symptoms in U.S. Army medics. *Military Psychology, 28*(4), 278–284.

Poon, C. S. (1999). Cardiac chaos: Implications for congestive heart failure. *Congestive Heart Failure, 5*(6), 270–274.

Porcelli, P., Laera, D., Mastrangelo, D., & Di Masi, A. (2012). Prevalence of allostatic overload syndrome in patients with chronic cardiovascular disease. *Psychotherapy and Psychosomatics, 81*(6), 375–377.

President, M. (2017). Spiritual transcendence and subjective social status: Resources African American professional women utilize in the pursuit of worklife balance. *Dissertation Abstracts International, 78*(2-B(E)).

Prys-Picard, C. O., & Niven, R. (2008). Dysfunctional breathing in patients with asthma. *Thorax, 63*(6), 568.

Raby, K. L. (2016). Early interpersonal antecedents of physiological reactivity in adult romantic relationships. *Dissertation Abstracts International, 76*(11-B(E)).

Raimundo, R. D., de Abreu, L. C., Adami, F., Vanderlei, F. M., de Carvalho, T. D., Moreno, I. L., et al. (2013). Heart rate variability in stroke patients submitted to an acute bout of aerobic exercise. *Translational Stroke Research, 4*(5), 488–499.

Ramirez, J. M. (2014). The integrative role of the sigh in psychology, physiology, pathology, and neurobiology. *Progress in Brain Research, 209,* 91–129.

Ramnero, J., Folke, F., & Kanter, J. W. (2016). A learning theory account of depression. *Scandinavian Journal of Psychology, 57*(1), 73–82.

Raveis, V. H., VanDevanter, N., Kovner, C. T., & Gershon, R. (2017). Enabling a disaster-resilient workforce: Attending to individual stress and collective trauma. *Journal of Nursing Scholarship, 49*(6), 653–660.

Read, N. W. (1992). Role of gastrointestinal factors in hunger and satiety in man. *Proceedings of the Nutrition Society, 51*(1), 7–11.

Reilly, C. C., Jolley, C. J., Ward, K., MacBean, V., Moxham, J., & Rafferty, G. F. (2013). Neural respiratory drive measured during inspiratory threshold loading and acute hypercapnia in healthy individuals. *Experimental Physiology, 98*(7), 1190–1198.

Reiterer, F., & Muller, W. (2003). Assessment of the single-occlusion technique for measurements of respiratory mechanics and respiratory drive in healthy term neonates using a commercially available computerized pulmonary function testing system. *Biology of the Neonate, 83*(2), 117–122.

Ritz, T., Meuret, A. E., & Simon, E. (2013). Cardiovascular activity in blood-injection-injury phobia during exposure: Evidence for diphasic response patterns? *Behaviour Research and Therapy, 51,* 460–468.

Rosellini, A. J., Dussaillant, F., Zubizarreta, J. R., Kessler, R. C., & Rose, S. (2018). Predicting posttraumatic stress disorder following a natural disaster. *Journal of Psychiatric Research, 96,* 15–22.

Rosen, C. C., & Perrewé, P. L. (Eds.). (2017). *Research in occupational stress and well-being: Vol. 15. Power, politics, and political skill in job stress.* Bingley, UK: Emerald.

Roth, W. T. (2005). Physiological markers for anxiety: Panic disorder and phobias. *International Journal of Psychophysiology, 58,* 190–198.

Sabik, N. J., Falat, J., & Magagnos, J. (2020). When self-worth depends on social media feedback: Associations with psychological well-being. *Sex Roles, 82,* 411–421.

Samper Villagrasa, M. P., Ventura Faci, M. P., Fabre Gonzalez, E., Bescos Pison, J. L., & Perez Gonzalez, J. (1989). [Quantification of heart rate variability in newborn infants and during the first 6 months of age]. *Anales Espanoles de Pediatria, 31*(3), 196–200.

Scott, K. M., Al-Hamzawi, A. O., Andrade, L. H., Borges, G., Caldas-de-Almeida, J. M., Fiestas, F., et al. (2014). Associations between subjective social status and DSM-IV mental disorders: Results from the world mental health surveys. *JAMA Psychiatry, 71*(12), 1400–1408.

Selye, H. (1978). *The stress of life* (rev. ed.). Oxford, UK: McGraw-Hill.

Shen, T. W., & Wen, H. J. (2013). Aerobic exercise affects T-wave alternans and heart rate variability in postmenopausal women. *International Journal of Sports Medicine, 34*(12), 1099–1105.

Shirom, A., & Mayer, A. (1993). Stress and strain among union lay officials and rank-and-file members. *Journal of Organizational Behavior, 14*(5), 401–413.

Spiegler, M. D., & Guevremont, D. C. (2016). *Contemporary behavior therapy.* Boston: Cengage Learning.

Srole, L., Langner, T. S., Michael, S. T., Oplear, M. K., & Rennie, T. A. C. (1962). *Mental health in the metropolis: The Midtown Manhattan study.* New York: McGraw-Hill.

Stoppelbein, L., McRae, E., & Greening, L. (2017). A longitudinal study of hardiness as a buffer for posttraumatic stress symptoms in mothers of children with cancer. *Clinical Practice in Pediatric Psychology, 5*(2), 149–160.

Thompson, J., Haber, J. D., & Tearnan, B. H. (1981). Generalization of frontalis electromyographic feedback to adjacent muscle groups: A critical review. *Psychosomatic Medicine, 43*(1), 19–24.

Treadway, D. C., Campion, E. D., & Williams, L. V. (2017). Sensitivity and adaptability in the face of powerlessness: The roles of political will and political skill within the experience of powerlessness and its impact on stress-related outcomes. In C. C. Rosen & P. L. Perrewé (Eds.), *Research in occupational stress and well-being: Vol. 15. Power, politics, and political skill in job stress* (pp. 81–103). Bingley, UK: Emerald.

Troesch, L. M., & Bauer, C. E. (2017). Second career teachers: Job satisfaction, job stress, and the role of self-efficacy. *Teaching and Teacher Education, 67,* 389–398.

Trump, S., Bieg, M., Gu, Z., Thurmann, L., Bauer, T., Bauer, M., et al. (2016). Prenatal maternal stress and wheeze in children: Novel insights into epigenetic regulation. *Scientific Reports, 6,* 28616.

Uchida, S., Hara, K., Kobayashi, A., Funato, H., Hobara, T., Otsuki, K., et al. (2010). Early life stress enhances behavioral vulnerability to stress through the activation of REST4-mediated gene transcription in the medial prefrontal cortex of rodents. *Journal of Neuroscience, 30*(45), 15007–15018.

van den Bosch, G. E., Dijk, M. V., Tibboel, D., & de Graaff, J. C. (2017). Long-term effects of early exposure to stress, pain, opioids and anaesthetics on pain sensitivity and neurocognition. *Current Pharmaceutical Design, 23*(38), 5879–5886.

Varga, S., & Heck, D. H. (2017). Rhythms of the body, rhythms of the brain: Respiration, neural oscillations, and embodied cognition. *Consciousness and Cognition, 56,* 77–90.

Vaschillo, E. G., Lehrer, P. M., Rishe, N., & Konstantinov, M. (2002). Heart rate variability biofeedback as a method for assessing baroreflex function: A preliminary study of resonance in the cardiovascular system. *Applied Psychophysiology and Biofeedback, 27*(1), 1–27.

Vaschillo, E. G., Vaschillo, B., Buckman, J. F., Pandina, R. J., & Bates, M. E. (2012). Measurement of vascular tone and stroke volume baroreflex gain. *Psychophysiology, 49*(2), 193–197.

Vigo, D. E., Guinjoan, S. M., Scaramal, M., Siri, L. N., & Cardinali, D. P. (2005). Wavelet transform shows age-related changes of heart rate variability within independent frequency components. *Autonomic Neuroscience: Basic and Clinical, 123*(1–2), 94–100.

Vlemincx, E., Abelson, J. L., Lehrer, P. M., Davenport, P. W., Van Diest, I., & Van den Bergh, O. (2013). Respiratory variability and sighing: A psychophysiological reset model. *Biological Psychology, 93*(1), 24–32.

Vlemincx, E., Meulders, M., & Abelson, J. L. (2017). Sigh rate during emotional transitions: More evidence for a sigh of relief. *Biological Psychology, 125,* 163–172.

Vlemincx, E., Van Diest, I., Lehrer, P. M., Aubert, A. E., & Van den Bergh, O. (2010). Respiratory variability preceding and following sighs: A resetter hypothesis. *Biological Psychology, 84*(1), 82–87.

Vlemincx, E., Van Diest, I., & Van den Bergh, O. (2016). A sigh of relief or a sigh to relieve: The psychological and physiological relief effect of deep breaths. *Physiology and Behavior, 165,* 127–135.

Voisey, J., Young, R. M., Lawford, B. R., & Morris, C. P. (2014). Progress towards understanding the genetics of posttraumatic stress disorder. *Journal of Anxiety Disorders, 28*(8), 873–883.

Wagner, N., Mills-Koonce, R., Willoughby, M., Propper, C., Rehder, P., & Gueron-Sela, N. (2017). Respiratory sinus arrhythmia and heart period in infancy as correlates of later oppositional defiant and callous–unemotional behaviors. *International Journal of Behavioral Development, 41*(1), 127–135.

Wang, Y., Dong, Y., & Li, Y. (2014). Perioperative psychological and music interventions in elderly patients undergoing spinal anesthesia: Effect on anxiety, heart rate variability, and postoperative pain. *Yonsei Medical Journal, 55*(4), 1101–1105.

Wayne, P. M., Manor, B., Novak, V., Costa, M. D., Hausdorff, J. M., Goldberger, A. L., et al. (2013). A systems biology approach to studying tai chi, physiological complexity and healthy aging: Design and rationale of a pragmatic randomized controlled trial. *Contemporary Clinical Trials, 34*(1), 21–34.

Williams, M. T., Crozier, M., & Powers, M. (2011). Treatment of sexual-orientation obsessions in obsessive–compulsive disorder using exposure and ritual prevention. *Clinical Case Studies, 10*(1), 53–66.

Wong, H. S., & Germain, R. N. (2017). Robust control of the adaptive immune system. *Seminars in Immunology, 29,* 29.

Wright, K. N., & Meyer, P. B. (1978). Corrections, deviance, and social system stability. *Behavioral Science, 23*(3), 148–156.

Wright, R. J. (2007). Prenatal maternal stress and early caregiving experiences: Implications for childhood asthma risk. *Paediatric and Perinatal Epidemiology, 21*(Suppl. 3), 8–14.

Wright, R. J., Mitchell, H., Visness, C. M., Cohen, S., Stout, J., Evans, R., et al. (2004). Community violence and asthma morbidity: The Inner-City Asthma Study. *American Journal of Public Health, 94*(4), 625–632.

Zanella, S., Doi, A., Garcia, A. J., III, Elsen, F., Kirsch, S., Wei, A. D., et al. (2014). When norepinephrine becomes a driver of breathing irregularities: How intermittent hypoxia fundamentally alters the modulatory response of the respiratory network. *Journal of Neuroscience, 34*(1), 36–50.

Zeer, E. F., Yugova, E. A., Karpova, N. P., & Trubetskaya, O. V. (2016). Psychological predictors of human hardiness formation. *International Journal of Environmental and Science Education, 11*(14), 7035–7044.

Zhang, W., Liu, H., Jiang, X., Wu, D., & Tian, Y. (2014). A longitudinal study of posttraumatic stress disorder symptoms and its relationship with coping skill and locus of control in adolescents after an earthquake in China. *PLOS ONE, 9*(2). Retrieved from *https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0088263.*

CHAPTER 3

The Psychology of Relaxation, Meditation, and Mindfulness

An Introduction to RMM Theory, Practice, and Assessment

Jonathan C. Smith

People relax in many ways and for many reasons. Some might take nature walks, listen to music, or contemplate sacred scriptures. Others may practice yoga, breathing, progressive muscle relaxation, meditation, or biofeedback. My concern is with *self-relaxation,* activities and exercises one can do by oneself, without the necessity of external equipment (biofeedback) or personal assistance (massage). This chapter does not consider active, goal-directed leisure and recreational activities such as sports, reading, art, music, television, nature walks, and the like. All may well have profoundly relaxing effects, but they are not pure forms of self-relaxation.

But what is relaxation and how does it work? As can be seen in this volume, various physiological and neurological perspectives offer great insight. In this chapter I describe my psychological perspective. To begin, I find it useful to consider relaxation as part of a larger construct: relaxation/meditation/mindfulness (RMM). Exercises defined as relaxation by most texts and training manuals (Smith, 1985, 1986, 1990, 1999, 2001, 2005, 2017, 2019) include the following: progressive muscle relaxation, yoga and tai chi, breathing exercises, autogenic training, and visualization/imagery. My definition is based on popular use; *relaxation* is what most professionals call relaxation. To this list we can add *meditation* and *mindfulness. Meditation* can be defined generically as sustaining quiet, simple focus (Smith, 2017, 2019). In focused attention (FA) meditation (Lutz, Jha, Dunne, & Saron, 2015), one restricts attention to a singular stimulus, such as a mantra or the flow of breath. Mindfulness (OM, or open monitoring) involves quietly attending to the flow of all stimuli (or restricted domain of stimuli, such as sounds) as a neutral observer.

For decades I have argued that it is misleading to view relaxation, meditation, and mindfulness as separate approaches or phenomena. There is a bit of relaxation in all of meditation and mindfulness. There is a bit of meditation and mindfulness in all of relaxation. Nearly all texts and training programs teach blends of RMM. Although different techniques clearly have a different pattern of effects, all can be placed on the same psychological map. All can evoke experiences from the same lexicon (Smith, 2017, 2019).

NEUROPHYSIOLOGICAL PERSPECTIVES

For over a century, neurophysiological perspectives have dominated the study of RMM. In the 1840s, surgeon James Braid (1853) introduced the term *neurohypnotism* (sleep of the nerves). The term stuck over the decades in its current incarnation, *hypnosis*. In the 20th century, Jacobson (1929) and Schultz (1932) introduced progressive muscle relaxation and autogenic training as methods for moderating neuromuscular and autonomic processes. In the 1970s Benson (1975) popularized the "relaxation response" as the mirror opposite of the nonspecific stress "fight-or-flight" response, mediated by sympathetic nervous system arousal. Benson theorized that such arousal reduction underlies all approaches to RMM. More recently, meditation and mindfulness researchers (Goleman & Davidson, 2017) have focused on brain structures (notably the prefrontal cortex, amygdala, and posterior cingulate) and activity patterns (high-amplitude EEG gamma).

Neurophysiological models of RMM have served us well and continue to form the primary rationale in training. These models are accessible and credible to trainees. Abundant research shows diverse positive health and performance outcomes. Body- and brain-based models have lifted techniques from the darkness of religion, pseudoscience, and the occult to genuine professional credibility. Indeed, the current flood of interest in meditation, mindfulness, and yoga can be traced to brain imaging research at the turn of the millennium.

BEYOND THE BODY AND BRAIN: RMM THEORY

An exclusive emphasis on neurophysiology risks missing something important (Smith, 1990). Most practitioners of RMM appear to master arousal-reducing skills in a month or so, yet many go on to practice for years and decades. Often masters of meditation and yoga claim to progress deeper in their practice even after a lifetime of practice, even after brain changes are no longer detectable. Clearly, such individuals are discovering something more than a reduced metabolic rate or alterations in brain structure and functioning.

As is apparent in this volume, psychological theory forms the basis of other forms of stress management. Learning theory underlies desensitization. Cognitive psychology provides the foundation for cognitive therapy, stress inoculation training, and exposure treatments. Constructs from social psychology are central to anger management and conflict resolution. The list goes on. In contrast, RMM has been viewed more similarly to exercise, diet, or psychopharmacology—defined by what happens in the body. But for practitioners of relaxation, more is going on than can be measured in the body or brain.

Two decades ago (Smith, 1985, 1986), I proposed the beginnings of what I now call *RMM theory* (alternatively named, "Third-Generation Mindfulness theory"; Smith, 2017, 2019), a comprehensive integration of all approaches to relaxation, meditation, and mindfulness (Smith, 2005). My approach is not narrow-spectrum; it does not focus on a homogenous, static outcome state or trait (e.g., the relaxation response, focused awareness, or nonjudgmental acceptance). Instead, my approach is broad-spectrum and based on four ideas: (1) RMM has many defining effects, (2) these effects inform and influence each other, (3) they change over time, and (4) this change is not random or circular but evolves in a direction that is decreasingly self-referential and increasingly deep and encompassing. Simply, my broad-spectrum model is multidimensional, interactive, dynamic, and directional (for an elaboration, see Smith, 2017, 2019). In this chapter, I introduce the highlights of the latest version of RMM theory and review implications for research, practice, and assessment.

ON ASKING THE RIGHT QUESTION: NARROW-SPECTRUM AND BROAD-SPECTRUM APPROACHES

RMM theory begins with a simple question remarkably few trainers or researchers ask: *What do you experience when you relax, meditate, or practice mindfulness?* If you ask students of progressive muscle relaxation, yoga stretching, tai chi, breathing exercises, autogenic training, imagery, meditation, or mindfulness a narrow-spectrum question (*Are your muscles relaxed?*), you will likely get a narrow-spectrum answer that articulates a single state or trait (*I am still tense* or *I am relaxed*). However, if your question is broad-spectrum and open-ended, you will quickly encounter a rainbow of psychological states. Some feel *peaceful* or *rested*. Others feel *sleepy* and *far away,* or even *energized, loving, thankful,* and *joyful*. And practitioners of strictly secular approaches may have deeply spiritual feelings.

If you make it clear that you are listening with genuine openness, sincerity, and interest, your student may trust you enough to share a personal story of RMM. Consider these journal excerpts of a practitioner of progressive muscle relaxation:

> I am trying muscle relaxation to reduce my backache . . . my exercises are beginning to work and my pain is less . . . I can more readily detect tension and let go . . . My muscles feel pleasantly loose and relaxed. . . . I feel still and quiet, less distracted by preoccupying nagging muscle pain and worry about my body. . . . I can focus more easily. . . . I'm curious about meditation and am learning to treat my muscle relaxation as a mindfulness exercise. . . . I find myself being a neutral observer and view my occasional pain and all body sensations as passing thoughts that do not define me . . . sometimes I have brief but remarkable experiences . . . with sustained undistracted focus I view the world as a beautiful dance of which I am a part . . . I am filled with joy, awe and wonder, and even a touch of reverence. . . . I am part of something greater . . . I am thankful for this experience.

Note that this is indeed a training outcome. But it is not a narrow-spectrum homogeneous static state or trait (*My pain is less).* Our client has shared a story. As such, it is broad-spectrum: multidimensional, interactive, dynamic, and directional. A truly complete assessment of any approach to relaxation, meditation, and mindfulness should examine the kinds of stories that emerge, how they are similar and different, what conditions influence their development, and how they affect life's pains, problems, and challenges. No single questionnaire, brain scan, or behavioral task assessment can reveal such a living portrait. To systematically and scientifically explore the stories of RMM, we need to go beyond practitioner anecdotal reports or trainer dogmas and preconceptions. To do this, we need a standard, empirically based vocabulary of analysis, a comparative template, a lexicon of RMM.

AN RMM LEXICON

Since 1996 (Smith, Amutio, Anderson, & Aria, 1996), I have embarked on something of a quest for a comprehensive natural language or lexicon of RMM. Along with students and colleagues, our mission has been to identify those words practitioners actually use to describe their experiences. We began by studying the basic instructional texts of progressive muscle relaxation, yoga, tai chi, breathing, autogenic training, visualization/imagery, prayer, contemplation, meditation, and mindfulness. This search included ancient approaches as well as those that are new, approaches from the East and West, and approaches from spiritual and secular traditions. I reasoned that such manuals were likely to include words accessible to and used by actual practitioners, rather than the technical terms of scientists or the esoteric musings of mystics, philosophers, and theologians. I came up with an initial dictionary of more than 200 words.

Over the years, my colleagues and I have used two tools for screening and organizing our lexicon. First, we applied factor analysis to successively screen item lists. This 20-year effort involved more than 6,000 participants who have practiced more than 40 types of RMM exercises and activities (Borgogna & Smith, 2016; Smith, 1986, 1999, 2001, 2005, 2012; Smith et al., 1996). Second, we carefully surveyed the results from practitioner focus and training groups.

Factor Analyses

Factor analysis is an ideal tool for identifying which word items are accessible and which are not. Factors, of course, are groups of interrelated items. Factors show how many diverse items may be grouped and structured. Equally important, the loading of an item on a factor is often its correlation with the factor. An item that loads highly on a factor shares much of the factor's variance. For example, the words *at ease, peace, contented, rested, soothed, laid back, relaxed,* and *calm* consistently form a single RMM factor and can be presumed to measure the same thing. The highest loading words are *at ease* and *peace*. These are the best words for describing what the remaining words may depict. We can delete the remaining words on our initial list, confident that *at ease* and *peace* do the best job of communicating their general meaning. There are many reasons why some word items emerge as highly loading. I speculate that such items may be most readily understood and have a clearly differentiated meaning. Such words contribute to something of the natural language of RMM.

But factor analysis can be particularly tricky. I have found that the factor structure of RMM states varies according to practice populations. Advanced Buddhist monks may display a characteristic factor structure different from that of patient practitioners of progressive muscle relaxation, dieters practicing hatha yoga, or college freshmen after a college psychology class. This does not mean that RMM has no reliable factor structure, but that the factor structure of RMM is one important characteristic of the RMM population being studied. Rather than conclude that our study of Buddhist meditators does not replicate the factor structure found among practitioners of yoga, the proper conclusion should be that our study of meditators suggests a factor structure that may be unique to meditators, one different from that displayed by practitioners of progressive muscle relaxation. This is a direct challenge to several dozen factor-analytic comparative studies of RMM techniques (discussed later in this chapter).

Focus and Training Groups

Over the past 50 years, my students and I have taught more than 150 classes of more than 4,000 students. Each student learns and practices a complete menu of exercises, usually including yoga, tai chi, breathing exercises, muscle relaxation, autogenic self-suggestion, imagery, mantra meditation, and mindfulness (Smith, 2017). We attempt to teach relatively pure versions so students can more readily identify their unique effects. Generally, students are taught all techniques, spending 2 or 3 weeks on each. Students complete a version of an RMM word list questionnaire (the "RMM Tracker"; see *http://blogs.roosevelt.edu/jsmith*) to track progress and make technique comparisons.

My approach is egalitarian. Traditional RMM is typically authoritarian. An expert, leader, or guru presents his or her wisdom and techniques. Students passively listen and practice. In an egalitarian approach, the instructor is more of a guide, and both student and instructor are travelers along a path. Of course, the instructor has concepts and techniques to share. But each student has a unique set of training experiences and insights. Importantly, the learning atmosphere is one in which student and teacher instruct one another. Training manuals for authoritarian approaches tend to remain unchanged for years, decades, centuries, and even millennia. This is true for both approaches based on religious dogma as well as those that claim to be secular and evidence based. In contrast, an egalitarian approach to training is constantly evolving, continuously informed by the insights of student instructors. Last year's training manual may differ significantly from today's version.

In my version of egalitarian training, I make a point of asking students to describe their experiences. Each week I conduct focus groups in which students explain how RMM words fit their experience.

THE "5 + 1" MODEL

From these sources of data I have constructed the "5 + 1" model, a list of five levels and one category of 25 self-report RMM (Smith, 2017, 2019) states. Some reflect factors. Some reflect my experiences as a trainer. Following is a brief summary of what I describe more extensively elsewhere (Smith, 2017, 2019).

Level 1: Basic Relaxation (Alternatively Mindful Basic Relaxation)

For students of meditation or mindfulness (first, second, and third-generation; Smith, 2019), this level can be termed *Mindful Basic Relaxation*. Basic Relaxation is most frequently reported by beginning and intermediate practitioners of all approaches to RMM. I identify six components:

- ***RMM 1 (Far Away, Disengaged).*** Often, beginners in RMM discover the value of simply getting away from (or letting go of) the day's stressors. I define this as RMM 1 (Far Away, Disengaged), in which one feels *distant, far away, indifferent* to cares and concerns, and nicely *detached* from the surrounding world. Sometimes one loses awareness of the source of instructions or parts of the body. Such descriptors define a unified factor group, defined by the words *far away* and *disengaged*. In classes, RMM 1 (Far Away, Disengaged) is the most preferred and frequently reported state for students under high levels of anxiety or distress. This has been something of a revelation for me. I had thought that a stressed-out student would seek RMM to experience muscle relaxation, peace, or mindfulness. Instead, they seek RMM 1 (Far Away, Disengaged). Apparently, for them, RMM is getting away from the hassles of life.

- ***RMM 2 (Physically Relaxed).*** Perhaps the most direct psychological manifestation of reduced autonomic arousal can be seen in the self-report of physical relaxation, that is, practitioners' reduced autonomic arousal and muscle tension, as well as increased relaxed breathing. This RMM state can be experienced in various ways. Muscles may feel *warm, heavy,* or *tingling.* Breathing becomes *even* and *effortless.* Of the nearly two dozen words individuals use to describe such physiological states, the prime descriptor is *physically relaxed,* our title for RMM 2. This state often correlates highly with RMM 1.

- ***RMM 3 (At Ease, At Peace; Mentally Relaxed).*** Psychologically, stress is often described by such words as *frustration, pain, worry, fear, concern,* or *conflict.* When distress is reduced, we feel RMM 3 (At Ease, At Peace, or, alternatively, Mentally Relaxed). Here, factor analysis reflects semantic content analysis.

  RMM 3 is associated with how we deal with stressors. Someone hiding from a tornado may experience fear. When the threat is gone, a person feels *relief.* Or, a person hasn't eaten for hours. She goes to the refrigerator and finds a tasty sandwich. Hunger turns to *satisfaction.* A worker is having an argument with his boss over workload. He experiences conflict. The boss agrees with the worker's schedule requests. The conflict is resolved, and the worker is *at peace.*

  The dictionary reveals that such experiences have one thing in common. They all reflect negative reinforcement, relief from psychological distress, whether it be fear, strain, craving, conflict, or pain. Interestingly, such words consistently clump into a factor group, RMM 3, defined by the highest loading items At Ease/At Peace. Distress, whether it be tension, pain, or negative emotion, can be a barrier to growth in RMM, perhaps suggesting unfinished business requiring attention. A yoga student may be distracted by the fear that he left his home stove on. He needs to resolve this fear first, call home, and have someone turn the stove off. If a practitioner of progressive muscle relaxation is distracted by hunger, a banana can be simple solution. If one is stuck in meditation because of an unresolved home conflict, perhaps the conflict should be actively dealt with.

- ***RMM 4 (Refreshed).*** One type of mental relaxation that occasionally emerges as a distinct factor is the simple feeling of being *refreshed* and *energized*—RMM 4 (Refreshed). In terms of negative reinforcement, *refreshed* can be viewed as the reduction of fatigue.

- ***RMM 5 (Pleasant Mind Wandering).*** Mind wandering is often noted as technique distraction by trainers of meditation, mindfulness, yoga, and occasionally autogenic training. However, in my classes I have observed that when students practice RMM, often they report simply letting go of deliberately planning and doing things and enjoying the pleasures of undirected fantasy and random mind wandering. RMM 5 (Pleasant Mind Wandering) may well be an RMM state in and of itself, a sign that RMM training is working. Perhaps it represents an important RMM process. It is a state worth exploring.

- ***RMM 6 (Fantasy, Daydreaming).*** Practitioners of cognitive approaches to relaxation often report a positive type of mind wandering, fantasy, and daydreaming that is somewhat directed, perhaps with a plot or story (unlike RMM 5). Typically, we find RMM

6 does not load on any prominent RMM factor, suggesting it may not merit status as an RMM state. Rarely do students note it as a sign that an RMM technique is working. I include this state because it merits further exploration.

Level 2: Basic Quiet Focus (Alternatively Mindful Quiet Focus)

In addition to Basic Relaxation, most practitioners of RMM disciplines experience a degree of Basic Quiet Focus. Professionals might argue that Level 2 states more accurately depict meditation and mindfulness rather than relaxation techniques such as progressive muscle relaxation, yoga, and visualization/imagery. I disagree. First, my research and experience consistently show that even practitioners of ordinary professional relaxation techniques (such as progressive muscle relaxation) experience Level 2 as much as Level 1. Indeed, as I describe later, RMM research on the independence of Levels 1 and 2 has yielded inconsistent findings. Even Edmund Jacobson (1929), no friend of Eastern meditative approaches, noted that reports of mental *quiet* often followed training in the progressive muscle relaxation technique he popularized. Mental quiet, as we shall see, is a Level 2 RMM state.

To elaborate, the core act of meditation and mindfulness involves sustaining quiet simple focus. I believe this is a component of all techniques popularly and professionally presented as RMM. For progressive muscle relaxation, one sustains focus on the sensations of releasing tension, actively letting go of potentially distracting tension. In hatha yoga, one sustains focus on achieving and sustaining a posture. Any distracting mind wandering would disrupt focus and the yoga pose. Level 2 states can be an intensely rewarding, reinforcing technique practice. Our research and student experiences suggest a natural language of five Level 2 RMM States:

- ***RMM 7 (Focus, Absorption).*** Attention is fully directed to a target stimulus or task, engaged to the exclusion of competing stimuli. The targeted task can be the primary focus of any RMM exercise (yoga, progressive muscle relaxation, breathing, prayer, meditation, mindfulness, tai chi, imagery, autogenic training, and visualization/imagery, meditation, mindfulness).
- ***RMM 8 (Centered, Grounded).*** The emphasis of RMM 7 (Focus, Absorption) is an exercise target stimulus or task (one is *focused* on or *absorbed* in). For RMM 8 (Centered, Grounded) the emphasis is the accompanying general physical and mental state of the one doing the focusing and absorbing. RMM 7 is more about the object of attention, whereas RMM 8 reflects the attending subject. *Centered* and *grounded,* one may feel more in the *present moment* or *here and now*. Synonyms may include *whole, complete*, and *stable*. One may feel like a rock or tree firmly planted in the ground. *Centered* and *grounded* may have a proprioceptive component of physical stability and poise. To summarize once again, one experiencing RMM 7 may say *I feel focused on and absorbed in the flow of breath,* whereas one experiencing RMM 8 may say *I am centered and grounded in the present*. Generally, this definition is consistent with how centering has been used historically. Indeed, it reflects the definition that appears in the APA Dictionary of Psychology (*https://dictionary.apa.org/centering*). Confusingly, the word *centered* has been used by cognitive therapists to denote nearly the opposite, reification of or fusion with stressful cognitions. One is preoccupied, identifying with negative beliefs and assumptions concerning oneself and the world (Hanley et al., 2020; Safran & Segal, 1990).

- ***RMM 9 (Quiet).*** One experiences a reduction of thought and emotional activity. The mind is *quiet* and *still*. Even feelings related to other RMM states may be absent. For example, a quiet mind may not feel particularly Far Away (RMM 1), Physically Relaxed (RMM 2), At Ease (RMM 3), or At Peace (RMM 3). Put differently, the opposite of inner peace may well be inner conflict. It may also be inner quiet.
- ***RMM 10 (Unbothered).*** One is *accepting*. Negative thoughts or feelings might emerge; however, one is not "caught up" in them. They may be seen as simple thoughts rather than final realities. Thoughts are less distracting or impervious to control. They are less "sticky."
- ***RMM 11 (Easy, Effortless).*** It is easy to let go of mind wandering and distraction, return to task, and sustain focus. It is easy to *let things be, accept what is,* and go on. The task at hand, whether it be relaxation, meditation, mindfulness, or even work or recreation, feels *effortless*.

Level 3: Awakening (Mindful Awakening)

Level 3 is an extension of Level 2. *Awakening* refers to an increased awareness of oneself and the world, a consequence of sustained focus and attention and reduced self-referential thinking. We can consider four RMM states:

- ***RMM 12 (Observer).*** Here one simply stands aside and watches things come and go, as a neutral and objective witness.
- ***RMM 13 (Clear, Awake, Aware).*** As an observer, one may have a sense of experiencing things as they really are. Things may seem vivid or particularly real. One's mantra may cease to be a mechanical chant, becoming a sound with a life and direction of its own. A prayer may become more than a verbal repetition, more like words to and from God. The flow of the present moment may be seen clearly, as for the first time, perhaps as seen by a child.
- ***RMM 14 (Interested, Curious, Fascinated).*** When one is *interested, curious,* or *fascinated* by a task, whether it be progressive muscle relaxation, yoga, meditation, or mindfulness, one is more than a neutral observer experiencing a stimulus vividly as really real. An important new dimension is added: There is more than first appeared. The deeper reality of breath is more than the inflow and outflow of air. The deeper reality of the mantra is not just a repeated sound or syllable. The reality of the present moment is more than a series of events. What is this *more*? Emergence of this realization is depicted in the experience of RMM 14 (Interested, Curious, and Fascinated).
- ***RMM 15 (Beautiful).*** One experiences one important additional feature of the object of one's interest, curiosity, and fascination. Things seem *beautiful, harmonious*. Practitioners from the full range of RMM approaches report such experiences.

Level 4: Deepening (Mindful Deepening)

In Level 3, one opens up to various stimuli beyond a self-referential perspective. In Level 4, another feature emerges. Whether the focal task is a tension release, a simple stretch, pose, breath, image, mantra, or flow of stimuli, it no longer evokes a static state but one that is dynamic, that changes and evolves in a way that is experienced as increasingly deep and encompassing. I speculate at least four RMM states:

- ***RMM 16 (Going Deeper).*** Things are unexpected, new, interesting. One has a sense that experiences are *changing, opening up, being revealed*. It may feel like one is in *a different place or space*.
- ***RMM 17 (Spaciousness, Expansiveness).*** One has a sense of *spaciousness* and *expansiveness*.
- ***RMM 18 (Sense of Something Greater).*** One may feel the sense of *something greater than oneself* (God, a higher power, spirit, energy, love, or consciousness). If religiously inclined, you may feel that God is with you.
- ***RMM 19 (Meaning, Purpose, Direction).***

Level 5: Transcendence (Mindful Transformation/Transcendence)

In rare and special moments, the practitioner may come in touch with the deeper side of life. Again, in my experiences as a trainer and researcher, these can occur in all approaches to RMM, even those ordinarily thought of as strictly physical and secular. Transcendent states reflect awareness of a world larger or greater than oneself. These are often noted in the literature on mysticism and transcendence. Our research identifies the following:

- ***RMM 20 (Reverent, Prayerful).*** Feelings of *reverence* and *prayerfulness* reflect an emotional response to something larger or greater than oneself. These are expressive states, coming from oneself. One *reveres,* one *prays*. It is important to note that feelings of reverence and prayerfulness do not require belief in any supernatural deity or paranormal entity or dimension.
- ***RMM 21 (Awe/Wonder, Deep Mystery).*** Although RMM 21 states Awe/Wonder and Deep Mystery frequently load on the same factor, it is instructive to consider their differences. Awe and Wonder reflects a nonanalytical and goalless awareness of a larger and greater reality that is new, awesome, beyond ordinary familiar comprehension and expectations. It is a reality that is beyond description, beyond words. Our language provides many phrases that convey this notion: *shock of the new, blinding truth, dumbstruck, speechless, far out, mind-blowing, knocks one's socks off,* or simply *Wow!* or *Amazing!* However expressed, one's adult, verbal, analytic thinking cap has been knocked askew; one is temporarily freed or released from self-referential or automatic habitual conceptualizations and one sees things anew.

  Deep Mystery is somewhat familiar to most people. We all have discovered things we do not understand. Sometimes we encounter profound questions and mysteries that seem to transcend any possibility of understanding.

  There is a subtle difference between Awe/Wonder and Deep Mystery. Awe/Wonder suggests we simply do not have the words to describe what we experience. Deep Mystery implies we do not understand it. We may understand the geology of the Grand Canyon but experience it with awe and wonder. We may have words to describe the complex constellations in the sky but recognize the mysteries of the expanding universe, the Big Bang, dark matter, and so on.

- ***RMM 22 (Spiritual, Mystical).*** Practitioners of all approaches can experience moments of profound and personal meaningful experience—a sudden awakening or insight. These can have several facets:

    - Feelings of an underlying hidden truth. One might feel as if one has special and important insightful and intuitive knowledge. There is a sense of certainty of encounter with ultimate reality, a sense of seeing or knowing what is really real.
    - Feelings of being *at one* with the universe or others, a sense of selflessness.
    - Difficulty in describing or communicating one's experience of truth, ultimate reality, oneness, or selflessness to others.

I loosely define transcendent experiences as a profound reduction in self-referential thinking and increased awareness of something larger or greater than oneself. This experience may be dualistic in that it includes awareness of oneself and of something other ("I feel so small when gazing at the immense Grand Canyon"). At extreme levels, it can be nondualistic in that the sense of self dissolves or merges with the world, and all is seen as timeless, eternal, or *at one*. This conceptualization is somewhat similar to what Yaden, Haidt, Hood, Vago, and Newberg (2017) termed *self-transcendence*: "the subjective sense of one's self as an isolated entity can temporarily fade into an experience of unity with other people or one's surroundings, involving the dissolution of boundaries between the sense of self and *other*" (p. 143).

Such experiences can emerge in any RMM exercise. At times, they provide a defining or guiding context. One can argue that, like some positive emotions (explained later), they enhance singular focus, reduce self-referential mind wandering, and provide motivation to practice.

Maslow is noted for providing long, unorganized lists of related "peak experiences" (Maslow, 1964). More systematic accounts can be found in the scientific literature of mystical states (Maclean, Leoutsakos, Johnson, & Griffiths, 2012; Pahnke, 1963, 1969; Stace, 1960). This literature articulates the following facets:

- Unity (oneness, internal and external merging of self)
- Transcendence of time and space
- Noetic quality (deep understanding of hidden mysteries)
- Sacredness
- Positive mood (which we differentiate as a separate RMM category, Transcendent Positive Emotion)
- Ineffability

Transcendent Positive Emotion (Mindful Transcendent Positive Emotion)

Transcendent positive emotions involve reduced self-referential thinking. In contrast, feelings of pride, self-control, personal power, superiority, conquest, success, ownership, domination, and so on may be positive, but they highlight or enhance one's sense of self. For transcendent positive emotions, one's experience of self becomes secondary. Such emotions can emerge in RMM and help one sustain simple focus, reduce needless effort and judgment, and let go of self-referential thinking and mind wandering. Our factor analytic research differentiates three:

- ***RMM 23 (Happy, Optimistic, Trusting).***
- ***RMM 24 (Loving, Caring).***
- ***RMM 25 (Thankful).***

A long research tradition has examined various self-reported transcendent positive emotions (Cohn & Fredrickson, 2012; Gillham, Shatté, Reivich, & Seligman, 2001; Keltner & Haidt, 2003; Scheier & Carver, 1987; Yaden, 2017):

- Admiration
- Awe
- Compassion
- Elevation
- Gratitude
- Happiness
- Love
- Optimism

Fredrickson has proposed a "broaden-and-build" hypothesis. Transcendent positive emotions broaden one's "thought–action repertoire"; they build one's personal resources, whether physical, intellectual, or social. These "prompt individuals to discard time-tested or automatic (everyday) behavioral scripts and to pursue novel, creative, and often unscripted paths of thought and action" (Fredrickson, 1998, p. 311).

Negative emotions narrow attentional focus (Easterbrook, 1959). Transcendent positive emotions have an opposite effect—expansion of attentional focus—that enables one to see both the forest and the trees, the figure and ground, and that increases creativity and flexibility. Transcendent positive emotions counter the aftereffects of negative emotion, enabling one to loosen the hold that (no-longer-relevant) negative emotions have on an individual's mind, a notion similar to letting go in RMM. It is not surprising that Fredrickson proposes that transcendent positive emotion may well be one mechanism underlying meditation and, by extension, all of RMM (Fredrickson, Cohn, Coffey, Pek, & Finkel, 2008). Specifically, they help channel or focus attention, help one put aside distraction and mind wandering, and provide motivation to continue practicing. I propose that transcendent positive emotions are not incidental descriptors, triggers, or effects, but a part of what RMM is all about.

Are All RMM States Really Part of RMM?

Those who embrace strictly neurophysiological definitions of RMM may protest that our list of 25 RMM states is too broad. My response is that this lexicon reflects what practitioners of progressive muscle relaxation, yoga and tai chi, breathing exercises, autogenic training, and visualization/imagery, meditation, and mindfulness actually experience. Of the 150 RMM classes I have taught over the past 50 years, involving over 4,000 students, in every single class at least one student reports even the rarest RMM state. This is reflected in factor analyses of practitioner experiences, research reviewed later in the chapter. Of course, some (Level 1, Basic Relaxation; Level 2, Basic Quiet Focus; Level 5, Transcendent Positive Emotion) are more common than others (Deepening, Transcendence). But each reaction is experienced by at least some practitioners. The fact that many trainers may find this surprising suggests that they should perhaps listen more to their students. They have important stories to tell. Progressive muscle relaxation can be more than identifying and releasing tension. Hatha yoga can be more than stretching and assuming certain postures. Breathing exercises can go beyond breath. Autogenic training can be more passively repeating phrases suggesting relaxing physical states. Mindfulness can go beyond nonjudgmental present-centered awareness.

RESEARCH

When RMM techniques are narrowly viewed in terms of physiological variables, relatively few differences emerge. However, in dramatic contrast, every single study that has compared a broad spectrum of RMM states has found significant, consistent, and dramatic differences among techniques. In this section I present a sample of studies. Over a dozen versions of our RMM questionnaires have evolved over the decades. Currently, researchers use the Smith Relaxation States Inventory 3 (SRSI3) and its successor, the RMM Tracker Series (*http://blogs.roosevelt.edu/jsmith*). We use both in research and training. In the following summary, I present findings in terms of current RMM terminology.

The Factor Structure of RMM States

Scholars may be surprised that no fewer than 31 separate published factor analyses support our differentiation of 25 RMM states (Smith, 2019). Factor-analytic research on precursors to current RMM inventories began in 1996. Smith et al. (1996) presented early versions of RMM word lists to 940 practitioners of massage, progressive muscle relaxation, yoga, breath exercises, imagery, and meditation. Practitioners described technique effects. Ten factors emerged: Joyful, Distant (currently Far Away, Disengaged), Calm (currently At Ease, At Peace), Aware, Prayerful, Accepted (Unbothered), Untroubled (Unbothered), Limp (Physically Relaxed), Silent (Quiet), and Mystery (Awe/Wonder, Deep Mystery). This is the first study to reveal the rich and complex psychological states the full range of RMM techniques can evoke.

Smith et al. (2000) summarized 13 factor-analytic studies completed prior to 2000. The combined sample included 1,904 participants reporting on a diverse range of RMM techniques. Using a rigorous selection criterion, only RMM items that loaded consistently and exclusively (.70) on a factor were included. Six factors consistently emerged: Centered Positive Affect (Transcendent Positive Emotion), Sleepiness (not a part of current tests), Disengagement (Far Away, Disengaged), Physical Relaxation, Mental Quiet (Quiet), and Spiritual.

Subsequent RMM research has found a similar set of factors. Corbeil, Marcaurell, and Belanger (2015), examining 531 college students, replicated the RMM trait factors of Physical Relaxation (relaxation combined with mindfulness), Positive Emotion, and Spirituality. Corbeil and colleagues found that relaxation and basic mindfulness items load on the same factor (Physical Relaxation).

Borgogna and Smith (2016) gave RMM state questionnaires to 119 practitioners (average of 3 years' practice) of yoga/meditation (after 40-minute practice sessions) and 115 nonpractitioners. Consistent with Corbeil and colleagues (2015), we found three factors: Mindful Relaxation (mindfulness and relaxation items), Transcendence, and Positive

Emotion. In contrast, Malia (2018) gave a state version of the RMM to 210 seasoned practitioners (average of 4 years' practice) of various blends of hatha yoga and mindfulness after practice sessions averaging 46 minutes in length and found the factors Mindful Transcendence, Mindful Focus, Mindful Positive Emotion, Basic Mindful Relaxation, Pleasant Fantasy, and Unbothered Observer. Mindful Relaxation was defined by Physical Relaxation, Centered/Grounded, and Quiet. A separate mindfulness factor emerged, Mindful Focus, defined by Focus, Absorption, and Easy, Effortless.

In sum, factor-analytic studies of RMM states find distinct RMM factors reflecting four of our five levels: Basic Relaxation, Quiet Focus, Transcendence, as well as Transcendent Positive Emotion. (Level 4, Deepening, items were added recently and have not been subjected to factor analysis.) However, the general pattern for some factors to appear in some studies and not others and to at times merge into a single factor should not be seen as a sign of unreliability.

With a bit of humility, I propose a change in how we study RMM states. As noted earlier, the factor structure of RMM states may depend on the RMM population under study. Buddhist monks and Roman Catholics at a prayer retreat may well display distinct patterns of RMM states. The pursuit of a universal factor structure is a fool's errand. First, I suggest researchers examine what factor patterns are characteristic of specific populations. This is particularly relevant to emerging discussions as to whether relaxation and meditation/mindfulness evoke the same states or distinct sets of states (Borgogna & Smith, 2016; Corbeil et al., 2015; Malia, 2018). Second, when do Basic Relaxation states and Basic Quiet Focus manifest as one factor? Perhaps for nonrelaxers and beginning practitioners. Perhaps these states differentiate into two or more factors as training continues. Third, I recommend including a supplementary analysis at the item level in which each of 25 items is treated as a separate variable. Currently, my team is examining RMM 1, 5, 12, 16, and 18.

RMM 1 (Far Away, Disengaged)

Perhaps one of the most important fruits of RMM state research has been the discovery of RMM 1 (Far Away, Disengaged). In factor analysis, this state is defined by three dimensions: spatial, attitudinal, and somatic. Each reflects withdrawal from and reduced awareness of the world. Terms such as feeling *distant, far away, disengaged,* and *in my own world* are primarily spatial. In contrast, statements such as feeling *detached, indifferent, not caring about anything, unmoved,* or *unbothered* represent an attitude of withdrawal. As relaxation progresses, one may display a type of disengagement in which one becomes less aware of one's limbs and parts of one's body. A client may realize that he or she has lost awareness of hands, arms, legs, or feet. More dramatically, one may have an out-of-body experience in which one feels or actually hallucinates oneself floating above and observing one's own physical body. Clinically, one might view RMM 1 at least in part as low-level, potentially adaptive dissociation.

RMM 1 (Far Away, Disengaged) correlates negatively with the Sixteen Personality Factor Questionnaire (16-PF) factor of Emotional Stability and positively with Vigilance, Abstractedness, Apprehension, and Anxiety (Leslie & Clavin, 2001). On the Millon Index of Personality Styles, it correlates positively with Persevering, Accommodating, Introversing, Hesitating, Dissenting, and Complaining (Sohnle, 2001). On the Symptom Checklist–90—Revised, it correlates positively and highly with all scales of psychopathology except Paranoid Ideation (Anderson, 2001).

Such research suggests that those under distress are most likely to conceptualize effective relaxation as simply *getting away from it all, tuning out,* or becoming *indifferent,* all of which are defining descriptors of RMM 1 (Far Away, Disengaged). This RMM 1 appears to be one of the first to emerge in relaxation training, especially progressive muscle relaxation, and it may be a prerequisite to becoming successfully Physically Relaxed. To relax the body, one must first learn to disengage from the stressors of the world. Progressive muscle relaxation (and, I hypothesize, rudimentary autogenic standard exercises) may well be among the most effective tools for becoming Disengaged. Yoga stretching and breathing exercises appear less likely to evoke this RMM state (Ghonchec & Smith, 2004; Matsumoto & Smith, 2001).

Technique Comparisons

Progressive muscle relaxation is initially associated with RMM 1 and 2 (Far Away, Disengaged; Physically Relaxed), whereas both yoga stretching and breathing exercises are more associated with RMM 13 (Clear, Awake, Aware; Boukydis, 2004; Ghonchec & Smith, 2004; Matsumoto & Smith, 2001; Rice, Cucci, & Williams, 2001; Ritchie, Holmes, & Allen, 2001; Smith & Jackson, 2001).

Ghonchec and Smith (2004), Matsumoto and Smith (2001), and Boukydis (2004) have used a promising new design for evaluating techniques. These researchers assigned participants to one of two approaches. Ghonchec and Smith (2004) compared progressive muscle relaxation and yoga stretching, whereas Boukydis (2004) and Matsumoto and Smith (2001) looked at progressive muscle relaxation and breathing exercises. Participants practiced their assigned technique once a week for 5 weeks in a supervised group setting, using standardized matched 28-minute recordings. Samples were diverse. Matsumoto and Smith (2001) examined college undergraduates; Ghonchec and Smith (2004) used bank employees; and Boukydis (2004) used a clinical sample of outpatients who had been in therapy for an average of 6 years for anxiety and depression.

RMM states were assessed before and after each session. At weeks 1 and 5, RMM state *aftereffects* were assessed. An aftereffect (Smith, 1999) is a state that emerges after a 3-minute pause at the end of training and after an initial posttest. During the intervening 3 minutes, participants are instructed to casually think about the forthcoming day's and week's activities. All studies found that progressive muscle relaxation consistently evokes RMM 2 (Physically Relaxed) and (with the exception of Boukydis, 2004) RMM 1 (Far Away, Disengaged). Interestingly, for all studies, some effects took 4 or 5 weeks to emerge and did not show up on an immediate posttest but as an aftereffect 3 minutes after posttesting. For example, both Ghonchec and Smith (2004) and Matsumoto and Smith (2001) found that RMM 9 (Quiet), and RMM 13 (Happy, Optimistic, Trusting) emerge as aftereffects.

Little research has examined the impact of combining relaxation techniques. Two cross-cultural studies on senior citizens provide some intriguing leads. Bang (1999) examined relaxation scripting with 22 non-English-speaking Korean American nursing home residents. Half were assigned to a no-treatment control group and half received combination training. Specifically, participants were taught a different technique each day for 6 days (progressive muscle relaxation, autogenic training, breathing, yoga, imagery, and meditation). On the 7th day a group combination script was constructed incorporating exercise components that participants voted to include. The group script was then practiced for the remaining 14 days. Gonzales (2001) repeated this design on 24 senior citizens in Rio Piedras, Puerto Rico. Both studies found significantly reduced Beck

Depression Inventory scores and increased scores on RMM 1 and 2 (Far Away, Disengaged; Physically Relaxed). In addition, Bang (1999) found increased scores on RMM 23 (Happy, Optimistic, Trusting), RMM 24 (Loving, Caring), and RMM 25 (Thankful). Gonzales (2001) found higher levels of RMM 9 (Quiet), RMM 4 (Refreshed), and RMM 3 (At Ease, At Peace).

Additional technique studies are worth noting. Gillani and Smith (2001) found that advanced Zen meditators report higher levels of RMM 9 (Quiet); RMM 25 (Thankful); and RMM 20 (Reverent, Prayerful). Smith and Joyce (2004) compared the relaxing impact of Mozart's music, new age music, and reading popular magazines. Individuals who selected and listened to Mozart's "Eine Kleine Nachtmusik" reported higher levels of RMM 9 (Quiet), RMM 13 (Clear, Awake, Aware), and RMM 21 (Awe/Wonder, Deep Mystery). Both Mozart listeners and listeners to new age music (Halpern's "Serenity Suite") reported higher levels of RMM 3 (At Ease, At Peace), RMM 4 (Refreshed), and RMM 24 and 25 (Loving, Caring; Thankful).

IMPLICATIONS FOR TRAINING AND ASSESSMENT

Over the past half century, I have devised a number of relaxation training and assessment protocols (Smith, 1985, 1990, 1999, 2005, 2017). As noted earlier, my approach has evolved, informed by the experiences of over 4,000 trainees and guided by two general ideas: (1) Different approaches to RMM have diverse, different, and changing effects. This suggests the value of combining pure and combination training formats; (2) Self-report is an important part of training and assessment. Training and assessment should be targeted to client strengths, needs, and interests. In addition, conscientious trainers should provide clients with a map for understanding how RMM may change over time, presenting new challenges and opportunities.

Pure and Combination Training Formats

I begin by teaching relatively pure versions of techniques. This makes it easier for students to detect and articulate RMM states that may be unique to specific techniques. Such an approach is meaningful only to the extent to which the procedural components of various approaches can be differentiated. For example, it makes little sense to contrast the effects of hatha yoga, progressive muscle relaxation, mindfulness, and breathing exercises when all of these approaches incorporate breathing exercises and imagery.

Initial training covers a full spectrum of RMM techniques. Each student receives 2 weeks of progressive muscle relaxation, seated yoga, breathing exercises, autogenic training, imagery, and meditation/mindfulness. Four types of meditation/mindfulness are presented: body scanning, breath scanning, FA meditation, and OM meditation. Of these, students prefer progressive muscle relaxation, yoga, breathing, and FA meditation. Students often change their preferences as skills develop. Only after teaching several families of relaxation do I combine families of relaxation into an individualized program. For example, it is at this stage that imagery and breathing might be incorporated with progressive muscle relaxation.

My combination approach is not particularly original. Careful examination of what master trainers of RMM actually do (as opposed to what they say they do) reveals a preference for combining approaches. Many forms of progressive muscle relaxation blend letting go with an occasional quick stretch, often paced with inhaling and releasing breath, incorporated with some imagery ("imagine a tight wad of string slowly unwinding") and physically targeted suggestions ("Let the tension melt away . . ."). Traditional hatha yoga is a rich mixture of stretching, breathing, physically targeted suggestion, and, often, letting go. Autogenic standard exercises deploy physically targeted suggestion and passive breathing exercises. More advanced autogenic exercises introduce imagery. And all approaches incorporate a bit of targeted and sustained meditative focus (Smith, 2005).

Although expert trainers may combine approaches, often they do so without a clear rationale. A good example of this is the preference among many well-known meditation and mindfulness instructors for preparatory stretching or breathing exercises. Why not use progressive muscle relaxation as a preparation? Indeed, our research (Ghonchec & Smith, 2004; Matsumoto & Smith, 2001) has found that progressive muscle relaxation (and not stretching or breathing) can evoke RMM 9 (Quiet), which is strongly associated with the practice of meditation (Gillani & Smith, 2001). Indeed, as mentioned earlier, Jacobson (1924) himself described his approach as a neuromuscular method for reducing worry or, as meditators prefer, "distracting thought." Devotees of various schools of meditation or mindfulness may bristle at any deviation from millennia of *time-tested* tradition. However, we need to be mindful that such tradition may reflect the popularity of charismatic masters (and their favorite techniques), the endurance of pseudoscientific beliefs (and associated exercises), religious dogma, and unsystematic and careless trial and error. There are no substitutes for rigorous research.

Assessment

Traditionally, the assessment of RMM focuses on symptom reduction. I supplement this by teaching students to identify, articulate, and differentiate RMM states More generally, I prompt students to discover how RMM can go beyond a targeted outcome state or trait and become a journey that is multidimensional, interactive, dynamic, and directional. To these ends, I use two strategies. First, I have students perform a "Double Present Moment Check" of what they feel before and after an exercise. The specific instructions consist of four steps:

1. **Check.** "Just before your exercise, ask yourself: 'What am I feeling right now, the present moment? What one or two words or images best describe my experience?' Do not try to analyze this question or deliberately figure out the right or correct answer. Simply let words or images come to mind on their own and pick those that feel right for the moment. This is your first Present Moment Check.
2. **Record and let go.** Put your words or images aside. You may briefly note them on paper. You are finished with this present moment check and are ready to move on to your exercise. There is no need to review or reconsider what you have done. That is history and not a part of what you are about to do. By putting your first present check aside, you are formally beginning your exercise.
3. **Practice.** Do your selected exercise. It is important that you practice sincerely and correctly. What you are about to do is important, not casual.
4. **Check.** Do a second Present Moment Check. As before, simply ask 'What am I feeling right now, the present moment?' "

Second, students complete the 25-item RMM Tracker after their best session for each approach (inventory available at *http://blogs.roosevelt.edu/jsmith*). Completing

RMM Trackers on a regular basis helps trainees articulate effects, compare techniques, identify changes that may be occurring, and uncover potential directions of practice. For example, one trainee may learn this sequence, practicing each approach for 3 weeks: progressive muscle relaxation → yoga stretching and breathing exercises → imagery → loving-kindness meditation → mindfulness meditation. One form of meditation is practiced throughout as a "home exercise." The pattern of RMM states experienced may evolve over time. They may at first experience RMM 1 (Far Away, Disengaged) followed by RMM 2 (Physically Relaxed). As distractions subside, they may begin to experience RMM 7, 8, and 9 (Focus, Absorption; Centered, Grounded; Quiet). Meditative skills deepen, and they may experience RMM 9, 10, 11, and 13 (Quiet; Unbothered; Easy, Effortless; Clear, Awake, Aware). In time, they may experience an awareness of the deeper potential of practice and a need to review practice goals (RMM 12, Observer; RMM 14, Interested, Curious, Fascinated). Such experiences may renew motivation to practice regularly, contributing to RMM 16 (Going Deeper), RMM 18 (Sense of Something Greater), and RMM 19 (Meaning, Purpose, Direction). In time, an occasional transcendent experience may further illuminate deeper reasons for practice (RMM 20, Reverent, Prayerful; RMM 21, Awe/Wonder, Deep Mystery; RMM 22, Spiritual, Mystical).

In my experience as a trainer, I find that students of all techniques become increasingly sensitized and able to differentiate subtle differences between RMM states as training continues. Advanced practitioners report more RMM states than beginners. This should not be surprising and is a pattern for many types of training. Beginning chef students may be able to differentiate Asian, Italian, and Mexican cuisine. Advanced chefs, with their refined palates and culinary vocabularies, may be able to identify a variety of foods in each region, say Ahnui, Cantonese, Fujian, Hunan, Jiangsu, Shandong, Sichuan, and Zhejiang cuisines. I believe an important part of RMM training is helping students identify, articulate, and differentiate RMM states.

In sum, the outcome of RMM training is not a single state or trait but a broad spectrum of RMM states, one that is multidimensional, interactive, dynamic, and directional. Initial targeted outcomes (feeling Physically Relaxed) may pave the way and prepare for newer outcomes (Centered, Quiet, Easy), which in turn lead to additional unanticipated outcomes (Curious, Purpose, Sense of Something Greater). The outcome of RMM training is not a narrow-spectrum state or trait but a broad-spectrum and evolving story.

CONCLUSION

RMM theory presents a challenge to trainers, researchers, and students of relaxation, meditation, and mindfulness. Most programs promote a favored goal or, in our terms, preferred RMM states. Training in progressive muscle relaxation may emphasize symptom relief and feeling Physically Relaxed. A yoga retreat might teach Physically Relaxed; Centered, Grounded; and Easy, Effortless. A mindfulness system might emphasize Focus, Absorption; Quiet; and Unbothered. A loving-kindness or metta meditation group might focus on Loving, Caring. A group practicing centering prayer may focus on Reverent/Prayerful; At Ease, Peaceful; and Happy, Optimistic, Trusting. In the past, such defining RMM states have been influenced by the shifting sands of history, tradition, charisma, dogma, and bias. RMM theory presents a map of an empirically derived set of states not based on any particular tradition. Such a universal lexicon invites us to look beyond our islands of favored techniques and consider the larger world of relaxation, meditation, and mindfulness.

REFERENCES

Anderson, K. P. (2001). The Symptom Checklist–90—Revised and relaxation states during one's preferred relaxation activity. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 138–140). New York: Springer.

Bang, S. C. (1999). *ABC relaxation training as a treatment for depression for the Korean elderly*. Unpublished master's thesis, Roosevelt University, Chicago, IL.

Benson, H. (1975). *The relaxation response*. New York: Morrow.

Borgogna, N., & Smith, J. C. (2016, June 16–19). *The factor structure of relaxation and mindfulness: Two states or one?* Poster presented at the Association for Contextual Behavioral Science Annual World Conference 14, Seattle, WA.

Boukydis, N. N. (2004). *Progressive muscle relaxation, breathing exercises, and ABC relaxation training in an outpatient clinical population*. Unpublished master's thesis, Roosevelt University, Chicago, IL.

Braid, J. (1853). *Neurypnology or the rationale of nervous sleep considered in relation with animal magnetism*. London: Churchill.

Cohn, M. A., & Fredrickson, B. L. (2012). Positive emotions. In S. J. Lopez & C. R. Snyder (Eds.), *The Oxford handbook of positive psychology* (2nd ed., pp. 13–24). New York: Oxford University Press.

Corbeil, M. E., Marcaurell, R., & Belanger, B. (2015). Adaptation française et validation du Smith Relaxation States Inventory 3. *Revue Francophone de Clinique Comportementale et Cognitive, 20*(1), 15–34.

Easterbrook, J. A. (1959). The effect of emotion on cue utilization and the organization of behavior. *Psychological Review, 66,* 183–201.

Fredrickson, B. L. (1998). What good are positive emotions? *Review of General Psychology, 2*(3), 300–319.

Fredrickson, B. L., Cohn, M. A., Coffey, K. A., Pek, J., & Finkel, S. M. (2008). Open hearts build lives: Positive emotions, induced through loving-kindness meditation, build consequential personal resources. *Journal of Personality and Social Psychology, 95*(5), 1045–1062.

Ghonchec, S., & Smith, J. C. (2004). Progressive muscle relaxation, yoga stretching, and ABC relaxation theory. *Journal of Clinical Psychology, 60,* 131–136.

Gillani, N. B., & Smith, J. C. (2001). Zen meditation and ABC relaxation theory: An exploration of relaxation states, beliefs, dispositions, and motivations. *Journal of Clinical Psychology, 57,* 839–846.

Gillham, J. E., Shatté, A. J., Reivich, K. J., & Seligman, M. E. P. (2001). Optimism, pessimism, and explanatory style. In E. Chang (Ed.), *Optimism and pessimism: Implications for theory, research, and practice* (pp. 53–75). Washington, DC: American Psychological Association.

Goleman, D., & Davidson, R. J. (2017). *Altered traits: Science reveals how meditation changes your mind, brain, and body*. New York: Penguin Random House.

Gonzales, R. (2001). ABC relaxation training as a treatment for depression for Puerto Rican elderly. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 209–211). New York: Springer

Hanley, A. W., Bernstein, A., Nakamura, Y., Hadash, Y., Rojas, J., Tennant, K. E., et al. (2020). The Metacognitive Processes of Decentering Scale: Development and initial validation of trait and state versions. *Psychological Assessment*. [Epub ahead of print]

Jacobson, E. (1929). *Progressive relaxation*. Chicago: University of Chicago Press.

Keltner, D., & Haidt, J. (2003). Approaching awe, a moral, spiritual, and aesthetic emotion. *Cognition and Emotion, 17,* 297–314.

Leslie, K. A., & Clavin, S. L. (2001). The Sixteen Personality Factor Questionnaire and recalled relaxation states in one's preferred relaxation activity. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 122–125). New York: Springer.

Lutz, A., Jha, A. P., Dunne, J. D., & Saron, C. D. (2015). Investigating the phenomenological matrix of mindfulness-related practices from a neurocognitive perspective. *American Psychologist, 70*(7), 632–658.

Maclean, K. A., Leoutsakos, J.-M. S., Johnson, M. W., & Griffiths, R. R. (2012). Factor analysis of the Mystical Experience Questionnaire: A study of experiences occasioned by the hallucinogen psilocybin. *Journal for the Scientific Study of Religion, 51,* 721–737.

Malia, T. (2018). *Self-reports of relaxation and mindfulness states among experienced practitioners of mindfulness-related disciplines* (Doctoral dissertation). Ann Arbor, MI: ProQuest Dissertations. (ProQuest No. 107521124)

Maslow, A. H. (1964). *Religions, values, and* *peak-experiences*. Columbus: Ohio State University Press.

Matsumoto, M., & Smith, J. C. (2001). Progressive muscle relaxation exercises, breathing exercises, and ABC relaxation theory. *Journal of Clinical Psychology, 57,* 1551–1557.

Pahnke, W. N. (1963). *Drugs and mysticism: An analysis of the relationship between psychedelic drugs and the mystical consciousness*. Cambridge, MA: Harvard University Press.

Pahnke, W. N. (1969). Psychedelic drugs and mystical experience. *International Psychiatry Clinics, 5*(4), 149–162.

Rice, S., Cucci, L., III, & Williams, J. (2001). Practice variables as predictors of stress and relaxation dispositions for yoga and meditation. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 193–196). New York: Springer.

Ritchie, T. D., Holmes, R. C., III, & Allen, D. (2001). Preferred relaxation activities and recalled relaxation states. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 187–189). New York: Springer.

Safran, J. D., & Segal, Z. V. (1990). *Interpersonal process in cognitive therapy*. New York: Rowman & Littlefield.

Scheier, M. F., & Carver, C. S. (1987). Dispositional optimism and physical well-being: The influence of generalized outcome expectancies on health. *Journal of Personality, 55,* 169–210.

Schultz, J. H. (1932). *Das autogene training—Konzentrative Selbstentspannung*. Leipzig, Germany: Thieme.

Smith, J. C. (1985). *Relaxation dynamics: Nine world approaches to self-relaxation*. New York: Springer.

Smith, J. C. (1986). Meditation, biofeedback, and the relaxation controversy: A cognitive-behavioral perspective. *American Psychologist, 41,* 1007–1009.

Smith, J. C. (1990). *Cognitive-behavioral relaxation training: A new system of strategies for treatment and assessment*. New York: Springer.

Smith, J. C. (1999). *ABC relaxation theory*. New York: Springer.

Smith, J. C. (2001). *Advances in ABC relaxation: Applications and inventories*. New York: Springer.

Smith, J. C. (2005). *Relaxation, meditation, and mindfulness: A mental health professional's guide to new and traditional approaches*. New York: Springer.

Smith, J. C. (2017). *Stress and coping: The eye of mindfulness*. Dubuque, IA: Kendall Hunt

Smith, J. C. (2019). *Third-generation mindfulness and the universe of relaxation*. Dubuque, IA: Kendall Hunt.

Smith, J. C., Amutio, A., Anderson, J. P., & Aria, L. A. (1996). Relaxation: Mapping an uncharted world. *Biofeedback and Self-Regulation, 21,* 63–90.

Smith, J. C., & Jackson, L. (2001). Breathing exercises and relaxation states. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 202–204). New York: Springer.

Smith, J. C., & Joyce, C. A. (2004). Mozart vs. new age music: Relaxation states, stress, and ABC relaxation theory. *Journal of Music Therapy, 41,* 215–224.

Smith, J. C., Wedell, A. B., Kolotylo, C. J., Lewis, J. E., Byers, K. Y., & Segin, C. M. (2000). ABC relaxation theory and the factor structure of relaxation states, recalled relaxation activities, dispositions, and motivations. *Psychological Reports, 86,* 1201–1208.

Sohnle, S. (2001). The Millon Index of Personality Styles and recalled relaxation states for one's preferred relaxation activity. In J. C. Smith (Ed.), *Advances in ABC relaxation: Applications and inventories* (pp. 132–137). New York: Springer.

Stace, W. T. (1960). *Mysticism and philosophy*. New York: MacMillan Press.

Yaden, D. B., Haidt, J., Hood, R. W., Jr., Vago, D. R., & Newberg, A. B. (2017). The varieties of self-transcendent experience. *Review of General Psychology, 21,* 143–160.

CHAPTER 4

Stress, Inflammatory Cytokines, and the Brain

An Overview

Alexander W. Kusnecov
Sara Norton
Marialaina Nissenbaum

Studies at the interface of stress and inflammation fall into the domain of *psychoneuroimmunology,* a multidisciplinary field devoted to understanding interactions between the brain and behavior as they relate to the immune and endocrine systems (Ader, Cohen, & Felten, 1995). This field considers a multiplicity of interacting biological variables, including, in more recent years, the gut microbiome (Fulling, Dinan, & Cryan, 2019; Rieder, Wisniewski, Alderman, & Campbell, 2017). What characterizes psychoneuroimmunology is a multidisciplinary approach to understanding health and disease. The focus can shift according to an investigator's primary discipline, settling on pathologies within the immune system or the brain, and contingent on the particular scientific problem in question (e.g., searching for immune mediators of Alzheimer's disease or explaining autoimmune disease in terms of functional changes in the endocrine system). One of the seminal observations in this field is that the pattern of neurobiological responses often observed in response to psychological stressors can be reenlisted by immune factors. That is, the immune response is "sensed" by the brain, which coordinates behavioral and neural adjustments accordingly. This fundamental observation spawned a large number of basic studies, which expanded to include investigations of how stress-related peripheral immune mechanisms affect psychiatric disease, a literature now comprising studies that number in the thousands.

In this chapter, we provide a selective overview of these developments, so that the reader may appreciate the impact that immunological processes may have on neurobiological function. This entails a basic description of some of the neurotransmitter and neuroendocrine changes that define the stress response from a neurobiological standpoint. We then turn the discussion to the nature of inflammatory immune-related processes, both peripheral and central, that elicit neurobiological changes indicative of a stress-like state. In so doing, we place the major focus on a class of immune-derived proteins called cytokines. These are the molecular intercellular messengers that have been shown to affect the brain and alter behavior. This may have relevance to mental health and the development of psychiatric conditions.

INFLAMMATION

The term *inflammation* can be difficult to define, as it encompasses multiple cellular and molecular components, as well as different causal conditions. Because we are concerned in this chapter with the impact of the immune system on the brain, and because the literature has opted to describe such events as inflammatory-based or neuroinflammatory, our use of the term *inflammation* will assume the involvement of cytokines and/or immune cells. In fact, the immune system is central to inflammation. Although, clinically, visual inspection of inflamed tissue would look for telltale signs of redness, swelling, and tenderness (Punchard, Whelan, & Adcock, 2004), not all inflammatory conditions are visually apparent, as inflammation occurs in all organs. Importantly, inflammation involves the immune system, and increased inflammatory activity is a reaction to infection and/or tissue damage that is attended by local vascular and endothelial changes (Kumar, 2019a; Medzhitov, 2008; Punchard et al., 2004). Inflammation without an immune response is rare. Tissue cells that are undergoing distress or are dying typically invite infiltration of immune cells, which further amplifies the presence of more immune cells and their cytokine products. In cases in which the immune response is self-directed, as in autoimmune disease, tissue damage is a defining clinical condition (e.g., arthritic joints) that is attended by an accumulation of leukocytes and their soluble products (cytokines and antibodies). This is the canonical view of inflammation—incorporating tissue disruption, immune components, and infection.

Inflammation as a term or even an explanatory concept was expanded in more recent years to cover aseptic or noninfectious conditions, resulting in the term *sterile inflammation*. Studies on stress, in particular, have utilized this term (Fleshner, Frank, & Maier, 2017), which is applied when the presence of increased circulating cytokines—or inflammatory markers—occurs in the absence of attributional causal factors such as pathogens and/or obvious injury. In fact, studies of depression and stress often measure circulating inflammatory mediators (mainly cytokines), often without associated evidence of frank infection (Fleshner et al., 2017; Marsland, Walsh, Lockwood, & John-Henderson, 2017). Indeed, the association between depression and increased parameters of immunity was reported by Maes and colleagues, who showed that increased numbers of leukocytes were found in the blood of patients with depression (Maes et al., 1992). This was in keeping with an immune theory of depression (Maes, Smith, & Scharpe, 1995) that subsequently evolved to focus on cytokines and their actions on the central nervous system (CNS; Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Felger & Lotrich, 2013; Himmerich, Patsalos, Lichtblau, Ibrahim, & Dalton, 2019). Indeed, cytokine immunotherapy using interferon-alpha was found to induce depression and anxiety symptoms in patients with cancer and hepatitis-C (Capuron et al., 2002; Greenberg et al., 2000; McNutt et al., 2012). Although cytokine immunotherapy has the drawback of involving high-dose regimens, as well as affecting individuals burdened with disease, such effects on mood suggest that, in humans, circulating increases in cytokines might have neurobehavioral effects. The reader is referred to a recent review of cytokine involvement in depression, along with problems of interpretation, by Himmerich and colleagues (Himmerich et al., 2019).

Neuroinflammation

The CNS consists of the brain and spinal cord, and the presence of inflammation within it is called *neuroinflammation*. This may refer to immune-like events (increased glial cell activation and cytokine concentration) that emerge solely from within the brain. However, to the extent that infiltration of immune cells (leukocytes) from the circulation and into nervous tissue proper can release soluble mediators that may affect neuronal functions, this also can serve as the basis of neuroinflammation. In fact, it has been argued by some that the term should only be used if all these conditions are present (cellular infiltration and intrinsic immune-like activation; Estes & McAllister, 2014). Nonetheless, whatever the makeup of neuroinflammation, the suspected mechanism of neuromodulation observed in "neuroinflammatory" conditions typically points to cytokines, as well as glial cells, which are discussed in the final section of this chapter.

One question to consider is whether there is adaptive significance to neuroinflammation or, at the very least, cytokine elevations in the brain. The answer here is not simple. Animal studies have suggested that deletion of intrinsic cytokines in the brain (i.e., those produced by either neurons or glial cells) results in deficient cognitive functions, whereas excessive cytokine concentrations and cellular infiltration into the brain can impair recovery of neuronal function following injury. It seems, therefore, that cytokines in the brain may contribute to optimal neurobehavioral functions but within circumscribed limits. This has led to the *double-edged sword* hypothesis of neuroinflammation, which argues that a balance of proinflammatory and anti-inflammatory mechanisms needs to be maintained to ensure successful tissue repair and/or normal neuronal function (Wyss-Coray & Mucke, 2002). A similar argument can be leveled at interpretation of peripheral cytokine measures. To what degree are such elevations—especially after stressor exposure—indicative of pathology? Studies of healthy human participants in whom stressors induced cytokine increases have not provided evidence for tissue injury or infection (Marsland et al., 2017). In the absence of such evidence, the physiological meaning of stressor-induced elevations in inflammatory cytokines remains unknown. One interpretation is that cytokine elevations may represent a normal reflex response to stress (like neuroendocrine and autonomic responses). This is likely to influence adaptational immunological processes—for example, preparing immune cells to encounter an infectious agent—but it may also have the potential to promote pathology. In fact, inflammatory cytokines are implicated in a variety of pathophysiological conditions, including heart disease, musculoskeletal problems, autoimmune disease, allergies, susceptibility to viral and bacterial infections, and mental health problems (Barnes, 2018; Himmerich et al., 2019; Holdsworth & Gan, 2015; Kaptoge et al., 2014; Nikoopour, Schwartz, & Singh, 2008; Wojdasiewicz, Poniatowski, & Szukiewicz, 2014).

Cytokines and the CNS: Relevance to Mental Health

The importance of cytokines in mental health is underscored by their ability to modulate neuronal activity and activate neural-controlled physiological systems, especially the hypothalamic–pituitary–adrenal (HPA) axis and autonomic nervous system (ANS). By virtue of their CNS actions, cytokines also exert changes across the cognitive, emotional, and motivational domains of behavior, changes that collectively have been referred to as "sickness behavior" (Dantzer et al., 2008). The induction of behavioral change by the immune system is likely an evolutionary advantage that controls exposure to infectious agents and creates recuperative strategies. Interestingly, it was pointed out that sickness behavior maps onto clinical criteria for depressive symptoms, which reinforced the *inflammation or immune hypothesis* of depression (Maes et al., 1995). This raised the question of whether symptoms we normally associate with depression are in the short term adaptive and of evolutionary value. That is, behavioral patterns of reduced motivation, movement, and food intake may very well be universal responses to a variety of stressors, whether immunological or psychological. As such, they may be adaptive by facilitating a period of rest and recuperation. However, failure to recover from this behavioral state, with an unusual prolongation of lethargy and reduced motivation, may be suggestive of clinical depression. Identifying the biological conditions that precipitate and maintain this behavioral state has preoccupied depression researchers for several decades, with inflammatory cytokines maintaining considerable attention as a possible influence.

Not surprisingly, immune or inflammatory processes are potential etiological or precipitating factors in other mental health disorders. For example, both schizophrenia and autism spectrum disorders have been associated with inflammation and immune dysregulation (Estes & McAllister, 2015; Feigenson, Kusnecov, & Silverstein, 2014; Khandaker et al., 2015). These conditions are known to involve frank abnormalities in the development of brain architecture, which could in turn alter immune function; but at the same time, a growing literature posits that perhaps immune-related events shape the development of the brain and, if not successfully assembled, might increase vulnerability to disorders of mental health (Anisman, Hayley, & Kusnecov, 2018). Moreover, neuropsychiatric symptoms can arise from various inflammatory disease conditions (e.g., autoimmune disorders; Capuron & Castanon, 2017; Leon et al., 2018; Monastero et al., 2001). This reinforces the notion that immune processes can modify CNS function and cause behavioral abnormalities.

In summary, immunological processes are at the heart of inflammatory events, defining their cellular and molecular features. Critical to this is the elaboration of molecules called cytokines. These have figured prominently in our understanding of how the immune system—and inflammation generally—affects CNS function. To the extent that the brain is the substrate of behavior, it figures that the actions of cytokines on the CNS result in behavioral changes, which may progress to clinically relevant states, such as depression. To the extent that inflammation can also occur within the brain, it is also the case that cells within the brain, such as glia, produce cytokines and, consequently, may contribute to inflammatory changes that promote neurodegenerative pathology. This is at the heart of research into neuroinflammation. Later we describe the immune system and the various types of cytokines that immune cells can generate. In that context, we provide examples of how cytokines have figured in the activation of stress-related systems in the brain.

NEUROBIOLOGY OF THE STRESS RESPONSE: RELEVANCE OF CYTOKINES

To align cytokine-induced effects on the CNS with those observed in response to psychological stressors, this section provides a general overview of neurobiological systems—including neurotransmitters—that are responsive to stress. In particular, we focus on the HPA axis and mention only briefly relevant brain structures involved in its activation (see a more expanded treatment of neurobiological stress systems in Sime & Phillips, Chapter 21, this volume). Before proceeding, however, we consider the notion of stress as an explanatory concept, as evidence for immune-mediated effects on the CNS suggests an adaptive function that evolved to protect the organism during infection and/or inflammation. Indeed, as we discuss, immune effects on the CNS can be categorized as *systemic* stressors in order to distinguish them from stressors that are initially subjected to cognitive processes and are hence referred to as *processive* stressors.

At a fundamental level, stress is simply a relative physiological state that reflects a change or deviation from a condition of rest or inactivity. The "resting" state of cells in any biological system consists of the quiet manufacture of receptors, proteins, enzymes, and various other molecules that serve to accumulate vital resources that maintain the stability of the organism. In the case of the brain, this state maintains consciousness, muscle tone, cardiovascular and respiratory functions, and many other critical physiological functions. This also is critical for dealing with the imposition of stressors, as they elicit, through a series of neural relays and chemical transmitters, increased levels of activity within and between neurons. Therefore, when an organism is "stressed," biological functions are no longer "idle" and are subjected to greater energy demands and reliance on the pool of resources always available (and typically in abundance) to make and release neurotransmitters. And, of course, this is perfectly normal.

As stressors engage the brain, there is augmentation of mechanisms for vigilance and attention and arousal of neural circuits that implement strategies for action and goal-directed behavior (Anisman, Merali, & Stead, 2008). As such, what we call *stressors* are simply the problems most organisms face when ongoing behavior is interrupted or diverted by anticipated and unanticipated circumstances. In this way, stress is viewed as a *goal disruptor* (Cohen, Murphy, & Prather, 2019). But whether prepared or not, the brain fields responses that tackle these situations successfully. As an example, the brain's noradrenergic neurotransmitter system affects cortical neurons responsible for attention and decision making (Chandler, 2016). Because this neurotransmitter system is readily activated by stress (Anisman et al., 2008), it most likely is required—along with other neurotransmitters—to accommodate behavioral adjustments in the face of stressor exposure. Similarly, the hierarchy of neural systems that control movement (e.g., motor cortex, striatum, and cerebellum) are mobilized to act on specific stressor conditions exhibiting a range of purposeful and reflexive responses (e.g., freezing/immobility and/or increased locomotion). Ultimately, the "stress state" can be viewed as an amalgam of enhanced cognitive and emotional processing, altered motoric states, and changes in physiological systems that regulate metabolic and biological functions.

These stress-related changes can be considered functional and/or adaptive. However, their functionality can be compromised by failure to control, eliminate, and/or cope with stressors. This would serve to create persistent or chronic stressor exposure that imposes greater energy demands and a weakening or destabilization of the neurobiological stress response. To understand this, McEwen and colleagues introduced the concept of *allostatic load* (McEwen & Wingfield, 2003), which refers to the energy and resources required to return deviations in function to previously stable baselines, or what is commonly referred to as *homeostasis*. This concept implies that work is required not just to mount a stress response but also to restore physiological functions to normal. However, if allostatic mechanisms are persistently engaged because of continued stressor-induced deviations in function, then this previously normal "load" becomes an "allostatic overload," with the various corrective mechanisms in play now impaired and contributing to pathology (Fava et al., 2019). (See Lehrer, Chapter 2, this volume, for a further description of allostasis and allostatic overload.) Further below, we provide examples of counter-regulatory processes that serve to restore deviations in biological function.

Stressors Provoke the Release of Neurotransmitters

To facilitate functional neural–immune interactions, cytokines released by activated immune cells are registered by the brain in a process that involves modulation of neurotransmitters (Anisman et al., 2008). Among the many neurotransmitters in the brain, the monoamine transmitters (*monoamines* for short) have received the most attention in stress research. In fact, much of this was fueled by the monoamine theory of depression (Mulinari, 2012), wherein it was thought that either underutilization or poor availability of the monoamines accounted for mood fluctuations and behavioral despair. This has also figured prominently in efforts to explain some of the behavioral changes observed in response to immune-derived cytokines (Anisman et al., 2008).

The monoamines are norepinephrine (NE; also known as noradrenaline), dopamine (DA), and serotonin (also known as 5-hydroxytryptamine [5-HT]), and the neurons that release these neurotransmitters project to multiple sites in the brain. Many of these target sites overlap, such that a similar region (e.g., the prefrontal cortex) can receive input from all three types of neurons (i.e. 5-HT-releasing, NE-releasing and DA-releasing). Therefore, given such overlap, at any given time neurons in the target site are likely to be regulated simultaneously by a balance of NE, DA, and 5-HT levels. For example, noradrenergic input into the DA neurons of the ventral tegmental area (found in the midbrain) can influence reward processes, thereby implicating NE–DA interactions in anhedonia (Isingrini et al., 2016). Similarly, serotonergic modulation in the prefrontal cortex can suppress stress-elicited DA and NE changes, whereas treatment with the selective serotonin reuptake inhibitors (SSRIs) can diminish stressor-induced NE changes (Kaneko et al., 2016). Therefore, interactions between the monoamine neurotransmitters have important behavioral consequences, including changes in sleep, arousal, food ingestion, sexual behavior, reward, vigilance, and learning and memory. Given that the fundamental symptoms of depression include alterations in these behaviors, it is not surprising that the monoamine hypothesis for depression has shown considerable longevity.

In the mature brain, the release and action of monoamine neurotransmitters is controlled by regulatory neurotransmitters, namely, glutamate and gamma-aminobutyric acid (GABA). These amino acid transmitters represent the “accelerator” and “brake” systems in the brain. As such, they constitute the primary regulatory mechanisms by which increases and decreases in neuronal activity between neurons, and across the brain, are maintained. Both are ubiquitously distributed and present in all areas of the brain, especially those areas in which the monoamine neurotransmitter neurons originate and project. Glutamate is the chief excitatory neurotransmitter, which adds to and/or potentiates any excitatory influences of the monoamines. Alternatively, GABA—which, initially, in the immature prenatal and early postnatal brain, is thought to promote excitatory functions (Ben-Ari et al., 2012)—serves as the chief inhibitory neurotransmitter in the adult brain; its role is to slow down the activity of excitatory neurons. Although somewhat of an oversimplification, this generally captures the relative roles of GABA and glutamate, which can modulate the activity of monoamine-releasing neurons. For example, glutamatergic and GABAergic neurons are found in areas that contain DA neurons, such as those in the midbrain. Their differential influence on DA neurons can regulate either the release of DA or the inhibition of DA release in target areas, such as the nucleus accumbens and prefrontal cortex (Morales & Margolis, 2017). Both of these regions are known to drive goal-directed behavior, attention, and the experience of reward (Otis et al., 2017). Therefore, dysregulation of DA neuron activity in these areas, through the complex interplay between GABAergic and glutamatergic neurons, may induce anhedonia, poor motivation, and impaired cognitive focus—all indices of depression (Grace, 2016).

In fact, several conditions exemplify the hazards of an imbalance between the glutamatergic and GABAergic systems. For example, targeting the GABAergic receptor, GABA-A, with benzodiazepines is the primary purpose of anxiolytic pharmacotherapy. And the presence of seizures—as in epilepsy—is due to the loss of the "brake" system, as excitatory neuronal effects are allowed to proceed at rates and frequencies that result in loss of behavioral control (e.g., motor dysregulation, confusion, and loss of conscious processing of information, or the so-called blackout). In addition, with regard to the ability of people to regulate stress, a recent review of neuroimaging studies examined investigations of GABA and glutamate functioning in participants with posttraumatic stress disorder (PTSD; Averill et al., 2017). It was noted that variations in GABA levels occurred across different brain regions, including differential sensitivity to GABA receptor agonists. And although a reduction of GABA levels has been found (which would imply less opportunity for inhibition of neuronal activity), this is not a consistent finding. With regard to glutamate, only indirect evidence was provided—largely preclinical and implying that reduced astrocyte function leads to elevated glutamate concentrations in the brain, which would suggest a role for glutamate in facilitating PTSD (Averill, Averill, Kelmendi, Abdallah, & Southwick, 2018). More concrete evidence was provided by Holmes and colleagues (2017), who demonstrated, through positron emission tomography (PET) imaging, increased cortical expression of the mGluR5 metabotropic receptor for glutamate in patients with PTSD. To the extent that stimulation of mGluR5 promotes fear conditioning, whereas antagonism of mGluR5 has anxiolytic effects and attenuates learned fear responses (reviewed in Holmes et al., 2017), it is possible that excessive glutamate activity is associated with PTSD.

Another major neurotransmitter is acetylcholine (ACh), which was the first fully characterized neurotransmitter. It is the main neurotransmitter in the parasympathetic nervous system (PNS), as well as the major neurotransmitter at neuromuscular junctions, which form the interface between the peripheral nervous system and skeletal musculature, and thereby allows for CNS control of motor behavior. Release of ACh at neuromuscular junctions stimulates ACh-specific receptors expressed by myocytes (muscle cells). Immune-mediated impairment of myocyte ACh receptors, as occurs in the autoimmune disease myasthenia gravis, results in loss of motor control. Additionally, ACh is released by the vagus nerve, which forms part of the PNS, and controls the activities of the heart, lung, and gastrointestinal system. Vagal innervation of cardiomyocytes (heart cells) regulates myocardial contraction rate, slowing down heart rate as needed. This is mediated by ACh and is the basis of heart rate variability (HRV), which is the rate of change in heartbeat intervals. The impact of stress on HRV has been extensively studied and is the focus of other chapters in this volume (see Gevirtz, Chapter 6, and Lehrer, Chapter 10).

In the brain, ACh is involved in arousal and wakefulness, but it also plays a primary role in learning and memory (Blake & Boccia, 2018; Majdi, Kamari, Vafaee, & Sadigh-Eteghad, 2017). Maintaining high levels of ACh in people with mild cognitive impairment (MCI) may slow down the onset of Alzheimer's dementia, if MCI in a given individual is a prodromal condition. Indeed, the ACh receptor subtype known as the nicotinic receptor (because it was discovered to be stimulated by nicotine) has received a great deal of attention as a potential target for cognitive enhancement (Gould & Leach, 2014; Majdi et al., 2017; Yakel, 2014). Later we discuss how this receptor figures in the inflammatory response to a bacterial toxin.

Many of these neurotransmitter systems are sensitive indicators of neuronal adaptation in response to a variety of physical, psychological, and immune stressors. With regard to psychological stressors, greater serotonin release occurs in response to uncontrollable stressors when compared with a controllable stressor (Amat et al., 2005; Dolzani et al., 2016)—testimony to the higher utilization of neurotransmitters in the face of augmented cognitive and emotional challenges. The behavioral consequences of this increased monoamine utilization, however, may not be adaptive, as persistent inability to control stressor termination contributes to impairments in learning and potentiates anxiety-like behavior (Maier, 2015). The reasons for this are unclear, but it may result in desensitization of neurotransmitter receptors (Dolzani et al., 2016), which essentially limits the communicative potential of increased neurotransmitter output—that is, signaling of relevant neuronal circuits needed to optimize decision making and problem solving during stressful situations.

In the short term, however, increased neurotransmitter release is essential to drive behavior and physiological regulation, which otherwise would collapse in the face of stressor exposure. Under tonic conditions, synapses (points of communication between neurons) maintain steady-state concentrations of neurotransmitter. As circumstances demand, these concentrations change as neurons alter their electrochemical properties under the weight of increased levels of stimulation from outside and inside the organism. However, neurotransmitters need to be made (synthesized), packaged for quantal release into the synaptic cleft (the communicative space between one neuron and another), and then recycled and/or degraded. This requires energy (e.g., adenosine triphosphate [ATP], the major molecular instrument that drives energy reactions in all cells), enzymes that enable neurotransmitter synthesis, and many other molecules that are needed to maintain the continued availability and liberation of neurotransmitters (Ostergaard, Jorgensen, & Knudsen, 2018). Moreover, on postsynaptic membranes (those regions of the neuron that receive the neurotransmitter from the releasing presynaptic neuron), a high density of neurotransmitter receptors is available to induce, within the postsynaptic cell, a range of biochemical processes that will allow for the continued transmission of chemical information from one neuron to another within a given circuit. The reality is that hundreds of these circuits exist in the brain, comprising millions of neurons communicating within specific regions (called *local networks*) and across regions (networks acting as nodes that connect and integrate information). The ultimate result is the stuff of behavior and mental life. And critical to this process are the neurotransmitters (and their receptors), which maintain a pattern of flow throughout the brain that is well regulated and ensures clarity of behavioral action. This behavioral action is either energized and refined or disturbed after stressor exposure, which naturally increases the rate of communication in many of these circuits.

As mentioned earlier, continued stressor exposure with little control over stressor termination can impair behavior (Maier, 2015). Alternatively, stressors can also arouse and facilitate adaptive cognitive processes. A key example is the work of James McGaugh (2015), who showed that under mild stressor or anxiogenic conditions, cognitive function can be improved and memories optimally stored and/or retrieved. This finding highlights the adaptational value of brief stressor exposure. For instance, peripheral catecholamines (epinephrine and norepinephrine) are rapidly released by the sympathetic nervous system within seconds of stressor onset but cannot cross the blood–brain barrier to directly affect brain function. Rather, they can indirectly influence brain function via afferent vagal input to the brainstem (Lawrence, Watkins, & Jarrott, 1995; Miyashita & Williams, 2006). This vagal modulation facilitates an increase in norepinephrine and glutamate release in the brain to enhance vigilance and fear learning (Hassert, Miyashita, & Williams, 2004; King & Williams, 2009). Therefore, the "fight or flight" response commonly attributed to sympathetic activation is very much an adaptive cognitive state that occurs pursuant to or in parallel with mobilization of increased output by various physiological systems (e.g., cardiovascular, gastrointestinal, respiratory, renal, musculoskeletal, and neuroendocrine systems). Interestingly, the vagus was shown to be a relevant pathway by which immune cytokines activated the brain and produced sickness-like behavior (Dantzer et al., 2008). Therefore, if the vagus serves to drive CNS changes that promote arousal and greater attention to the environment, it may be the case that it offers the same advantage under conditions of immunological activation, which is known to improve consolidation of learning and memory (Woodruff, Schorpp, Lawrenczyk, Chakraborty, & Kusnecov, 2011).

Neuroendocrinology of the Stress Response: The HPA Axis

Immunological stimuli not only increase the turnover and release of neurotransmitters but, in so doing, they also activate specific regions of the brain known to be involved in cognitive and emotional functions, as well as modulation of the HPA axis. This is a critical neuroendocrine system that promotes elevations in glucocorticoid hormones known to regulate a wide range of physiological processes. Three major forebrain regions influence the hypothalamus and the HPA axis: amygdala, hippocampus, and prefrontal cortex. These play an important role in activation of the HPA axis, influenced to a large degree by the neurotransmitters we have discussed, the source of which can be found in the brainstem. Specifically, the major origins of noradrenergic (locus coeruleus), dopaminergic (substantia nigra and ventral tegmental area), and serotoninergic (raphe nucleus) neurons project axons to the aforementioned forebrain regions and, in so doing, regulate cortical, hippocampal, and amygdaloid functions to the degree that these can activate the hypothalamus and the HPA axis in particular (Myers, McKlveen, & Herman, 2012).

The hypothalamus has long been a focal point for studying stressor effects, as it also represents the primary integrative site for neural information that is cognitive and systemic (or physiological) in nature. It was also the first region identified as a target of immune-derived soluble mediators (Besedovsky et al., 1983). In humans, the hypothalamus is estimated to be one cubic centimeter in size and is responsible for mediating circadian rhythms, food intake, temperature regulation, reproductive behavior, and control of the endocrine and autonomic nervous systems. Emerging from the base of the hypothalamus is the pituitary gland, which sits inside a bony pocket called the *sella turcica* and is heavily vascularized to allow for the secretion of hormones into the circulation. The control of this hormonal release is regulated by the hypothalamus, which in turn is guided by physiological instructions operating through other brain regions, which include specific nuclei in the brainstem and, in other cases, the cortex, amygdala, and hippocampus (Lechan & Toni, 2000; Ulrich-Lai & Herman, 2009). Many of the pituitary hormones control growth, reproductive functions, bone development, thyroid function, metabolism, and gene regulation. This elevates the hypothalamus to the status of primary neural regulator of endocrine activity and constitutes the executive arm of the *neuroendocrine* system, with the final hormonal products of its activation arising from the target organs (e.g., testosterone from testes, estrogen from ovaries, thyroxine from the thyroid).

Neuroendocrine stress research has focused extensively on the HPA axis (Herman, 2018). This system requires an interplay between the paraventricular nucleus (PVN), a specialized group of neurons in the hypothalamus, and the anterior portion of the pituitary. Ultimately this interplay results in the release of a class of steroid hormones called *glucocorticoids* from the adrenal glands, fatty wedges of tissue affixed atop the kidneys, which are also the source of circulating adrenaline (or epinephrine). The primary glucocorticoid in humans is cortisol, whereas in rodents it is called *corticosterone* (Raff, 2016). Glucocorticoids act on two types of receptor: the aldosterone-selective mineralocorticoid receptor (MR) and the glucocorticoid receptor (GR). Both receptors are found in the cell cytoplasm and expressed in most tissues of the body, including the brain. Notably, relative to GRs, the MRs possess greater glucocorticoid-binding affinity and are the main binding site for glucocorticoids under nonstressed, basal conditions (De Kloet, 2014; De Kloet, Vreugdenhil, Oitzl, & Joëls, 1998). In contrast, under stressful conditions, glucocorticoid GR occupation is increased and serves as the main mechanism for restoring homeostatic deviations (De Kloet, 2014).

An elevation in circulating glucocorticoids (e.g., cortisol) can exert a rapid negative feedback influence on the hypothalamus and pituitary (Crosby & Bains, 2012). But a slower negative feedback influence can be exerted through actions on the hippocampus and medial prefrontal cortex glucocorticoid receptors (Hill et al., 2011). And within the amygdala, which is known to drive HPA axis activation, glucocorticoids mediate reduction of stress-induced neuronal excitability (Gray et al., 2015; Karst, Berger, Erdmann, Schütz, & Joëls, 2010). These negative feedback mechanisms not only rapidly terminate the HPA axis response but also limit damage to body tissues by prolonged cortisol exposure and attenuate anxiety due to stressor exposure (Gray et al., 2015). Under chronic stress situations, however, these feedback mechanisms are disrupted, increasing plasticity in the amygdala and neuronal dysfunction in the hippocampus, changes that can result in an "acceleration" of the stress response by the amygdala and loss of "braking capacity" by the hippocampus.

Whether in the brain or the immune system, the primary action of GR stimulation is regulation of gene activity, which can result in suppression of cell function (De Bosscher, Vanden Berghe, & Haegeman, 2003). On the other hand, if the genes being regulated or inhibited are *repressors* (segments of DNA that inhibit or "repress" activation of specific genes), then this may liberate hitherto restrained cellular activity (Newton, Shah, Altonsy, & Gerber, 2017). For the most part, however, glucocorticoids bear the connotation of being "inhibitors," exemplified in the clinical realm by treatment with synthetic versions of glucocorticoids (e.g., prednisone, hydrocortisone, dexamethasone), and which are classed as anti-inflammatory drugs. Alternatively, it has been proposed that glucocorticoids may also drive up inflammatory processes (Sorrells, Caso, Munhoz, & Sapolsky, 2009) and, by virtue of suppressing anti-inflammatory cytokines in the hippocampus, may potentially increase neuroinflammation and impair recovery of neurons from degenerative influences (Sorrells, Munhoz, Manley, Yen, & Sapolsky, 2014).

The release of glucocorticoids from the adrenal glands is dependent on increased blood concentrations of the pituitary hormone, ACTH (i.e., *a*drenocorti*c*o*t*ropic *h*ormone; also called *corticotropin*), which is induced by corticotropin-releasing hormone (CRH; also known as CRF, for corticotropin-releasing factor), a neuropeptide released from the neurosecretory cells of the PVN (Herman, 2018). These CRH neurons extend axons to a vascular bed located in the basal area of the hypothalamus called the *median eminence*. Here CRH enters the blood supply, traversing the median eminence and pituitary, and eventually extravasates (leaves the blood) in the pituitary to stimulate pituicytes that make and release ACTH into the blood (the hormone that causes glucocorticoid release from the adrenal cortex). Under these conditions, CRH is said to be an ACTH secretagogue (i.e. release-inducer), but interestingly it is not the only one. Other important neuropeptides, such as vasopressin and oxytocin, are capable of increasing ACTH release from the pituitary, although their role appears to be secondary to that of CRH; however, if released simultaneously, the actions of vasopressin and CRH can potentiate ACTH output (Romero & Sapolsky, 1996). Nonetheless, the primary role of CRH as an ACTH secretagogue is well established.

Subsequent to stressor exposure, CRH is often elevated in PVN neurons and the median eminence (Romero & Sapolsky, 1996), and this also occurs after administration of cytokines or immune activation (Berkenbosch, van Oers, Del Rey, Tilders, & Besedovsky, 1987; Kusnecov, Liang, & Shurin, 1999; Sapolsky, Rivier, Yamamoto, Plotsky, & Vale, 1987). Drugs that block CRH receptors will also reduce ACTH and corticosterone elevations in rodents after stress or immunological challenge (Aguilera, Nikodemova, Wynn, & Catt, 2004; Rossi-George, Urbach, Colas, Goldfarb, & Kusnecov, 2005). Interestingly, CRH-containing neurons of the PVN also regulate autonomic nervous system activity during stress (Busnardo, Tavares, Resstel, Elias, & Correa, 2010). Indeed, CRH has become the canonical stress neuropeptide, promoting both HPA and autonomic activity in response to stressors, as well as inducing a variety of behaviors normally associated with stress: anxiety, anorexia, and novelty-induced hyperactivity. However, changes in behavior are not thought to derive solely from the actions of CRH-producing neurons in the PVN; rather, stress-induced activation of CRH-producing neurons occurs in the amygdala, the bed nucleus of the stria terminalis (an extension of the amygdala), and the locus coeruleus (Kovács, 2013).

Amygdala

The amygdala is particularly important to the activation of the HPA axis, as well as in promoting stress-related behaviors, including anxiety and fear responses. Decades of animal research confirmed the primacy of the amygdala as the main neural body associated with fear and wariness. This was corroborated in human neuropsychological studies, as well as neuroimaging data (functional magnetic resonance imaging [fMRI] or PET studies) that highlighted greater amygdaloid activation in response to stimuli associated with fear (e.g., facial expressions of fright; Barrett, 2018; de Gelder, Morris, & Dolan, 2005; Marek, Strobel, Bredy, & Sah, 2013). Interestingly, some amygdaloid activation has been observed in response to positive emotions, but this is orders of magnitude less than the response to negative emotions. This speaks to the notion that the amygdala serves as an arousal mechanism, but one intimately connected to negative emotional states. The activation patterns in the amygdala may affect whether an individual tolerates or is overwhelmed by a given state of arousal. Part of this control is thought to include feedback circuits from the prefrontal cortex, via a closely aligned set of neurons found in the anterior cingulate. In neuroimaging studies of human participants, the anterior cingulate is active after emotional and attentional engagement (Carter, Botvinick, & Cohen, 1999; Davis, Taylor, Crawley, Wood, & Mikulis, 1997; Isomura & Takada, 2004), thereby suggesting that it's an important conduit to many subcortical arousal and cognitively related structures (e.g., amygdala and hippocampus). Studies have suggested that when there is weak inhibitory feedback to the amygdala from the prefrontal cortex and anterior cingulate, excessive arousal may ensue and result in higher levels of anxiety, as well as depression (Costafreda et al., 2013; Heinz et al., 2005; Pezawas et al., 2005). Subsequently, an increase in amygdaloid activity can result—through direct and indirect connections to the hypothalamus—in greater HPA axis activation, thereby resulting in elevated glucocorticoid levels (Ulrich-Lai & Herman, 2009).

Hippocampus

The hippocampus is a region well recognized in promoting learning and memory (Fanselow & Dong, 2010), but which also has important interactions with the amygdala and prefrontal cortex, thereby influencing emotional memory and responses to stress (LaBar & Cabeza, 2006; McEwen & Morrison, 2013). Moreover, the hippocampus is one of many structures that can influence the HPA axis, largely through indirect mechanisms.

It contains a high density of glucocorticoid receptors, which play a role in the delayed inhibition of the HPA axis (Myers et al., 2012). It is thought to do so through activation of glutamatergic neurons that activate GABA neurons projecting to the PVN (Myers et al., 2012; Radley & Sawchenko, 2011). This activation of GABA neurons may inhibit the PVN and reduce the activation of neurons responsible for releasing ACTH secretagogues (e.g., CRH) into the median eminence, which would then subsequently cause ACTH release from the pituitary.

Therefore, the hippocampus, which is noted for its importance in monitoring place and setting and consequently episodic and/or contextual memories, is also closely connected to emotional and neuroendocrine functions. Bidirectional connections between hippocampal and amygdaloid neurons confer affective meaning to events, and control of the HPA axis influences glucocorticoid elevations, which ultimately can feed back to stimulate glucocorticoid receptors on hippocampal neurons. The impact of glucocorticoids on the hippocampus may also influence consolidation of learning and memory, there being evidence for both facilitation of storage and impairment (Huzard et al., 2019; Kim, Pellman, & Kim, 2015). This may be potentially important in terms of the immune effects on HPA axis activation. Immunological influences (viz., cytokines) on cognitive functions typically occur under conditions of HPA axis activation. This raises the hypothesis that part of the immunologically mediated behavioral adjustment mentioned earlier with regard to sickness behavior may involve a sharpening of cognitive functions, as opposed to being an impairment. Although there is some support for this idea in the animal literature (Woodruff et al., 2011), it has been demonstrated that administration of the cytokine, interleukin-1 beta (IL-1β), or inflammatory inducers, such as lipopolysaccharide (LPS, which is derived from gram-negative bacteria), can impair learning and memory (Dantzer et al., 2008; Rachal Pugh, Fleshner, Watkins, Maier, & Rudy, 2001). At the same time, immune-related cytokines that are normally present at basal levels in the brain and/or periphery may be necessary for normal cognitive function (Avital et al., 2003; Goshen et al., 2007), a notion we turn to in our concluding remarks.

In summary, the HPA axis is a highly regulated neuroendocrine system that is affected by inputs from multiple brain areas. We have cited only the influence of the amygdala and hippocampus, although the prefrontal cortex is also an important regulator of a variety of subcortical forebrain regions. Although best identified as promoting executive functions—a general umbrella term for a host of different behaviors that include evaluating stimuli, forming judgments, regulating actions, and performing working memory (just to name a few)—it has also received considerable attention as a regulator of stress responses (McEwen & Morrison, 2013). The medial portions of the prefrontal cortex, in particular, can exert a negative influence on the HPA axis and ultimately reduce circulating levels of glucocorticoids (Myers et al., 2012). Moreover, neurons in the medial prefrontal cortex are also activated following exposure of animals to immunological stimuli (Serrats & Sawchenko, 2006), suggesting that higher order brain regions are engaged after immune activation. However, as the next section discusses, the transfer of immunological information to higher order cognitive centers is a secondary event, pursuant to initial activation of neuronal systems charged with the processing of internal or interoceptive physiological events.

Cytokines as Systemic Stressors

It has long been recognized that different stressors operating through alternative modalities can still produce activation of the HPA axis. This was noted by Herman and Cullinan (1997), who argued that stressors differentiated as *processive* and *systemic* utilize alternative pathways of influence to activate the HPA axis. *Processive* stressors are largely "top down" psychogenic and/or neurogenic stressors that involve some cognitive component that interprets and assesses the nature of a given stressful situation or stimulus. Such stressors operate through traditional sensory modalities, which impose cognitive awareness of stimulus presence and threat—for example, the sight or sound of a home intruder. Alternatively, *systemic* stressors arise from changes in the physiological *internal milieu* and are nicely exemplified by changes in glucose and insulin concentrations but also by the immunological state of the organism. These types of stimuli require interoceptive receptor mechanisms and can either involve passage of molecules directly into the brain—for example, by crossing the blood–brain barrier—or stimulation of afferent vagal nerve pathways that convey visceral information directly to the brain (Goehler et al., 1999; Rao et al., 2017).

Processive stressors activate the HPA axis through a top-down flow of information. This arises from cortical information processing and interpretation of the stressor, which is relayed via the prefrontal cortex to GABAergic and glutamatergic neurons regulating the activation status of hypothalamic PVN neurons that secrete CRH (Ulrich-Lai & Herman, 2009). As such, a processive stressor may exert greater control over the activities of the PVN by virtue of cognitive interventions, such as judgments and coping strategies generated by prefrontal neurons. A similar process of top-down control by the prefrontal cortex may operate with regard to amygdaloid and hippocampal activity (Ishikawa & Ishikawa, 2019; Marek, Sun, & Sah, 2019).

In contrast to processive stressors, systemic stressors bypass cognitive control (at the outset), as all influences on brain function originate from internal physiological systems. For example, a sudden rise in insulin can lower circulating glucose concentrations, and this can rapidly affect brain metabolic activity given that glucose is a critical fuel for neuronal activity. Not surprisingly, hypoglycemia can quickly result in delirium and loss of consciousness. Therefore, changes in metabolic and/or inflammatory status imply the presence of ongoing events that require immediate physiological attention and implementation of relevant efferent outputs, including an increase in cortisol and catecholamines after HPA axis and ANS activation. Indeed, both glucocorticoids and catecholamines will increase the generation of glucose from the liver (Kuo, McQueen, Chen, & Wang, 2015). Interestingly, the cytokine interleukin-1β (discussed further in a later section) not only activates the HPA axis and ANS but also regulates, from within the brain, neuronal consumption of glucose (Del Rey et al., 2016). Therefore, systemic stressors, whether metabolic or immune-derived, elicit regulatory feedback of physiological processes by way of neuroendocrine and autonomic activation that originates in the PVN of the hypothalamus. As such, hypothalamic PVN activation by systemic stressors is a "bottom-up" process (Ericsson, Kovacs, & Sawchenko, 1994; Herman & Cullinan, 1997).

Several mechanisms for such bottom-up signaling by the immune system have been proposed. These include stimulation of afferent components of the vagus nerve, utilization of specialized protein-based carriers that transport cytokines from the blood and into the brain, and, finally, areas of the brain, such as the area postrema (in the brainstem) that contain a weak blood–brain barrier, which allows large immune-derived molecules, such as cytokines, to stimulate local midbrain or brainstem neurons (Hosoi, Okuma, & Nomura, 2002; McCusker & Kelley, 2013). The afferent components (that send signals to the brain) of the vagus nerve terminate in the brainstem, where they activate neurons of the nucleus tractus solitarius (NTS). These neurons connect with forebrain regions and transmit, through direct and indirect pathways, ascending information to the amygdala and hypothalamus, thereby providing critical information about the chemical state of the body. Part of this NTS effect on forebrain activation may rely on neuronal connections to the locus coeruleus, which provides widespread noradrenergic projections to the cortex, amygdala, hippocampus, and hypothalamus. Needless to say, activation of these projections from the brainstem indicates that the initial PVN response to inflammatory cytokines can bypass top-down cortical processing. Neurochemically, this influence is mediated by ascending noradrenergic (i.e., norepinephrine releasing) projections from the brainstem to the PVN (Cunningham & Sawchenko, 1988). This ensures direct stimulation of adrenergic receptors that facilitate the eventual release of CRH-containing neurons (in the PVN; Ericsson et al., 1994). This is followed by release of ACTH from the pituitary and glucocorticoid regulation of the inflammatory events that in the first instance stimulated the brain. As we discuss in the later section on cytokines and the CNS, this has potentially important adaptive consequences for the organism.

An important question concerns the cognitive–emotional consequences of forebrain activation by bottom-up systemic immune stressors. If during the response to immune-derived systemic stressors there is cognitive awareness, this is likely to be delayed in onset and unlikely to be specific to any particular stimulus. As a result, whereas for processive stressors behavior is instrumental in deciding that an event is a threat (cognitive appraisal), systemic stressors alter brain function through a subsensory cascade of neural events that first involves systemic stimulation of vagal afferents and/or entry of cytokines into the brain. The appearance of new behavioral parameters of cognition and/or emotion (i.e., a change from some resting or baseline level of ongoing behavior) are a consequence of this initial bottom-up process. Typically, these behavioral changes are at first fundamental and involve altered motivational drives consistent with hypothalamic activation (e.g., reduced appetite or lethargy), but they can then engage higher order brain structures (e.g., cortico–hippocampal circuits) that allow perception of illness and associated changes in cognition. We noted earlier that animal studies showed immune activation to result in altered cognition, with deficits in learning and memory being reported in response to inflammatory stimuli, such as endotoxin. In animal studies, the use of endotoxin models (viz., LPS injections) or severe bacterial infections can produce excessive stress-like activation in the brain that may interfere with memory consolidation (Dantzer et al., 2008; Rachal Pugh et al., 2001). Similar findings are not widespread in human participants, although there have been reports of modest cognitive changes, mostly related to mood, though rarely affecting memory (Schedlowski, Engler, & Grigoleit, 2014).

In summary, therefore, there are multiple sources of stress in the brain. Cognitive or processive stressors are the type we commonly ascribe to human participants engaged in the daily business of social interactions and making ends meet. Challenges in the social and psychological domains strongly affect our cognitive and emotional circuits, as these are required to solve the problems of psychosocial engagement. However, the same brain regions will ultimately be engaged in response to physiological imperatives, such as infection-induced cytokine elevations. In general, because the immune system can induce activation of regions of the brain that are also activated by psychological stressors, this suggests that signals from the immune system serve as systemic stressors and, in so doing, promote behaviorally and physiologically adaptive functions.

THE IMMUNE SYSTEM: AN OVERVIEW

Up to this point, we have provided a general framework for the HPA axis stress response and, where appropriate, highlighted the involvement of immune-derived cytokines as systemic stressors. Here we more formally describe the cytokines and consider (1) how psychological stress in humans is associated with increased circulating cytokines and (2) how cytokines and/or immune activation signal CNS activation (such as the HPA axis) to regulate immunophysiological processes. But to understand cytokines, it is important first to consider them within the context of the immune system, so that the relevance of cytokines as key "intercellular messengers" can be appreciated. Therefore, we give a brief and simplified overview of the immune system strictly to provide context for the major source of cytokines in the body. For those unfamiliar with immunobiology, many useful introductory monographs and online resources are available to provide a more complete understanding of the immune system. For example, a particularly useful website is provided by the British Society for Immunology (*immunology.org*). Here, we provide just enough information to appreciate discussions relating to brain–immune interactions.

The immune system is a heterogeneous group of specialized cells—collectively referred to as leukocytes (or white blood cells)—that circulate throughout the body to protect against infectious disease. Furthermore, immune cells are classified as part of either the *innate* or the *adaptive* immune response, a convention that highlights distinct functional properties of different cells in relation to the type and timing of an immune response to specific pathogens. Innate immune cells—which include monocytes and macrophages—develop in the bone marrow within what is known as the *myeloid cell lineage*. Alternatively, the cells mediating the adaptive or acquired immune response consist mainly of *lymphocytes* and develop in the bone marrow and thymus gland within what is called the *lymphoid cell lineage*. Although this categorization into innate and adaptive responses may imply that there are parallel immune processes in operation after exposure to an infectious agent, in fact, cells in both compartments cooperate to engage and eliminate the pathogen.

Myeloid Cells

There are multiple types of myeloid cells, but most prominent are monocytes, which circulate in the blood and travel to most organs, where they can be retained permanently to become tissue macrophages. One function of macrophages is to detect a foreign substance (e.g., bacteria) and then engulf and degrade the cell through phagocytosis (literally "eating up"). This function is a key feature of phagocytic myeloid cells that is observed also in granulocytes, cells that also go by the name *polymorphonuclear cells* (PMNs).

Lymphoid Cells

The lymphoid lineage consists of two major types of lymphocytes: B cells and T cells. These cells reside mainly in lymph nodes, the spleen, and the thymus. When lymphocytes encounter antigen (a term used for any substance that stimulates an immune response), they respond in a number of ways, including destroying the invading microbe, self-duplication (through mitosis or cell division), and creating memory cells. Mitotic proliferation increases the pool of lymphocytes that can respond to an antigen, and when the antigen is cleared, the newly formed cells die through programmed cell death. What remains are the original cells, along with the memory cells that can respond more quickly and strongly should the antigen return (note that this is the purpose of vaccinations). A key feature of lymphocytes is the production of antibody, which is produced by B cells. This class of lymphocytes is critical to the long-term protection of the organism, as they can continue to make antibody for many months to years against a given antigen. In fact, the basis of vaccination is to ensure that B cells are primed and memory cells created that will quickly pepper a given virus (e.g., influenza) with antibody should an individual be infected.

The second main class of lymphocytes are T cells (for thymus-derived). These cells are critical in the fight against viral infection, as well as aiding B cells to make antibody. Consequently, there are various subtypes of T cells: cytotoxic T cells (Tc) and helper T cells (Th); these cells are also referred to as CD8 and CD4 T cells (i.e., the molecules CD8 and CD4 are expressed on cytotoxic and helper T cells, respectively). The cytotoxic $CD8^+$ T cell will attack and destroy any cell that is infected with virus, which includes self-tissue. Because viruses can take up residence in any particular organ, it is expected that Tc cells will initially lyse (kill and/or burst open) infected cells, which will then liberate the virus and expose it to antibody. The $CD4^+$ helper T cells, on the other hand, release specific types of cytokines that will regulate the activities of Tc cells, as well as promote B cell antibody responses to antigen. Without Th cells, the immune system is severely compromised. An example of this is acquired immunodeficiency syndrome (AIDS), which occurs by virtue of Tc cells destroying HIV-infected Th cells. This severely undermines the capacity of B cells to make antibody against other infectious agents, thereby contributing to an increased rate of infection in AIDS patients.

The Immune Response: Innate and Adaptive Immune Cell Cooperation

The immune system responds to any infectious challenge with a two-tiered approach: first, a generalized targeting and destruction of the pathogen by phagocytic myeloid cells (innate response), and second, a specialized attack on the specific invader (adaptive response). Lymphocytes represent the second part of this process, as these cells specifically recognize antigens on the surface of viruses and bacteria as foreign (non-self). This recognition results in a highly selective response that, after elimination of the infectious agents, retains memory for the antigen. The myeloid cells of the innate immune system are less selective and will respond to most pathogens in a nondiscriminative manner. However, they do possess pathogen recognition receptors (PRRs, such as the Toll-like receptor, TLR), that are responsive to pathogen-associated membrane patterns (PAMPs) expressed on microbial surfaces (Iwasaki & Medzhitov, 2010). Indeed, there are many types of TLRs expressed on myeloid cells, which has led to a numerical categorization (e.g., TLR1, TLR2). The endotoxin LPS, for example, stimulates macrophages by stimulating TLR4, and TLRs have been found to be expressed in the nervous system (Kumar, 2019b), providing further evidence of commonly evolved systems in the immune and nervous systems.

In responding to microbial invasion, myeloid cells, such as macrophages, represent the first line of defense that directly engages an infectious agent. Once the myeloid cells have degraded the pathogens, Th and B lymphocytes will be presented with molecular components of the degraded products, thereby allowing for the generation of billions of molecules of antibody that can recognize thousands of different components of the overall larger pathogen entity (e.g., the protein-rich envelope of viruses and the cell walls of bacteria). The actual antigen presentation is facilitated by cells of the myeloid lineage, as well as another cell, the dendritic cell, which is considered a professional antigen presenting cell (APC). These APCs serve up different protein fragments of the processed microbial agent to the Th cells, which will then secrete cytokines that allow B cells to increase their production of antibody.

CYTOKINES: REGULATORS OF IMMUNITY

The term *cytokine* stands for "cell messenger," which captures perfectly the role played by cytokines within the immune system. The varied cellular components of the immune system optimize their antimicrobial responses through different intercellular forms of communication that can amplify and regulate the magnitude and duration of the immune response. The first required form of communication is through cell-to-cell contact and recognition of specific cell-surface molecules. Subsequently, immune cells can convey additional signals to multiply and assume more aggressive roles (e.g., bacterial killing or antibody production) against the infectious agent. This additional signaling is mediated by soluble cytokines, which convey information that changes the functional state of a cell and ensures the success of an immune response, as well as its denouement. However, it has become apparent that cytokines not only have an impact on cells within the immune system but also act on a variety of other cell types, including those in the brain. In this sense, cytokines have come to be perceived as endocrine signaling molecules (i.e., acting at a distance through humoral, blood-borne means), which is contrasted with their more local actions in lymph nodes, where signaling is paracrine (affecting adjacent or neighboring cells) or autocrine (a cytokine feeding back on its cellular source to ensure more or less production of a specific cytokine).

As a general class of molecules, cytokines are proteins, peptides, or glycoproteins secreted by a wide range of cell types. This includes *immune cells,* such as *macrophages,* B lymphocytes, and T lymphocytes, as well as *endothelial cells,* fibroblasts, and various *stromal cells*. Functionally speaking, cytokines are signaling molecules that regulate various cellular functions, including proliferation, phagocytosis, antigen presentation, antibody release, cell death, and cytokine production in and of itself. These and many other functions act via functional cytokine receptors and influence cells that are distant (the endocrine effect) or adjacent to their source (the autocrine and paracrine effects). In addition, a subclass of cytokines, called *chemokines,* are able to recruit circulating leukocytes to a site of microbial accumulation or tissue damage. For example, during a response to a pathogen, tissue macrophages will release chemokines, which "flag down" leukocytes that are passing by and divert them to the site of inflammation, where they contribute to the elimination of the microbial antigen. It is now understood that chemokines operate not only in peripheral tissues but also in the CNS, where they can contribute to the development of the nervous system, as well as recruitment of leukocytes to the brain (Lopes Pinheiro et al., 2016; Trettel, Di Castro, & Limatola, 2020).

Receptors for cytokines are found on all cells of the immune system and have been found on neurons and glia of the nervous system. Moreover, cytokine receptors are found in soluble form, circulating in the blood and interstitial tissue. In part, their role is largely to compete with cell-bound receptors as cytokine concentrations increase, thereby regulating cytokine signaling of the target cells (Cameron, 2003). Alternatively, binding of cytokines to their particular soluble receptor can form a complex that enhances biological activity (Scheller, Chalaris, Schmidt-Arras, & Rose-John, 2011).

Inflammatory and Anti-Inflammatory Cytokines

It is common to classify cytokines as proinflammatory and anti-inflammatory. This terminology arose from within the context of an immune response to an infectious agent or even against self-tissue (as in autoimmunity). A proinflammatory cytokine may stimulate and augment an immune response, thereby accelerating the process of pathogen elimination. Associated with this may be fully adaptive and useful physiological changes, such as an increase in body temperature, metabolism, and neuroendocrine activation. Even changes in mood and feeling may be reflective of the need to engage in self-awareness and to adjust behavior to avoid excessive energy expenditure, movement, and social interaction; the typical behavioral profile of someone with the flu is a telling example of social withdrawal, irritability, impaired appetite, and cognitive confusion (Eccles, 2005). Whether elevations of inflammatory cytokines are driving such behaviors in the absence of actual infection remains to be determined and remains a driving hypothesis. However, the concern for inflammatory cytokine measures under aseptic conditions—when the individual has no known infections (and referred to as *sterile inflammation*)—is whether the cytokines are chronically elevated and, if so, whether they are doing more harm than good. Additionally, little is known in human studies regarding the degree to which measures of inflammation in the periphery (i.e., blood) reflect what might be present in the brain. That is, cytokines are known to enter the brain and can also induce further cytokine production within the brain. This de novo synthesis of cytokines in the brain may influence sickness behavior and cognitive function (Maier & Watkins, 1998), in addition to the influence exerted by the extra CNS cytokines produced by the immune system.

Anti-inflammatory cytokines serve to decrease and eventually terminate an immune response. Whereas inflammatory cytokines can raise the pool of reactive immune cells, anti-inflammatory cytokines can kill cells (through programmed cell death, known as *apoptosis*) or inhibit cell function, which includes reducing or blocking synthesis of other (proinflammatory) cytokines produced by T cells, macrophages, monocytes, and neutrophils (Cameron, 2003). Interestingly, some cytokines can play a dual role, such as IL-6 and tumor necrosis factor-α (TNFα). In the case of TNFα, this promotes inflammatory responses but can also inhibit the inflammatory response through a specialized receptor, TNF-RII (Arnett et al., 2001; Caminero, Comabella, & Montalban, 2011). Ultimately, the right balance between pro- and anti-inflammatory cytokines is necessary for a healthy and efficient immune response (and CNS function), as the induction of inflammatory cytokines initially is to promote effective cellular responses. Once this is achieved, the entire process needs to be curtailed by anti-inflammatory mechanisms, which can also include soliciting glucocorticoid release through activation of hypothalamic CRH neurons. Indeed, this can occur early during an immune response. For example, the T cell response to staphylococcal enterotoxins derived from the common bacteria, *Staphylococcus aureus,* promotes HPA axis activation through release of CRH induced by circulating TNFα (Rossi-George et al., 2005).

Cytokines can be placed into one of five major categories, which also lend their initials to specific cytokine nomenclature. These are interleukins (IL; origin—"between leukocytes"), interferons (IFN; origin—"interferes with virus"), tumor necrosis factors (TNF; origin—"causes cell death"), transforming growth factors (TGF; origin—"causes cell transformation or change in function or growth") and miscellaneous hematopoietin ("blood-forming") cytokines. The most commonly discussed cytokines are IL-1, IL-2, IL-4, IL-6, IL-10, TNFα, and IFN. Note that Greek nomenclature recognizes that there can be subtypes for each cytokine (e.g., IL-1α, IL-1β, TNFα, TNFβ, IFNα), although we focus here on the most common types. Of the above cytokines, IL-10 is considered the quintessential anti-inflammatory cytokine, as it downregulates most immunological processes, including proinflammatory cytokine production (Saraiva & O'Garra, 2010). One might say that it is the "glucocorticoid" of the immune system, given its general counter-regulatory effect on the immune response. Most other cytokines, on the other hand, tend to facilitate specific types of immune responses. For example, IL-4 and IL-6 can be both anti- and proinflammatory (Chen, Bozec, Ramming, & Schett, 2019; Junttila, 2018; Scheller et al., 2011).

Major cytokines produced by T cells are IL-2, IL-4 and IFNγ, with IL-2 and IFNγ being proinflammatory, as IL-2 (once called *T cell growth factor*) promotes lymphocyte proliferation and IFNγ induces molecular changes in cells to initiate responses against antigens. Interleukin-4, however, can be viewed as anti-inflammatory because it promotes mechanisms for reducing the magnitude of inflammatory responses, as well as refining antibody production by B cells. These different cytokine roles are part of the so-called Th1:Th2 balance of T cell–mediated immune responses. This refers to whether a given T cell–mediated immune response is predominated by different subtypes of T helper cells that promote inflammatory (Th1) or anti-inflammatory (Th2) responses. The cytokines IL-2 and IFNγ are considered to be Th1 cytokines, whereas IL-4 (and another cytokine, IL-13) is produced by Th2 cells. A skewing of the response in favor of Th2 cytokine production is considered anti-inflammatory, although such responses have been identified with pathological responses. For example, asthma is associated with increased production of IL-4 and IL-13, with IL-4 promoting allergic airway responses, whereas IL-13 induces increased mucus production (Gour & Wills-Karp, 2015). Another Th2-derived cytokine, IL-5, is also involved in promoting inflammatory responses (e.g., by innate immune cells called *eosinophils,* which have been strongly implicated in asthmatic allergic responses; Greenfeder, Umland, Cuss, Chapman, & Egan, 2001). Therefore, although Th2 cytokine responses are associated with anti-inflammatory functions, they can also contribute to inflammatory responses in certain diseases. Further below, we cite some evidence for stress-induced effects on Th2 cytokines.

The cytokines IL-1, IL-6, and TNFα are often viewed as the classic triumvirate of inflammatory cytokines; but note that TNFα and IL-6 do have the capacity to inhibit cell function, and in the brain they have been noted to have neuroprotective properties (Arnett et al., 2001; Li et al., 2009; Suzuki, Tanaka, & Suzuki, 2009). These cytokines are typically produced at high levels by cells of the innate immune system, although small amounts are generated by T cells and B cells (especially TNFα and IL-6). The predominant sources for these cytokines include monocytes, macrophages, and neutrophils—that is, cells of the myeloid lineage. What is most relevant is that these cytokines also constitute the primary immune-derived activators of the HPA axis. In fact, in a number of studies, it was noted that they can act synergistically, in that simultaneous administration of each cytokine can result in much greater effects than either alone would have on sickness-like behavioral and HPA changes (Brebner, Hayley, Zacharko, Merali, & Anisman, 2000).

EFFECTS OF STRESSORS ON HUMAN IMMUNITY AND INFLAMMATORY CYTOKINES

Human immune function is highly susceptible to stressor-induced perturbation. This is supported by an enormous literature derived from laboratory studies in which blood leukocytes have been assayed for a variety of functional parameters, most notably cell division (proliferation) and cytokine production, as well as susceptibility to infection and measurement of in vivo cytokine elevations (Fagundes, Glaser, & Kiecolt-Glaser, 2013; Glaser & Kiecolt-Glaser, 2005; Segerstrom & Miller, 2004). Analysis of several hundred studies in which immune measures were conducted in vitro after isolating leukocytes from blood samples concluded that, in healthy volunteers, acute or single-episode stressors (e.g., social engagement, public speaking, mental arithmetic challenges, and parachuting) can enhance Natural Killer (NK) cell function (a type of immune cell that is thought to attack cancer cells and/or virus-infected cells) but at the same time suppress the proliferation or expansion of B and T lymphocytes (Segerstrom & Miller, 2004). The adaptive significance of increased NK function is readily appreciated, whereas suppressed lymphocyte proliferation is more difficult to conceptualize. The immune system's ability to expand its pool of antiviral or antibacterial lymphocytes is an important tool. For this reason, suppressed proliferation induced by acute stress does not appear to be consistent with an adaptive change. Alternatively, the limiting of potential autoimmune reactivity, and hence avoidance of autoimmune disease, would be consistent with a stressor-induced suppression of lymphocyte proliferation. Paradoxically, however, this has been associated with increased in vitro cytokine production (viz., IL-6 and IFNγ), including the impact of social stressors (e.g., giving a lecture to a large student body) on increased salivary cytokine concentrations of TNFα, IL-2, and IL-4 (Filaire et al., 2011). Therefore, it has become clear that seeking to make sense of the adaptational meaning of stress-related immune changes is complicated by the variety of different components of immune function that have been assessed and what each measure might mean. Most certainly, increased cytokine production implies support for any ongoing immune cell activity, which ostensibly—as in the case of NK cells—might be preparing for an infectious viral challenge. However, this is complicated by well-established evidence that T cell–mediated inhibition of latent herpes virus expression or shedding (e.g., herpes simplex virus and Epstein–Barr virus [EBV]) is disrupted by the stress of academic exams and can lead to infectious mononucleosis (Glaser & Kiecolt-Glaser, 2005; Glaser, Rabin, Chesney, Cohen, & Natelson, 1999). Therefore, evidence that stress might elevate antiviral mechanisms such as NK cells does not provide reassurance that infection will not be precipitated by stressors.

Another example of seemingly protective or anti-inflammatory responses being associated with stress-induced pathology is asthma. It has long been known that asthma is influenced by emotional states and that up to 40% of asthmatic responses are influenced by psychological stress (Isenberg, Lehrer, & Hochron, 1992; Lehrer, Feldman, Giardino, Song, & Schmaling, 2002). As we noted earlier, asthma can be influenced by increased inflammation in the lungs, precipitated by allergens and other irritants (Gour & Wills-Karp, 2015). Interestingly, people with asthma who are exposed to acute or repeated stressors showed elevated levels of the Th2 cytokines IL-4 and IL-5 (Kang et al., 1997; Marin, Chen, Munch, & Miller, 2009). Given that both cytokines are associated with the pathophysiology of asthmatic responses (Boonpiyathad, Sözener, Satitsuksanoa, & Akdis, 2019; Gour & Wills-Karp, 2015; Greenfeder et al., 2001), it is likely that stress exacerbates asthmatic symptoms through an immune-mediated mechanism.

Immune function is also affected by chronic stress. Here the literature shows suppressive effects on T cell proliferation, with variable effects (reduced or not affected) on antibody responses to influenza virus and other antigens (Cohen, Miller, & Rabin, 2001; Miller et al., 2004; Pedersen, Zachariae, & Bovbjerg, 2009). If this did not also occur for acute stressors, the suppressive effect on T cell function would be understandable based on the notion that chronic stress is more likely to influence disease onset. Antibody responses to vaccines, however, are generally reduced, being related to increased loneliness and reduced social support (Pressman et al., 2005). Indeed, research by Cohen and colleagues (Cohen et al., 1998; Cohen et al., 2001) has shown that more prolonged experience of perceived stress increased the likelihood of developing symptoms for the common cold after intranasal rhinovirus inoculation. This was buffered by social support, in that having a social network reduced the impact of perceived stress on viral-induced cold susceptibility (Cohen et al., 1998). Interestingly, after rhinovirus infection, intranasal IL-6 and TNFα concentrations were increased by greater stressor-related threat (Cohen et al., 2012). Relatedly, greater cytokine output was supported by a recent meta-analysis of more than 49 human studies that measured a variety of different cytokines in response to acute laboratory stressors (e.g., social evaluation and mental challenges; Marsland et al., 2017). This revealed a consistent effect on elevated circulating levels (i.e., measured in blood plasma) of IL-1ß, IL-6, IL-10, and TNFα. Elevations in stimulated cytokines (blood leukocytes activated in vitro) were also observed, but mainly for IL-1ß, IL-4 and IFNγ. Notably, circulating levels of c-reactive protein (CRP), an inflammatory biomarker that is produced by the liver and linked to cardiovascular disease, was not found to be affected (Marsland et al., 2017). Importantly, however, it should be emphasized that aside from the studies that performed in vitro stimulation of leukocytes to induce cytokine production, in vivo cytokine measures (in blood serum or plasma) are conducted without a knowledge of the cellular source the particular cytokine measured. Certain cytokines, such as IL-6, are known to be produced by nonimmune cells. For example, IL-6 can be produced by the adrenal or the pituitary (Zhou, Kusnecov, Shurin, DePaoli, & Rabin, 1993). Moreover, in animal studies, stressor exposure increased IL-6 (without the need for infection) in the liver, but not in immune organs, such as the spleen (Kitamura et al., 1997). Therefore, stressor-induced increases in circulating IL-6 may not necessarily be of immune origin.

Overall, in the absence of additional evidence (e.g., collecting leukocytes and measuring intracellular levels of cytokines), it will remain a matter of speculation whether in humans stressor-induced elevations in circulating cytokines are due to a direct effect of neurohormonal systems (e.g., epinephrine) on immune cells. Indeed, Marsland and colleagues (2017) noted that no elevations of circulating IL-2 or IFNγ were observed after stressor exposure. As these cytokines are largely derived from T lymphocytes, it is unlikely that direct antigen-independent activation and induction of cytokines occurs in lymphocytes after stressor exposure.

Whatever the cellular origin of circulating cytokines following a psychological stressor, their functional significance in this context has not been explored in human studies. Therefore, one can only speculate on the reasons for this generally consistent finding. One cytokine, IL-6, has received particular attention, as it has been linked to depression, and a recent animal study has suggested that after chronic social stress, IL-6 in the blood may influence the brain to produce a depressive-like profile (Menard et al., 2017). This provides credence to the notion that in humans, circulating cytokines may indeed influence neurobiological functions, and especially those pathways related to stress.

EFFECT OF CYTOKINES ON THE HPA AXIS

The cytokine IL-1β was the first to be implicated in neuroendocrine activation and, ultimately, CNS modulation (Besedovsky, del Rey, Sorkin, & Dinarello, 1986; Shintani, Nakaki, Kanba, Kato, & Asai, 1995). Relative to other cytokines, IL-1β is orders of magnitude more powerful in affecting behavioral, endocrine, and autonomic effects after administration, as minute quantities (picogram amounts) of IL-1β have been administered to animals with significant behavioral and neural consequences (Anisman & Merali, 1999; Besedovsky et al., 1991). There are two main forms of IL-1: IL-1α and IL-1β. The former is typically retained in the cell membrane and rarely secreted in high amounts; in contrast, the beta form is readily secreted, and this is the chief neuromodulatory form of IL-1. For the better part of a decade after the initial observations of Besedovsky and colleagues (Besedovsky et al., 1986), a large number of studies were conducted that further characterized the effects of IL-1 on the HPA axis (Turnbull & Rivier, 1999). These studies reported that other cytokines, such as TNFα and IL-6, were effective stimuli for HPA axis activation. However, many of these studies utilized recombinant forms of cytokines and, in a majority of cases, the human form. Information on endogenous cytokines was not as heavily investigated.

To study the role of endogenous proinflammatory cytokines, experimental studies opted for LPS (also known as endotoxin) derived from the cell walls of gram-negative bacteria, such as *E. coli*. Administration of LPS to animals stimulates macrophages and results in high circulating levels of IL-1β, with associated neuronal activation and monoamine release in many of the regions previously discussed: amygdala, hypothalamus, prefrontal cortex, and hippocampus (Hayley, Lacosta, Merali, van Rooijen, & Anisman, 2001). In addition, there is marked activation of the HPA axis, along with increased ACTH and corticosterone elevations (Beishuizen & Thijs, 2003). In humans, low-dose endotoxin treatment has been shown to elevate cortisol, and although measures of IL-1β were lacking, IL-6 and TNFα were elevated (Engler et al., 2016). Similarly, work by Lowry and colleagues (Alvarez et al., 2007) has shown in human volunteers that administration of low-dose endotoxin will elevate cortisol, and this was associated with increased TNFα and IL-6 levels. Interestingly, they also found that TNFα production was steroid sensitive (i.e., easily suppressed by glucocorticoid treatment), but IL-6 was steroid insensitive, being resistant to glucocorticoid inhibition. This suggests that cytokines are not equally regulated by glucocorticoids.

Attending the neural and endocrine effects of endotoxin are pronounced sickness behaviors, including hypophagia, reduced social/environmental exploration and movement, and changes in cognitive function, which have also been noted in humans (Dantzer et al., 2008; Schedlowski et al., 2014). Administration of IL-1β replicates these effects of endotoxin, suggesting that IL-1β is the primary mediator of the CNS effects induced by activation of macrophages with LPS. Moreover, antagonism of IL-1 after endotoxin treatment can fully or partially attenuate some of these effects. Partial attenuation speaks to the fact that the neural effects of endotoxin exposure can involve more than one cytokine. As mentioned earlier, IL-1β, TNFα, and IL-6 can exert synergistic effects (Brebner et al., 2000), suggesting that when they are induced together—as can occur in response to endotoxin—an integrated influence is exerted on the CNS. Indeed, in a classic mouse study, it was shown that in response to LPS, optimal release of ACTH from the pituitary was dependent on all three cytokines being present in the circulation. Blocking the actions of any one of the triumvirate significantly reduced ACTH output; however, complete blockade of the ACTH response required all three cytokines to be neutralized (Perlstein, Whitnall, Abrams, Mougey, & Neta, 1993).

As mentioned earlier, the benefit of cytokine-induced activation of the HPA axis is elevation of glucocorticoid blood levels. This effect serves to regulate ongoing immunological activity, ensuring that immune responses are restrained and the development of pathology, such as autoimmune disease, is prevented. This effect of HPA axis activation was demonstrated long ago using a variety of animal species that were prone to develop autoimmune thyroiditis or experimental allergic encephalomyelitis, a model of multiple sclerosis (Del Rey, Klusman, & Besedovsky, 1998). Moreover, IL-1 effects on HPA axis activation appear to represent one such neuroendocrine mechanism for imposing control over the immune response. For example, IL-1 is known to potentiate T and

B cell responses to antigen. However, when this potentiation also involved parallel IL-1-induced activation of the HPA axis and an elevation in blood corticosterone, the latter imposed a limitation on the magnitude of antibody produced to a foreign protein antigen (Kusnecov & Rossi-George, 2001).

Given the importance of glucocorticoids in regulating autoimmunity (Straub & Cutolo, 2016), the ability of the immune system to increase glucocorticoid production through HPA axis activation represents a self-regulatory protective mechanism that limits erroneous immune responses against self-tissue. Such anti-inflammatory effects of inducing endogenous glucocorticoids are also in keeping with other evidence that, when normally safe levels of endotoxin are administered to animals rendered incapable of generating a glucocorticoid response, morbidity and mortality were significantly augmented (Beishuizen & Thijs, 2003; Quatrini et al., 2017). The glucocorticoid response, therefore, is a major source of physiological regulation that can rescue the organism from an overly exuberant immune response. This is in keeping with the well-documented ability of glucocorticoids to suppress most aspects of the immune response, from innate immune cells to T and B lymphocytes (Cain & Cidlowski, 2017).

However, it should be mentioned that glucocorticoids do not always operate in the service of suppressing inflammatory responses. For example, glucocorticoid actions in the brain may augment proinflammatory effects of LPS administration in rats (Munhoz, Sorrells, Caso, Scavone, & Sapolsky, 2010). Moreover, in a model of excitotoxic neural injury, glucocorticoids suppressed chemokine factors that normally restrain other inflammatory cytokines, thereby enhancing the neurotoxic effects of inflammation (Sorrells et al., 2014). In addition, low levels of glucocorticoids can enhance inflammatory gene expression, whereas the inflammatory response to LPS is augmented if glucocorticoids modulate immune cells prior to endotoxin challenge (Cain & Cidlowski, 2017). Therefore, although the bulk of evidence supports glucocorticoids as a major anti-inflammatory influence, it is generally not a guarantee that they will always be effective in suppressing unwanted proinflammatory events.

Cholinergic Influences within the Immune System

In activating the HPA axis, the immune system elicits counter-regulatory glucocorticoid effects that generally control the magnitude of the inflammatory response. A similar effect of a CNS-derived feedback regulation has been noted for activation of the parasympathetic and sympathetic branches of the ANS. Immune cells—in particular, macrophages—express the α7 nicotinic receptor for acetylcholine (α7nAChR; Pavlov & Tracey, 2012; Wang et al., 2003). Recall that acetylcholine (ACh) is the principal neurotransmitter of the parasympathetic nervous system and is released from the vagus nerve to slow down cardiac activity. In animals that lack the α7nAChR, endotoxin treatment induced hyperelevated TNFα production, the consequence of which was increased morbidity (Wang et al., 2003). Moreover, vagal stimulation is required to inhibit the TNF response if the α7nAChR is present. Therefore, endotoxin-induced cytokine responses activate central mechanisms controlling the vagus nerve, providing a regulatory cholinergic mechanism for preventing morbid pathology. An extension to this finding was that sympathetic activation through release of norepinephrine in the spleen serves an important intermediary function. The release of NE in the splenic nerve stimulates β2 adrenergic receptors on T cells in the spleen. In a study by Tracey and colleagues (Rosas-Ballina et al., 2011), this was shown to result in T cell release of ACh (notably a non-neuronal source of ACh), which then kept TNF levels in check through stimulation of macrophage α7nAChR. Overall, therefore, in response to endotoxin treatment (a systemic stressor), feedback regulation of the inflammatory response can be restrained not only by glucocorticoids but also by cholinergic mechanisms. The implications of this for the psychological stress response are somewhat speculative at present. However, these findings do suggest that T cell–derived ACh may be induced by psychogenic stressors that cause increased sympathetic release of norepinephrine. At present, however, there are no reports that this occurs.

GLIA: THE CELLULAR BASIS OF NEUROINFLAMMATION

It is well known that IL-1β, TNFα, and IL-6 are expressed in the brain. The major cellular source of these cytokines is likely glial cells, which include activated microglia, perivascular macrophages (that surround cerebral blood vessels), oligodendrocytes, and astrocytes. Glial cells (apart from the perivascular macrophages) are often considered the accessory cells of the nervous system that support the functions of neurons and that, under specific conditions, can alter the course of neuronal activity. Oligodendrocytes provide the fatty white matter of the brain, known as myelin, which serves to increase the speed of communication between neurons. They are also structurally important to the extent that myelin loss results in neuronal degeneration, as well as impaired motor function (e.g., witness the motor dysfunction observed in demyelinating diseases such as multiple sclerosis). Astrocytes support the blood–brain barrier, regulate metabolic activity in the brain and neurotransmitter synthesis (e.g., glutamate and GABA), and produce cytokines (including the chemokines; Sofroniew, 2014). Although astrocytes are likely to play an important role in affecting neuronal function through cytokine release, microglia have dominated the spotlight in recent years, being referred to as the resident immune cells of the brain.

Figure 4.1 shows histological images of mouse microglial cells in the hippocampus. These cells are often present in high numbers and in an activated form under neurodegenerative conditions, such as Alzheimer's and Parkinson's disease (Wolf, Boddeke, & Kettenmann, 2017). Moreover, they are also activated by exposure to repeated psychogenic stress (Sugama, Takenouchi, Fujita, Conti, & Hashimoto, 2009; Walker, Nilsson, & Jones, 2013), which may occur via their expression of receptors for local neurotransmitters (Pocock & Kettenmann, 2007).

Microglial cells perform many of the functions ascribed to macrophages (including phagocytosis), which is why they are often regarded as the brain's resident immune cells. Given that immune function is much more than cytokine production and phagocytosis, this is perhaps an overcategorization, but nonetheless it conveys the point: These cells sweep the immediately surrounding area for any signs of damage or threat. In performing this surveillance, they stand ready to act in a phagocytic capacity, but they also may regulate neuronal functions in the process. For example, in response to injury, microglial cells display rapid chemotactic responses in which they extend their processes and display movement toward the site of injury (Davalos et al., 2005). This is not only a response to danger but also very likely an effort to "clean up" and repair any damaged synapses. Indeed, during prenatal, early postnatal, and adolescent development, microglia are active in shaping neuronal networks by pruning synapses to ensure optimal numbers of connections in the brain (Mallya, Wang, Lee, & Deutch, 2019; Schafer et al., 2012). This evidence has been used to promote the hypothesis that such conditions as autism and schizophrenia—in which brain development is known to be altered—may perhaps be

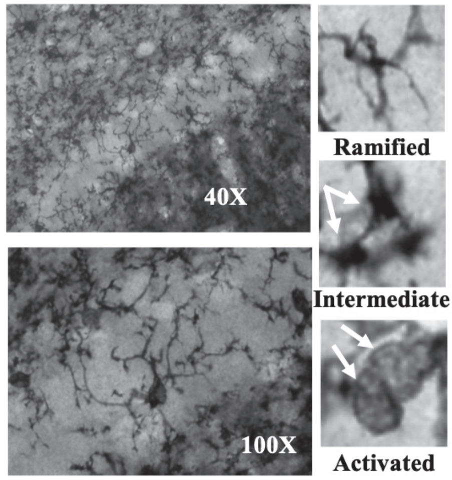


FIGURE 4.1. Images of microglia in the mouse dentate gyrus (DG) region of the hippocampus, in which brain tissue sections were subjected to immunohistochemistry using an antibody that recognizes a cell surface marker, CD11b, found on microglial cells. Images are provided at different microscope magnifications (40× and 100×), with zoomed-in images of individual cells from 100× image capture. These latter images illustrate how microglia assume different morphologies that are thought to reflect different activational states. In the "activated" state shown, microglia retract their processes, inflate their cell body, and assume an ameboid shape that is associated with phagocytosis, a state that allows them to "eat up" or ingest particulate matter and cellular debris. This function is thought to be involved in synaptic pruning, as mentioned in the chapter. In the ramified state, processes are in constant motion as they survey the surroundings and synapses where neurotransmitter is released.

due to altered microglial cell activity (De Picker, Morrens, Chance, & Boche, 2017; Yin, Chen, Sathyamurthy, Xiong, & Mei, 2012).

The degree to which the neuronal impact of microglial cells results in adaptational changes is not fully understood. To be sure, it has been suggested that greater activation of microglia after chronic stress may impair working memory in rats (Walker et al., 2013); and in mice, chronic stress induced behavioral indices of anxiety and depression through increased monocyte trafficking to the brain, as well as microglial activation (Ramirez, Fornaguera-Trias, & Sheridan, 2017). In addition, mouse experiments showed that deficiencies in microglial development and function (e.g., less recruitment during neurodevelopment and reduced synaptic pruning) globally throughout the brain can similarly influence anxiety and depression (Winkler et al., 2017). This suggests that normal behavior is dependent on microglial functions operating within a circumscribed range outside of which perturbation of neuronal function is more easily achieved.

Human studies of stress-related microglial activation have been limited to using markers that can be visualized through neuroimaging procedures, such as PET. As microglia progress from a resting to an activated state, they upregulate the expression of a specific translocator protein (TSPO) that supports production of cytokines. The radiotracer [^{11}C]PBR28, binds TSPO and can serve as a molecular marker of microglial activation as measured by PET (Tronel et al., 2017). Human participants and nonhuman primates exposed to low concentrations of endotoxin were shown to increase [^{11}C]PBR28 binding to TSPO, suggesting that low, subclinical peripheral inflammation can affect microglia (Hannestad et al., 2012; Sandiego et al., 2015). However, in human participants diagnosed with depression or who displayed risk factors for depression, no significant changes in [^{11}C]PBR28 binding was observed (Dahoun et al., 2019; Hannestad et al., 2013). These studies, although potentially useful, do not reveal whether [^{11}C]PBR28 binding also reflects the cytokine-producing state of microglia, nor whether the activational status of microglia plays a role in affecting the overall neurobiological response to stressors that might trigger mood changes. Moreover, an additional problem is that TSPO expression is observed in nonmicroglial cells (e.g., astrocytes), which limits the specificity of these PET studies exclusively to microglia (Tronel et al., 2017). Because depression and stress have been associated with an increase in circulating cytokines, it appears that this may not be associated with microglial activation, or at the very least, markers of activation. This stands in contrast to postmortem studies of increased microglial density in the brains of people with depression who have committed suicide (cited in Hannestad et al., 2013). As further studies are conducted, more information may come to light regarding the activational status of microglia under conditions of depression and stress in human participants. To the extent that animal studies are quite clear in demonstrating that stressors elevate brain cytokines and induce changes in microglial activation, it is conceivable that similar outcomes might occur in the human brain.

CONCLUSION

An enormous literature has been gathered to show that stress in both animals and humans influences immune function. This was briefly reviewed in the current chapter, given that many such reviews have appeared over the years. What is perhaps a more important question is why the immune system would be affected by psychological stress. The answer to this question comes from the immune system itself. Since the early days of psychoneuroimmunology, a growing body of evidence revealed that immune cells, whether macrophages or T and B cells, express receptors for many of the neurotransmitters, neuropeptides, and hormones that are either found in the brain or controlled by neural mechanisms (e.g., neuroendocrine system and the ANS; Steinman, 2004). From an evolutionary perspective, these receptors were retained on immune cells as a natural advantage, conferring a mechanism by which the brain—when subjected to heightened arousal, as would occur during psychological stress—might be able to regulate ongoing immune activity. In the short term, it is unlikely that this regulation is unwelcome, as immune cells generate an array of signals that elicit the same neurobiological changes induced by processive or cognitive stressors. As the consequences of the neurobiological response involve HPA axis and ANS activation, and given that these protect the organism in the throes of infectious battle, it must be concluded that the brain is as much a part of the immune or inflammatory response against infection as is the primary mechanism: cells of the immune system. That is, psychological stressors likely affect the immune system as part of an initial adaptive response. However, if this is a recurring event, then immunological function may become compromised, accounting for evidence of increased susceptibility to infection following chronic stress.

We paid less attention to the behavioral changes that are involved after immune activation—such as inactivity and reduced social exploration, anhedonia, and anorexia (see Anisman & Merali, 1999). But it is worth mentioning that these also represent behavioral adjustments that indicate a motivational state of self-protection, and perhaps even protection of the group. Sick animals in isolation keep to themselves, reducing harm—by way of infection—to others. This is adaptive for all concerned. The individual organism rests and battles infection, and the group can remain in an infection-free zone. However, we also note that infectious states are not the only drivers of behavior, as it was recently reported that development of social behavior in animals was dependent on the T cell–derived cytokine, IFNγ (Filiano et al., 2016). Specifically, animals that lacked IFNγ showed a significant deficit in social exploration. Indeed, it may be the case that the immune system has the capacity to influence the development of a nervous system with the proclivity for social affiliation and interaction. What advantage might this serve? One can only speculate, but theories of herd immunity are predicated on the existence of social behavior (Fine, Eames, & Heymann, 2011). The development of common exposure to and protection against the same infectious organisms can be ensured by social interactions and would promote long-term survival of the community. Contemporary life ensures this through vaccination programs, but at one point sitting around the campfire may have been the best solution. That the biological development of the immune system and its various cellular and molecular players would also shape the development of critical mechanisms in the brain that are of communal value is surely a clever advantage. It has implications not only for theories of autism spectrum disorders but also for other psychiatric conditions in which social isolation is an underlying feature.

Putting this aside, however, it is evident that behavior and CNS function, and the various challenges to which the brain is subjected on a daily basis, both affect the immune system and are in turn influenced by immune-derived cytokines. This bidirectional interaction appears to be a functional reciprocal relationship that benefits the organism and ensures health. Disrupting this relationship, on the other hand, may result in pathology. Consequently, future investigations may best be served by a focus on the established *links* (and their maintenance) between the brain and the immune system. When such connections are impaired, disease may follow.

ACKNOWLEDGMENTS

This work was supported by U.S. Public Health Service Grant No. MH108994 and the Brain Health Institute of Rutgers University.

REFERENCES

Ader, R., Cohen, N., & Felten, D. (1995). Psychoneuroimmunology: Interactions between the nervous system and the immune system. *Lancet, 345*(8942), 99–103.

Aguilera, G., Nikodemova, M., Wynn, P. C., & Catt, K. J. (2004). Corticotropin-releasing hormone receptors: Two decades later. *Peptides, 25*(3), 319–329.

Alvarez, S. M., Katsamanis Karavidas, M., Coyle, S. M., Lu, S. E., Macor, M., Oikawa, L. O., et al. (2007). Low-dose steroid alters in vivo endotoxin-induced systemic inflammation but does not influence autonomic dysfunction. *Journal of Endotoxin Research, 13*(6), 358–368.

Amat, J., Baratta, M. V., Paul, E., Bland, S. T., Watkins, L. R., & Maier, S. F. (2005). Medial prefrontal cortex determines how stressor controllability affects behavior and dorsal raphe nucleus. *Nature Neuroscience, 8*(3), 365–371.

Anisman, H., Hayley, S., & Kusnecov, A. (2018). *The immune system and mental health*. London: Elsevier.

Anisman, H., & Merali, Z. (1999). Anhedonic and anxiogenic effects of cytokine exposure. *Advances in Experimental Medicine and Biology, 461,* 199–233.

Anisman, H., Merali, Z., & Stead, J. D. (2008). Experiential and genetic contributions to depressive- and anxiety-like disorders: Clinical and experimental studies. *Neuroscience and Biobehavioral Reviews, 32*(6), 1185–1206.

Arnett, H. A., Mason, J., Marino, M., Suzuki, K., Matsushima, G. K., & Ting, J. P. (2001). TNF alpha promotes proliferation of oligodendrocyte progenitors and remyelination. *Nature Neuroscience, 4*(11), 1116–1122.

Averill, L. A., Averill, C. L., Kelmendi, B., Abdallah, C. G., & Southwick, S. M. (2018). Stress response modulation underlying the psychobiology of resilience. *Current Psychiatry Reports, 20*(4), 27.

Averill, L. A., Purohit, P., Averill, C. L., Boesl, M. A., Krystal, J. H., & Abdallah, C. G. (2017). Glutamate dysregulation and glutamatergic therapeutics for PTSD: Evidence from human studies. *Neuroscience Letters, 649,* 147–155.

Avital, A., Goshen, I., Kamsler, A., Segal, M., Iverfeldt, K., Richter-Levin, G., et al. (2003). Impaired interleukin-1 signaling is associated with deficits in hippocampal memory processes and neural plasticity. *Hippocampus, 13*(7), 826–834.

Barnes, P. J. (2018). Targeting cytokines to treat asthma and chronic obstructive pulmonary disease. *Nature Reviews Immunology, 18*(7), 454–466.

Barrett, L. F. (2018). Seeing fear: It's all in the eyes? *Trends in Neurosciences, 41*(9), 559–563.

Beishuizen, A., & Thijs, L. G. (2003). Endotoxin and the hypothalamo–pituitary–adrenal (HPA) axis. *Journal of Endotoxin Research, 9*(1), 3–24.

Ben-Ari, Y., Woodin, M. A., Sernagor, E., Cancedda, L., Vinay, L., Rivera, C., et al. (2012). Refuting the challenges of the developmental shift of polarity of GABA actions: GABA more exciting than ever! *Frontiers in Cellular Neuroscience, 6,* 35.

Berkenbosch, F., van Oers, J., Del Rey, A., Tilders, F., & Besedovsky, H. (1987). Corticotropin-releasing factor-producing neurons in the rat activated by interleukin-1. *Science, 238*(4826), 524–526.

Besedovsky, H. O., Del Rey, A., Klusman, I., Furukawa, H., Monge Arditi, G., & Kabiersch, A. (1991). Cytokines as modulators of the hypothalamus–pituitary–adrenal axis. *Journal of Steroid Biochemistry and Molecular Biology, 40*(4–6), 613–618.

Besedovsky, H., Del Rey, A., Sorkin, E., Da Prada, M., Burri, R., & Honegger, C. (1983). The immune response evokes changes in brain noradrenergic neurons. *Science, 221*(4610), 564–566.

Besedovsky, H., Del Rey, A., Sorkin, E., & Dinarello, C. A. (1986). Immunoregulatory feedback between interleukin-1 and glucocorticoid hormones. *Science, 233*(4764), 652–654.

Blake, M. G., & Boccia, M. M. (2018). Basal forebrain cholinergic system and memory. *Current Topics in Behavioral Neurosciences, 37,* 253–273.

Boonpiyathad, T., Sözener, Z. C., Satitsuksanoa, P., & Akdis, C. A. (2019). Immunologic mechanisms in asthma. *Seminars in Immunology, 46,* 101333.

Brebner, K., Hayley, S., Zacharko, R., Merali, Z., & Anisman, H. (2000). Synergistic effects of interleukin-1 beta, interleukin-6, and tumor necrosis factor-alpha: Central monoamine, corticosterone, and behavioral variations. *Neuropsychopharmacology, 22*(6), 566–580.

Busnardo, C., Tavares, R. F., Resstel, L. B., Elias, L. L., & Correa, F. M. (2010). Paraventricular nucleus modulates autonomic and neuroendocrine responses to acute restraint stress in rats. *Autonomic Neuroscience, 158*(1–2), 51–57.

Cain, D. W., & Cidlowski, J. A. (2017). Immune regulation by glucocorticoids. *Nature Reviews Immunology, 17*(4), 233–247.

Cameron, M. J. (2003). Cytokines and chemokines—Their receptors and their genes: An overview. *Advances in Experimental Medicine and Biology, 520,* 8–32.

Caminero, A., Comabella, M., & Montalban, X. (2011). Tumor necrosis factor alpha (TNF-alpha), anti-TNF-alpha and demyelination revisited: An ongoing story. *Journal of Neuroimmunology, 234*(1–2), 1–6.

Capuron, L., & Castanon, N. (2017). Role of inflammation in the development of neuropsychiatric symptom domains: Evidence and mechanisms. *Current Topics in Behavioral Neurosciences, 31,* 31–44.

Capuron, L., Gumnick, J. F., Musselman, D. L., Lawson, D. H., Reemsnyder, A., Nemeroff, C. B., et al. (2002). Neurobehavioral effects of interferon-alpha in cancer patients: Phenomenology and paroxetine responsiveness of symptom dimensions. *Neuropsychopharmacology, 26*(5), 643–652.

Carter, C. S., Botvinick, M. M., & Cohen, J. D. (1999). The contribution of the anterior cingulate cortex to executive processes in cognition. *Reviews in the Neurosciences, 10*(1), 49–57.

Chandler, D. J. (2016). Evidence for a specialized role of the locus coeruleus noradrenergic system in cortical circuitries and behavioral operations. *Brain Research, 1641*(Pt. B), 197–206.

Chen, Z., Bozec, A., Ramming, A., & Schett, G. (2019). Anti-inflammatory and immune-regulatory cytokines in rheumatoid arthritis. *Nature Reviews Rheumatology, 15*(1), 9–17.

Cohen, S., Frank, E., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M., Jr. (1998). Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychology, 17*(3), 214–223.

Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., et al. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences of the USA, 109*(16), 5995–5999.

Cohen, S., Miller, G. E., & Rabin, B. S. (2001). Psychological stress and antibody response to immunization: A critical review of the human literature. *Psychosomatic Medicine, 63*(1), 7–18.

Cohen, S., Murphy, M. L. M., & Prather, A. A. (2019). Ten surprising facts about stressful life events and disease risk. *Annual Review of Psychology, 70,* 577–597.

Costafreda, S. G., McCann, P., Saker, P., Cole, J. H., Cohen-Woods, S., Farmer, A. E., et al. (2013). Modulation of amygdala response and connectivity in depression by serotonin transporter polymorphism and diagnosis. *Journal of Affective Disorders, 150*(1), 96–103.

Crosby, K. M., & Bains, J. S. (2012). The intricate link between glucocorticoids and endocannabinoids at stress-relevant synapses in the hypothalamus. *Neuroscience, 204,* 31–37.

Cunningham, E. T., Jr., & Sawchenko, P. E. (1988). Anatomical specificity of noradrenergic inputs to the paraventricular and supraoptic nuclei of the rat hypothalamus. *Journal of Comparative Neurology, 274*(1), 60–76.

Dahoun, T., Calcia, M. A., Veronese, M., Bloomfield, P., Reis Marques, T., Turkheimer, F., et al. (2019). The association of psychosocial risk factors for mental health with a brain marker altered by inflammation: A translocator protein (TSPO) PET imaging study. *Brain, Behavior, and Immunity, 80,* 742–750.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*(1), 46–56.

Davalos, D., Grutzendler, J., Yang, G., Kim, J. V., Zuo, Y., Jung, S., et al. (2005). ATP mediates rapid microglial response to local brain injury in vivo. *Nature Neuroscience, 8*(6), 752–758.

Davis, K. D., Taylor, S. J., Crawley, A. P., Wood, M. L., & Mikulis, D. J. (1997). Functional MRI of pain- and attention-related activations in the human cingulate cortex. *Journal of Neurophysiology, 77*(6), 3370–3380.

De Bosscher, K., Vanden Berghe, W., & Haegeman, G. (2003). The interplay between the glucocorticoid receptor and nuclear factor-kappaB or activator protein-1: Molecular mechanisms for gene repression. *Endocrine Reviews, 24*(4), 488–522.

de Gelder, B., Morris, J. S., & Dolan, R. J. (2005). Unconscious fear influences emotional awareness of faces and voices. *Proceedings of the National Academy of Sciences of the USA, 102*(51), 18682–18687.

De Kloet, E. R. (2014). From receptor balance to rational glucocorticoid therapy. *Endocrinology, 155*(8), 2754–2769.

De Kloet, E. R., Vreugdenhil, E., Oitzl, M. S., & Joëls, M. (1998). Brain corticosteroid receptor balance in health and disease. *Endocrine Reviews, 19*(3), 269–301.

De Picker, L. J., Morrens, M., Chance, S. A., & Boche, D. (2017). Microglia and brain plasticity in acute psychosis and schizophrenia illness course: A meta-review. *Frontiers in Psychiatry, 8,* 238.

Del Rey, A., Klusman, I., & Besedovsky, H. O. (1998). Cytokines mediate protective stimulation of glucocorticoid output during autoimmunity: Involvement of IL-1. *American Journal of Physiology, 275*(4), R1146–1151.

Del Rey, A., Verdenhalven, M., Lorwald, A. C., Meyer, C., Hernangomez, M., Randolf, A., et al. (2016). Brain-borne IL-1 adjusts glucoregulation and provides fuel support to astrocytes and neurons in an autocrine/paracrine manner. *Molecular Psychiatry, 21*(9), 1309–1320.

Dolzani, S. D., Baratta, M. V., Amat, J., Agster, K. L., Saddoris, M. P., Watkins, L. R., et al. (2016). Activation of a habenulo-raphe circuit is critical for the behavioral and neurochemical consequences of uncontrollable stress in the male rat. *eNeuro, 3*(5).

Eccles, R. (2005). Understanding the symptoms of the common cold and influenza. *Lancet: Infectious Diseases, 5*(11), 718–725.

Engler, H., Benson, S., Wegner, A., Spreitzer, I., Schedlowski, M., & Elsenbruch, S. (2016). Men and women differ in inflammatory and neuroendocrine responses to endotoxin but not in the severity of sickness symptoms. *Brain, Behavior, and Immunity, 52,* 18–26.

Ericsson, A., Kovacs, K. J., & Sawchenko, P. E. (1994). A functional anatomical analysis of central pathways subserving the effects of interleukin-1 on stress-related neuroendocrine neurons. *Journal of Neuroscience, 14*(2), 897–913.

Estes, M. L., & McAllister, A. K. (2014). Alterations in immune cells and mediators in the brain: It's not always neuroinflammation! *Brain Pathology, 24*(6), 623–630.

Estes, M. L., & McAllister, A. K. (2015). Immune mediators in the brain and peripheral tissues in autism spectrum disorder. *Nature Reviews Neuroscience, 16*(8), 469–486.

Fagundes, C. P., Glaser, R., & Kiecolt-Glaser, J. K. (2013). Stressful early life experiences and immune dysregulation across the lifespan. *Brain, Behavior, and Immunity, 27*(1), 8–12.

Fanselow, M. S., & Dong, H. W. (2010). Are the dorsal and ventral hippocampus functionally distinct structures? *Neuron, 65*(1), 7–19.

Fava, G. A., McEwen, B. S., Guidi, J., Gostoli, S., Offidani, E., & Sonino, N. (2019). Clinical characterization of allostatic overload. *Psychoneuroendocrinology, 108,* 94–101.

Feigenson, K. A., Kusnecov, A. W., & Silverstein, S. M. (2014). Inflammation and the two-hit hypothesis of schizophrenia. *Neuroscience and Biobehavioral Reviews, 38,* 72–93.

Felger, J. C., & Lotrich, F. E. (2013). Inflammatory cytokines in depression: Neurobiological mechanisms and therapeutic implications. *Neuroscience, 246,* 199–229.

Filaire, E., Larue, J., Portier, H., Abed, A., Graziella, P. D., Teixeira, A., et al. (2011). Lecturing to 200 students and its effects on cytokine concentration and salivary markers of adrenal activation. *Stress Health, 27*(2), e25–e35.

Filiano, A. J., Xu, Y., Tustison, N. J., Marsh, R. L., Baker, W., Smirnov, I., et al. (2016). Unexpected role of interferon-gamma in regulating neuronal connectivity and social behaviour. *Nature, 535*(7612), 425–429.

Fine, P., Eames, K., & Heymann, D. L. (2011). "Herd immunity": A rough guide. *Clinical Infectious Diseases, 52*(7), 911–916.

Fleshner, M., Frank, M., & Maier, S. F. (2017). Danger signals and inflammasomes: Stress-evoked sterile inflammation in mood disorders. *Neuropsychopharmacology, 42*(1), 36–45.

Fulling, C., Dinan, T. G., & Cryan, J. F. (2019). Gut microbe to brain signaling: What happens in vagus. *Neuron, 101*(6), 998–1002.

Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: Implications for health. *Nature Reviews Immunology, 5*(3), 243–251.

Glaser, R., Rabin, B., Chesney, M., Cohen, S., & Natelson, B. (1999). Stress-induced immunomodulation: Implications for infectious diseases? *Journal of the American Medical Association, 281*(24), 2268–2270.

Goehler, L. E., Gaykema, R. P., Nguyen, K. T., Lee, J. E., Tilders, F. J., Maier, S. F., et al. (1999). Interleukin-1beta in immune cells of the abdominal vagus nerve: A link between the immune and nervous systems? *Journal of Neuroscience, 19*(7), 2799–2806.

Goshen, I., Kreisel, T., Ounallah-Saad, H., Renbaum, P., Zalzstein, Y., Ben-Hur, T., et al. (2007). A dual role for interleukin-1 in hippocampal-dependent memory processes. *Psychoneuroendocrinology, 32*(8–10), 1106–1115.

Gould, T. J., & Leach, P. T. (2014). Cellular, molecular, and genetic substrates underlying the impact of nicotine on learning. *Neurobiology of Learning and Memory, 107,* 108–132.

Gour, N., & Wills-Karp, M. (2015). IL-4 and IL-13 signaling in allergic airway disease. *Cytokine, 75*(1), 68–78.

Grace, A. A. (2016). Dysregulation of the dopamine system in the pathophysiology of schizophrenia and depression. *Nature Reviews Neuroscience, 17*(8), 524–532.

Gray, J. M., Vecchiarelli, H. A., Morena, M., Lee, T. T., Hermanson, D. J., Kim, A. B., et al. (2015). Corticotropin-releasing hormone drives anandamide hydrolysis in the amygdala to promote anxiety. *Journal of Neuroscience, 35*(9), 3879–3892.

Greenberg, D. B., Jonasch, E., Gadd, M. A., Ryan, B. F., Everett, J. R., Sober, A. J., et al. (2000). Adjuvant therapy of melanoma with interferon-alpha-2b is associated with mania and bipolar syndromes. *Cancer, 89*(2), 356–362.

Greenfeder, S., Umland, S. P., Cuss, F. M., Chapman, R. W., & Egan, R. W. (2001). Th2 cytokines and asthma: The role of interleukin-5 in allergic eosinophilic disease. *Respiratory Research, 2*(2), 71–79.

Hannestad, J., DellaGioia, N., Gallezot, J. D., Lim, K., Nabulsi, N., Esterlis, I., et al. (2013). The neuroinflammation marker translocator protein is not elevated in individuals with mild-to-moderate depression: A [(1)(1)C]PBR28 PET study. *Brain, Behavior, and Immunity, 33,* 131–138.

Hannestad, J., Gallezot, J. D., Schafbauer, T., Lim, K., Kloczynski, T., Morris, E. D., et al. (2012). Endotoxin-induced systemic inflammation activates microglia: [(1)(1)C]PBR28 positron emission tomography in nonhuman primates. *NeuroImage, 63*(1), 232–239.

Hassert, D. L., Miyashita, T., & Williams, C. L.

(2004). The effects of peripheral vagal nerve stimulation at a memory-modulating intensity on norepinephrine output in the basolateral amygdala. *Behavioral Neuroscience, 118*(1), 79–88.

Hayley, S., Lacosta, S., Merali, Z., van Rooijen, N., & Anisman, H. (2001). Central monoamine and plasma corticosterone changes induced by a bacterial endotoxin: Sensitization and cross-sensitization effects. *European Journal of Neuroscience, 13*(6), 1155–1165.

Heinz, A., Braus, D. F., Smolka, M. N., Wrase, J., Puls, I., Hermann, D., et al. (2005). Amygdala-prefrontal coupling depends on a genetic variation of the serotonin transporter. *Nature Neuroscience, 8*(1), 20–21.

Herman, J. P. (2018). Regulation of hypothalamo–pituitary–adrenocortical responses to stressors by the nucleus of the solitary tract/dorsal vagal complex. *Cellular and Molecular Neurobiology, 38*(1), 25–35.

Herman, J. P., & Cullinan, W. E. (1997). Neurocircuitry of stress: Central control of the hypothalamo–pituitary–adrenocortical axis. *Trends in Neuroscience, 20*(2), 78–84.

Hill, M. N., McLaughlin, R. J., Pan, B., Fitzgerald, M. L., Roberts, C. J., Lee, T. T., et al. (2011). Recruitment of prefrontal cortical endocannabinoid signaling by glucocorticoids contributes to termination of the stress response. *Journal of Neuroscience, 31*(29), 10506–10515.

Himmerich, H., Patsalos, O., Lichtblau, N., Ibrahim, M. A. A., & Dalton, B. (2019). Cytokine research in depression: Principles, challenges, and open questions. *Frontiers in Psychiatry, 10,* 30.

Holdsworth, S. R., & Gan, P. Y. (2015). Cytokines: Names and numbers you should care about. *Clinical Journal of American Society of Nephrology, 10*(12), 2243–2254.

Holmes, S. E., Girgenti, M. J., Davis, M. T., Pietrzak, R. H., DellaGioia, N., Nabulsi, N., et al. (2017). Altered metabotropic glutamate receptor 5 markers in PTSD: In vivo and postmortem evidence. *Proceedings of the National Academy of Sciences of the USA, 114*(31), 8390–8395.

Hosoi, T., Okuma, Y., & Nomura, Y. (2002). The mechanisms of immune-to-brain communication in inflammation as a drug target. *Current Drug Targets: Inflammation and Allergy, 1*(3), 257–262.

Huzard, D., Vouros, A., Monari, S., Astori, S., Vasilaki, E., & Sandi, C. (2019). Constitutive differences in glucocorticoid responsiveness are related to divergent spatial information processing abilities. *Stress, 23*(1), 37–49.

Isenberg, S. A., Lehrer, P. M., & Hochron, S. (1992). The effects of suggestion and emotional arousal on pulmonary function in asthma: A review and a hypothesis regarding vagal mediation. *Psychosomatic Medicine, 54*(2), 192–216.

Ishikawa, J., & Ishikawa, A. (2019). The loop neural circuit between the medial prefrontal cortex and the amygdala in the rat brain. *Neuroscience Letters, 712,* 134476.

Isingrini, E., Perret, L., Rainer, Q., Amilhon, B., Guma, E., Tanti, A., et al. (2016). Resilience to chronic stress is mediated by noradrenergic regulation of dopamine neurons. *Nature Neuroscience, 19*(4), 560–563.

Isomura, Y., & Takada, M. (2004). Neural mechanisms of versatile functions in primate anterior cingulate cortex. *Reviews in the Neurosciences, 15*(4), 279–291.

Iwasaki, A., & Medzhitov, R. (2010). Regulation of adaptive immunity by the innate immune system. *Science, 327*(5963), 291–295.

Junttila, I. S. (2018). Tuning the cytokine responses: An update on interleukin (IL)-4 and IL-13 receptor complexes. *Frontiers in Immunology, 9,* 888.

Kaneko, F., Kawahara, Y., Kishikawa, Y., Hanada, Y., Yamada, M., Kakuma, T., et al. (2016). Long-term citalopram treatment alters the stress responses of the cortical dopamine and noradrenaline systems: The role of cortical 5-HT1A receptors. *International Journal of Neuropsychopharmacology, 19*(8).

Kang, D. H., Coe, C. L., McCarthy, D. O., Jarjour, N. N., Kelly, E. A., Rodriguez, R. R., et al. (1997). Cytokine profiles of stimulated blood lymphocytes in asthmatic and healthy adolescents across the school year. *Journal of Interferon and Cytokine Research, 17*(8), 481–487.

Kaptoge, S., Seshasai, S. R., Gao, P., Freitag, D. F., Butterworth, A. S., Borglykke, A., et al. (2014). Inflammatory cytokines and risk of coronary heart disease: New prospective study and updated meta-analysis. *European Heart Journal, 35*(9), 578–589.

Karst, H., Berger, S., Erdmann, G., Schütz, G., & Joëls, M. (2010). Metaplasticity of amygdalar responses to the stress hormone corticosterone. *Proceedings of the National Academy of Sciences of the USA, 107*(32), 14449–14454.

Khandaker, G. M., Cousins, L., Deakin, J., Lennox, B. R., Yolken, R., & Jones, P. B. (2015). Inflammation and immunity in schizophrenia:

Implications for pathophysiology and treatment. *Lancet Psychiatry, 2*(3), 258–270.
Kim, E. J., Pellman, B., & Kim, J. J. (2015). Stress effects on the hippocampus: A critical review. *Learning and Memory, 22*(9), 411–416.
King, S. O., II, & Williams, C. L. (2009). Novelty-induced arousal enhances memory for cued classical fear conditioning: Interactions between peripheral adrenergic and brainstem glutamatergic systems. *Learning and Memory, 16*(10), 625–634.
Kitamura, H., Konno, A., Morimatsu, M., Jung, B. D., Kimura, K., & Saito, M. (1997). Immobilization stress increases hepatic IL-6 expression in mice. *Biochemical and Biophysical Research Communications, 238*(3), 707–711.
Kovács, K. J. (2013). CRH: The link between hormonal-, metabolic- and behavioral responses to stress. *Journal of Chemical Neuroanatomy, 54,* 25–33.
Kumar, V. (2019a). Inflammation research sails through the sea of immunology to reach immunometabolism. *International Immunopharmacology, 73,* 128–145.
Kumar, V. (2019b). Toll-like receptors in the pathogenesis of neuroinflammation. *Journal of Neuroimmunology, 332,* 16–30.
Kuo, T., McQueen, A., Chen, T. C., & Wang, J. C. (2015). Regulation of glucose homeostasis by glucocorticoids. *Advances in Experimental Medicine and Biology, 872,* 99–126.
Kusnecov, A. W., Liang, R., & Shurin, G. (1999). T-lymphocyte activation increases hypothalamic and amygdaloid expression of CRH mRNA and emotional reactivity to novelty. *Journal of Neuroscience, 19*(11), 4533–4543.
Kusnecov, A. W., & Rossi-George, A. (2001). Potentiation of interleukin-1beta adjuvant effects on the humoral immune response to antigen in adrenalectomized mice. *Neuroimmunomodulation, 9*(2), 109–118.
LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience, 7*(1), 54–64.
Lawrence, A. J., Watkins, D., & Jarrott, B. (1995). Visualization of beta-adrenoceptor binding sites on human inferior vagal ganglia and their axonal transport along the rat vagus nerve. *Journal of Hypertension, 13*(6), 631–635.
Lechan, R. M., & Toni, R. (2000). Functional anatomy of the hypothalamus and pituitary. In K. R. Feingold, B. Anawalt, A. Boyce, G. Chrousos, K. Dungan, A. Grossman, et al. (Eds.), *Endotext.* South Dartmouth, MA: MDText.com.
Lehrer, P., Feldman, J., Giardino, N., Song, H. S., & Schmaling, K. (2002). Psychological aspects of asthma. *Journal of Consulting and Clinical Psychology, 70*(3), 691–711.
Leon, J., Hommer, R., Grant, P., Farmer, C., D'Souza, P., Kessler, R., et al. (2018). Longitudinal outcomes of children with pediatric autoimmune neuropsychiatric disorder associated with streptococcal infections (PANDAS). *European Child and Adolescent Psychiatry, 27*(5), 637–643.
Li, X. Z., Bai, L. M., Yang, Y. P., Luo, W. F., Hu, W. D., Chen, J. P., et al. (2009). Effects of IL-6 secreted from astrocytes on the survival of dopaminergic neurons in lipopolysaccharide-induced inflammation. *Neuroscience Research, 65*(3), 252–258.
Lopes Pinheiro, M. A., Kooij, G., Mizee, M. R., Kamermans, A., Enzmann, G., Lyck, R., et al. (2016). Immune cell trafficking across the barriers of the central nervous system in multiple sclerosis and stroke. *Biochimica et Biophysica Acta, 1862*(3), 461–471.
Maes, M., Lambrechts, J., Bosmans, E., Jacobs, J., Suy, E., Vandervorst, C., et al. (1992). Evidence for a systemic immune activation during depression: Results of leukocyte enumeration by flow cytometry in conjunction with monoclonal antibody staining. *Psychological Medicine, 22*(1), 45–53.
Maes, M., Smith, R., & Scharpe, S. (1995). The monocyte-T-lymphocyte hypothesis of major depression. *Psychoneuroendocrinology, 20*(2), 111–116.
Maier, S. F. (2015). Behavioral control blunts reactions to contemporaneous and future adverse events: Medial prefrontal cortex plasticity and a corticostriatal network. *Neurobiology of Stress, 1,* 12–22.
Maier, S. F., & Watkins, L. R. (1998). Cytokines for psychologists: Implications of bidirectional immune-to-brain communication for understanding behavior, mood, and cognition. *Psychological Review, 105*(1), 83–107.
Majdi, A., Kamari, F., Vafaee, M. S., & Sadigh-Eteghad, S. (2017). Revisiting nicotine's role in the ageing brain and cognitive impairment. *Reviews in the Neurosciences, 28*(7), 767–781.
Mallya, A. P., Wang, H. D., Lee, H. N. R., & Deutch, A. Y. (2019). Microglial pruning of synapses in the prefrontal cortex during adolescence. *Cerebral Cortex, 29*(4), 1634–1643.
Marek, R., Strobel, C., Bredy, T. W., & Sah, P. (2013). The amygdala and medial prefrontal cortex: Partners in the fear circuit. *Journal of Physiology, 591*(10), 2381–2391.

Marek, R., Sun, Y., & Sah, P. (2019). Neural circuits for a top-down control of fear and extinction. *Psychopharmacology (Berlin), 236*(1), 313–320.

Marin, T. J., Chen, E., Munch, J. A., & Miller, G. E. (2009). Double-exposure to acute stress and chronic family stress is associated with immune changes in children with asthma. *Psychosomatic Medicine, 71*(4), 378–384.

Marsland, A. L., Walsh, C., Lockwood, K., & John-Henderson, N. A. (2017). The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. *Brain, Behavior, and Immunity, 64,* 208–219.

McCusker, R. H., & Kelley, K. W. (2013). Immune–neural connections: How the immune system's response to infectious agents influences behavior. *Journal of Experimental Biology, 216*(Pt. 1), 84–98.

McEwen, B. S., & Morrison, J. H. (2013). The brain on stress: Vulnerability and plasticity of the prefrontal cortex over the life course. *Neuron, 79*(1), 16–29.

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior, 43*(1), 2–15.

McGaugh, J. L. (2015). Consolidating memories. *Annual Review of Psychology, 66,* 1–24.

McNutt, M. D., Liu, S., Manatunga, A., Royster, E. B., Raison, C. L., Woolwine, B. J., et al. (2012). Neurobehavioral effects of interferon-alpha in patients with hepatitis-C: Symptom dimensions and responsiveness to paroxetine. *Neuropsychopharmacology, 37*(6), 1444–1454.

Medzhitov, R. (2008). Origin and physiological roles of inflammation. *Nature, 454*(7203), 428–435.

Menard, C., Pfau, M. L., Hodes, G. E., Kana, V., Wang, V. X., Bouchard, S., et al. (2017). Social stress induces neurovascular pathology promoting depression. *Nature Neuroscience, 20*(12), 1752–1760.

Miller, G. E., Cohen, S., Pressman, S., Barkin, A., Rabin, B. S., & Treanor, J. J. (2004). Psychological stress and antibody response to influenza vaccination: When is the critical period for stress, and how does it get inside the body? *Psychosomatic Medicine, 66*(2), 215–223.

Miyashita, T., & Williams, C. L. (2006). Epinephrine administration increases neural impulses propagated along the vagus nerve: Role of peripheral beta-adrenergic receptors. *Neurobiology of Learning and Memory, 85*(2), 116–124.

Monastero, R., Bettini, P., Del Zotto, E., Cottini, E., Tincani, A., Balestrieri, G., et al. (2001). Prevalence and pattern of cognitive impairment in systemic lupus erythematosus patients with and without overt neuropsychiatric manifestations. *Journal of the Neurological Sciences, 184*(1), 33–39.

Morales, M., & Margolis, E. B. (2017). Ventral tegmental area: Cellular heterogeneity, connectivity and behaviour. *Nature Reviews Neuroscience, 18*(2), 73–85.

Mulinari, S. (2012). Monoamine theories of depression: historical impact on biomedical research. *Journal of the History of the Neurosciences, 21*(4), 366–392.

Munhoz, C. D., Sorrells, S. F., Caso, J. R., Scavone, C., & Sapolsky, R. M. (2010). Glucocorticoids exacerbate lipopolysaccharide-induced signaling in the frontal cortex and hippocampus in a dose-dependent manner. *Journal of Neuroscience, 30*(41), 13690–13698.

Myers, B., McKlveen, J. M., & Herman, J. P. (2012). Neural regulation of the stress response: The many faces of feedback. *Cellular and Molecular Neurobiology, 32,* 683–694.

Newton, R., Shah, S., Altonsy, M. O., & Gerber, A. N. (2017). Glucocorticoid and cytokine crosstalk: Feedback, feedforward, and co-regulatory interactions determine repression or resistance. *Journal of Biological Chemistry, 292*(17), 7163–7172.

Nikoopour, E., Schwartz, J. A., & Singh, B. (2008). Therapeutic benefits of regulating inflammation in autoimmunity. *Inflammation and Allergy—Drug Targets, 7*(3), 203–210.

Ostergaard, L., Jorgensen, M. B., & Knudsen, G. M. (2018). Low on energy?: An energy supply–demand perspective on stress and depression. *Neuroscience and Biobehavioral Reviews, 94,* 248–270.

Otis, J. M., Namboodiri, V. M., Matan, A. M., Voets, E. S., Mohorn, E. P., Kosyk, O., et al. (2017). Prefrontal cortex output circuits guide reward seeking through divergent cue encoding. *Nature, 543*(7643), 103–107.

Pavlov, V. A., & Tracey, K. J. (2012). The vagus nerve and the inflammatory reflex: Linking immunity and metabolism. *Nature Reviews Endocrinology, 8*(12), 743–754.

Pedersen, A. F., Zachariae, R., & Bovbjerg, D. H. (2009). Psychological stress and antibody response to influenza vaccination: A meta-analysis. *Brain, Behavior, and Immunity, 23*(4), 427–433.

Perlstein, R. S., Whitnall, M. H., Abrams, J. S., Mougey, E. H., & Neta, R. (1993). Synergistic roles of interleukin-6, interleukin-1, and tumor necrosis factor in the adrenocorticotropin response to bacterial lipopolysaccharide in vivo. *Endocrinology, 132*(3), 946–952.

Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S., et al. (2005). 5-HTTLPR polymorphism impacts human cingulate–amygdala interactions: A genetic susceptibility mechanism for depression. *Nature Neuroscience, 8*(6), 828–834.

Pocock, J. M., & Kettenmann, H. (2007). Neurotransmitter receptors on microglia. *Trends in Neurosciences, 30*(10), 527–535.

Pressman, S. D., Cohen, S., Miller, G. E., Barkin, A., Rabin, B. S., & Treanor, J. J. (2005). Loneliness, social network size, and immune response to influenza vaccination in college freshmen. *Health Psychology, 24*(3), 297–306.

Punchard, N. A., Whelan, C. J., & Adcock, I. (2004). The Journal of Inflammation. *Journal of Inflammation (London), 1*(1), 1.

Quatrini, L., Wieduwild, E., Guia, S., Bernat, C., Glaichenhaus, N., Vivier, E., et al. (2017). Host resistance to endotoxic shock requires the neuroendocrine regulation of group 1 innate lymphoid cells. *Journal of Experimental Medicine, 214*(12), 3531–3541.

Rachal Pugh, C., Fleshner, M., Watkins, L. R., Maier, S. F., & Rudy, J. W. (2001). The immune system and memory consolidation: A role for the cytokine IL-1beta. *Neuroscience and Biobehavioral Reviews, 25*(1), 29–41.

Radley, J. J., & Sawchenko, P. E. (2011). A common substrate for prefrontal and hippocampal inhibition of the neuroendocrine stress response. *Journal of Neuroscience, 31*(26), 9683–9695.

Raff, H. (2016). CORT, Cort, B, corticosterone, and now cortistatin: Enough already! *Endocrinology, 157*(9), 3307–3308.

Ramirez, K., Fornaguera-Trias, J., & Sheridan, J. F. (2017). Stress-induced microglia activation and monocyte trafficking to the brain underlie the development of anxiety and depression. *Current Topics in Behavioral Neurosciences, 31,* 155–172.

Rao, S., Schieber, A. M. P., O'Connor, C. P., Leblanc, M., Michel, D., & Ayres, J. S. (2017). Pathogen-mediated inhibition of anorexia promotes host survival and transmission. *Cell, 168*(3), 503–516.

Rieder, R., Wisniewski, P. J., Alderman, B. L., & Campbell, S. C. (2017). Microbes and mental health: A review. *Brain, Behavior, and Immunity, 66,* 9–17.

Romero, L. M., & Sapolsky, R. M. (1996). Patterns of ACTH secretagog secretion in response to psychological stimuli. *Journal of Neuroendocrinology, 8*(4), 243–258.

Rosas-Ballina, M., Olofsson, P. S., Ochani, M., Valdes-Ferrer, S. I., Levine, Y. A., Reardon, C., et al. (2011). Acetylcholine-synthesizing T cells relay neural signals in a vagus nerve circuit. *Science, 334*(6052), 98–101.

Rossi-George, A., Urbach, D., Colas, D., Goldfarb, Y., & Kusnecov, A. W. (2005). Neuronal, endocrine, and anorexic responses to the T-cell superantigen staphylococcal enterotoxin A: Dependence on tumor necrosis factor-alpha. *Journal of Neuroscience, 25*(22), 5314–5322.

Sandiego, C. M., Gallezot, J. D., Pittman, B., Nabulsi, N., Lim, K., Lin, S. F., et al. (2015). Imaging robust microglial activation after lipopolysaccharide administration in humans with PET. *Proceeding of the National Academy of Sciences of the USA, 112*(40), 12468–12473.

Sapolsky, R., Rivier, C., Yamamoto, G., Plotsky, P., & Vale, W. (1987). Interleukin-1 stimulates the secretion of hypothalamic corticotropin-releasing factor. *Science, 238*(4826), 522–524.

Saraiva, M., & O'Garra, A. (2010). The regulation of IL-10 production by immune cells. *Nature Reviews Immunology, 10*(3), 170–181.

Schafer, D. P., Lehrman, E. K., Kautzman, A. G., Koyama, R., Mardinly, A. R., Yamasaki, R., et al. (2012). Microglia sculpt postnatal neural circuits in an activity and complement-dependent manner. *Neuron, 74*(4), 691–705.

Schedlowski, M., Engler, H., & Grigoleit, J. S. (2014). Endotoxin-induced experimental systemic inflammation in humans: A model to disentangle immune-to-brain communication. *Brain, Behavior, and Immunity, 35,* 1–8.

Scheller, J., Chalaris, A., Schmidt-Arras, D., & Rose-John, S. (2011). The pro- and anti-inflammatory properties of the cytokine interleukin-6. *Biochimica et Biophysica Acta, 1813*(5), 878–888.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601–630.

Serrats, J., & Sawchenko, P. E. (2006). CNS activational responses to staphylococcal enterotoxin B: T-lymphocyte-dependent immune challenge effects on stress-related circuitry. *Journal of Comparative Neurology, 495*(2), 236–254.

Shintani, F., Nakaki, T., Kanba, S., Kato, R., & Asai, M. (1995). Role of interleukin-1 in stress responses: A putative neurotransmitter. *Molecular Neurobiology, 10*(1), 47–71.

Sofroniew, M. V. (2014). Multiple roles for astrocytes as effectors of cytokines and inflammatory mediators. *Neuroscientist, 20*(2), 160–172.

Sorrells, S. F., Caso, J. R., Munhoz, C. D., & Sapolsky, R. M. (2009). The stressed CNS: When glucocorticoids aggravate inflammation. *Neuron, 64*(1), 33–39.

Sorrells, S. F., Munhoz, C. D., Manley, N. C., Yen, S., & Sapolsky, R. M. (2014). Glucocorticoids increase excitotoxic injury and inflammation in the hippocampus of adult male rats. *Neuroendocrinology, 100*(2–3), 129–140.

Steinman, L. (2004). Elaborate interactions between the immune and nervous systems. *Nature Immunology, 5*(6), 575–581.

Straub, R. H., & Cutolo, M. (2016). Glucocorticoids and chronic inflammation. *Rheumatology (Oxford), 55*(Suppl. 2), ii6–ii14.

Sugama, S., Takenouchi, T., Fujita, M., Conti, B., & Hashimoto, M. (2009). Differential microglial activation between acute stress and lipopolysaccharide treatment. *Journal of Neuroimmunology, 207*(1–2), 24–31.

Suzuki, S., Tanaka, K., & Suzuki, N. (2009). Ambivalent aspects of interleukin-6 in cerebral ischemia: Inflammatory versus neurotrophic aspects. *Journal of Cerebral Blood Flow and Metabolism, 29*(3), 464–479.

Trettel, F., Di Castro, M. A., & Limatola, C. (2020). Chemokines: Key molecules that orchestrate communication among neurons, microglia and astrocytes to preserve brain function. *Neuroscience, 439,* 230–240.

Tronel, C., Largeau, B., Santiago Ribeiro, M. J., Guilloteau, D., Dupont, A. C., & Arlicot, N. (2017). Molecular targets for PET imaging of activated microglia: The current situation and future expectations. *International Journal of Molecular Sciences, 18*(4), 802.

Turnbull, A. V., & Rivier, C. L. (1999). Regulation of the hypothalamic–pituitary–adrenal axis by cytokines: Actions and mechanisms of action. *Physiological Reviews, 79*(1), 1–71.

Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience, 10*(6), 397–409.

Walker, F. R., Nilsson, M., & Jones, K. (2013). Acute and chronic stress-induced disturbances of microglial plasticity, phenotype and function. *Current Drug Targets, 14*(11), 1262–1276.

Wang, H., Yu, M., Ochani, M., Amella, C. A., Tanovic, M., Susarla, S., et al. (2003). Nicotinic acetylcholine receptor alpha7 subunit is an essential regulator of inflammation. *Nature, 421*(6921), 384–388.

Winkler, Z., Kuti, D., Ferenczi, S., Gulyas, K., Polyak, A., & Kovacs, K. J. (2017). Impaired microglia fractalkine signaling affects stress reaction and coping style in mice. *Behavioral Brain Research, 334,* 119–128.

Wojdasiewicz, P., Poniatowski, L. A., & Szukiewicz, D. (2014). The role of inflammatory and anti-inflammatory cytokines in the pathogenesis of osteoarthritis. *Mediators of Inflammation, 2014,* 561459.

Wolf, S. A., Boddeke, H. W., & Kettenmann, H. (2017). Microglia in physiology and disease. *Annual Review of Physiology, 79,* 619–643.

Woodruff, R. T., Schorpp, K. M., Lawrenczyk, A. J., Chakraborty, T., & Kusnecov, A. W. (2011). Effects of acute and repeated administration of Staphylococcal enterotoxin A on Morris water maze learning, corticosterone and hippocampal IL-1beta and TNFalpha. *Brain, Behavior, and Immunity, 25*(5), 938–946.

Wyss-Coray, T., & Mucke, L. (2002). Inflammation in neurodegenerative disease: A double-edged sword. *Neuron, 35*(3), 419–432.

Yakel, J. L. (2014). Nicotinic ACh receptors in the hippocampal circuit; functional expression and role in synaptic plasticity. *Journal of Physiology, 592*(19), 4147–4153.

Yin, D. M., Chen, Y. J., Sathyamurthy, A., Xiong, W. C., & Mei, L. (2012). Synaptic dysfunction in schizophrenia. *Advances in Experimental Medicine and Biology, 970,* 493–516.

Zhou, D., Kusnecov, A. W., Shurin, M. R., DePaoli, M., & Rabin, B. S. (1993). Exposure to physical and psychological stressors elevates plasma interleukin 6: Relationship to the activation of hypothalamic–pituitary–adrenal axis. *Endocrinology, 133*(6), 2523–2530.

CHAPTER 5

Stress and Stress Management in Children and Adolescents

Jonathan S. Comer
Laura Nelson Darling
Cristina del Busto
Erica D. Musser
Donna B. Pincus

At a global child population level, this is the safest, most peaceful, and most protective era in human history. Although wide disparities persist across and within regions, children born since the turn of the 21st century, relative to any previous generation, enjoy the lowest rate of child and infant mortality, the longest projected lifespan, the widest access to quality health care, and the lowest chance of dying from famine, war, or homicide (Pinker, 2012; Roser, Ortiz-Ospina, Ritchie, Hassell, & Gavrilov, 2019).

Importantly, the relative peace and security of these times has not been reflected in an improved sense of mental well-being among modern youth. For example, prevalence estimates suggest that 13% of youth worldwide currently suffer from at least one mental disorder (Polanczyk, Salum, Sugaya, Caye, & Rohde, 2015), that up to one-third of children experience clinical levels of anxiety even before reaching adulthood (Merikangas et al., 2010), that substance use–related fatalities are increasing among teenagers (Gaither, Shabanova, & Leventhal, 2018), and that suicide remains the second leading cause of mortality in adolescents (Heron, 2018). Indeed, despite historic shifts and massive worldwide improvements in safety and security, emotional stress in youth remains a very serious public health concern.

Accurate identification and effective management of stress in youth is critical. In this chapter, we present a general overview of the nature of stress, as well as leading clinical strategies and techniques for stress management, in children and adolescents. We begin with definitional issues in the study and treatment of youth stress, followed by a brief summary of the biological underpinnings of youth stress, common sources of youth stress, and signs and symptoms of elevated stress in youth.

DEFINING STRESS IN YOUTH

The concept of *stress* in youth has been inconsistently defined and applied across distinct research traditions. Historically, the *epidemiological tradition* has focused on child life experiences that are interpreted by researchers to be objectively stressful, whereas the *psychological tradition* has focused on children's subjective appraisals of life experiences and their perceptions of their ability to cope with them (Cohen, Gianaros, & Manuck, 2016). Moreover, the *biological tradition* has focused on brain-based psychophysiological processes and neuroendocrine systems that underlie youth stress reactivity, the regulation of stress-linked arousal, and coping mobilization. In this chapter, when considering youth stress and its relationships with well-being, we adopt an integrative biopsychosocial conceptualization (Rith-Najarian, McLaughlin, Sheridan, & Nock, 2014) that jointly emphasizes children's subjective appraisals of their environments (regardless of "objective" difficulty or demandingness) and their ability to respond effectively, physiological reactivity processes that underlie stress-linked arousal and regulation, and the interpersonal context in which this all unfolds.

BIOLOGICAL UNDERPINNINGS

Multiple biological systems directly play a role in the stress response, including the autonomic nervous (both parasympathetic and sympathetic branches), central nervous (i.e., the brain), and neuroendocrine systems (e.g., see Gunnar & Adam, 2012; Kreibig, 2010; Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Pagliaccio et al., 2015). The autonomic nervous system (ANS) is a regulatory system that (largely outside of awareness) controls bodily functions related to heart rate, digestion, respiratory rate, pupillary response, and a number of reflexes. Regulated by the hypothalamus, the ANS plays a principal role in youth stress response. The ANS has two main branches—the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is activated in response to stressful stimuli and facilitates psychological arousal—including the "fight or flight" response (or "sympatho-adrenal response") that prepares the body for action in the context of stressful situations. In contrast, the PNS is responsible for the maintenance of homeostasis and aiding in regulation of the stress response and recovery from exposure to stressful stimuli.

Regarding SNS functioning and youth stress, demanding stimuli that evoke *approach*-based responses (e.g., goals, rewards) are associated with physiological reactivity that can be indexed via cardiac pre-ejection period (PEP) captured by impedance cardiography (Richter & Gendolla, 2009; Sherwood, Turner, Light, & Blumenthal, 1990). PEP has been associated with a variety of sympathetically mediated functions, including emotional reactivity, mental effort, reward sensitivity, and stress-linked approach behaviors (Beauchaine, 2001; Kelsey, Ornduff, & Alpert, 2007). Among typically developing youth, shorter PEP in response to stressful stimuli generally indexes greater sympathetic influence over heart rate associated with stress-like approach behavior (Brenner & Beauchaine, 2011; Brenner, Beauchaine, & Sylvers, 2005). In contrast, a second index of SNS functioning, electrodermal activity (EDA), seems linked with *avoidance*-based stress responses via influence of the amygdala (Fowles, 1986; Salminen, Ravaja, Kallinen, & Saari, 2013).

Regarding PNS functioning and youth stress, respiratory sinus arrhythmia (RSA), captured via electrocardiogram, serves as an index of prefrontal-influenced vagal control of heart rate under conditions of stress (Berntson et al., 1997). Among youth, greater resting RSA is associated with effective coping, whereas lower resting RSA is associated with maladaptive coping and a range of mental health problems. Further, in specific contexts, RSA reactivity (i.e., withdrawal from resting levels) is associated with active coping to stressful stimuli, as well as the maintenance of homeostasis (Berntson et al., 1997; Calkins, 2007; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996); accordingly, reduced RSA reactivity (i.e., blunted RSA withdrawal) has consistently been observed among youth with a variety of mental health problems (Graziano & Derefinko, 2013).

Regarding neuroendocrine functioning and youth stress, cortisol is a glucocorticoid that serves as a reliable, valid index of amygdala influence over the hypothalamic–pituitary–adrenal (HPA) axis in response to stressful stimuli (e.g., Gunnar, 2003; Gunnar, Talge, & Herrera, 2009). The HPA includes the hypothalamus, pituitary, and adrenal glands and, among other functions, serves to regulate mood, emotions, and reactions to stress. In response to acute stress, a dynamic cortisol response marked by a rapid rise and decline in cortisol levels is adaptive and facilitates adaptive coping in response to stress. Reduced variation (i.e., "flattening") in morning-to-evening cortisol response to chronic stress has been associated with a range of child and adolescent mental health problems (e.g., Fairchild et al., 2008; Pruessner, Hellhammer, & Kirschbaum, 1999; Van Eck, Berkhof, Nicolson, & Sulon, 1996).

SOURCES OF CHILD AND ADOLESCENT STRESS

Before considering strategies for effectively managing child and adolescent stress, it is important to first consider the range of events and conditions that can trigger youth stress. Typically, such stressors can be organized along three dimensions: (1) External versus Internal, (2) Controllable versus Uncontrollable, and (3) Acute versus Chronic. Differential combinations across the levels of these three dimensions can be used to characterize eight distinct types of stressors (External/Controllable/Acute stressors, Internal/Controllable/Chronic stressors, etc.). Table 5.1 presents examples of each of the eight distinct types of stressors.

External versus Internal Stressors

External stressors in the lives of children and adolescents refer to circumstances and situations in children's environments and other outside factors that can provoke a stress response. Such external sources of stress can include circumstantial hardships and adversities (such as food or housing insecurity, family discord, parental mental illness, discrimination, or bullying), as well as major traumatic events (such as maltreatment and abuse, sexual assault, disaster exposure, and serious accidents). Roughly two-thirds of children and adolescents experience at least one traumatic event prior to adulthood, and over one-third experience multiple such events (Copeland, Keeler, Angold, & Costello, 2007; McLaughlin et al., 2013). Moreover, accumulated childhood adversities that may not individually constitute any direct and immediate physical threat (e.g., economic disadvantage, parental incarceration), external pressures and competing expectations (e.g., academic demands, family expectations, acculturation factors, peer pressures), and everyday hassles (keeping schedules, making deadlines) can similarly trigger considerable stress responses in developing children, especially when presenting in combination with one another (Centers for Disease Control and Prevention & Kaiser Permanente,

TABLE 5.1. Examples of Eight Youth Stressor Types That Vary along Key Dimensions

	Dimension			
Stressor type	Internal versus external	Controllable versus uncontrollable	Acute versus chronic	Examples
1	Internal	Controllable	Acute	• Teen believes if she fails specific upcoming test, she will fail the class. • Teen has fleeting thought that her boyfriend may be cheating.
2	Internal	Controllable	Chronic	• Child has enduring belief he must get a perfect score on every test. • Child believes he cannot tolerate negative emotions.
3	External	Uncontrollable	Acute	• Child is a passenger in a major car accident. • Region in which child lives is hit by a major hurricane.
4	External	Uncontrollable	Chronic	• Week after week, child's family lacks enough money for needed food. • Child suffers ongoing physical abuse in the home.
5	External	Controllable	Acute	• Teen who elects to play baseball has important upcoming game. • Teen's vocal group will be performing in front of a very large audience.
6	External	Controllable	Chronic	• Teen is continuously bullied by a close friend. • Teen carries tremendous responsibility as the team's quarterback.
7	Internal	Uncontrollable	Acute	• Child has a single panic attack out of the blue. • After staying up too late, child is unable to focus in class the next day.
8	Internal	Uncontrollable	Chronic	• Teen's intellectual disability interferes with functioning. • Child's autism spectrum disorder interferes with social success.

2016; Finkelhor, Shattuck, Turner, & Hamby, 2013; Furr, Comer, Villodas, Poznanski, & Gurwitch, 2018).

External factors do not need to be inherently negative experiences to function as stressors in children's lives. Life events and transitions (e.g., birth of a sibling, a new romantic relationship, a new role on a sports team) and normative developmental shifts (e.g., puberty) can similarly trigger stress responses in youth.

Internal stressors in the lives of children and adolescents refer to thoughts and feelings that can provoke a stress response, such as unrealistic personal standards, self-imposed pressures, unwarranted fears, catastrophic thinking, negative beliefs, and maladaptive attitudes. For example, children who exhibit an elevated intolerance of uncertainty (IU; Comer et al., 2009; Cornacchio et al., 2018) find ambiguity distressing and tend to react negatively on emotional, cognitive, and behavioral levels to unexpected events and situations. For such children, their negative beliefs about uncertainty and its consequences can overwhelm their emotional well-being and impair their ability to function, even when confronting mundane uncertainties and routine interactions (e.g., not knowing whom the teacher will call on next, not knowing exactly what a classmate is thinking).

Distress intolerance (DI) serves as another example of a potential internal source of stress. Children who exhibit elevated DI perceive themselves as unable to tolerate experiential discomfort and negative mood states, no matter how minor (Keller et al., 2019). Such perceptions about the intolerability of discomfort, in turn, can trigger significant stress responses in everyday situations and activities in which somatic fluctuations occur (e.g., exercise), frustration or disappointment may be possible (e.g., participating in a competitive game), there may be exposure to elevated temperatures (e.g., an outside event on a sunny day), or moods can shift (e.g., watching a movie with sad elements, listening to up-tempo or stirring music). To avoid inner feelings and experiential discomfort, youth with elevated DI often resort to maladaptive coping strategies, which can range from social withdrawal and emotional suppression to maladaptive eating behaviors, substance abuse, and self-injury.

Controllable versus Uncontrollable Stressors

Uncontrollable stressors refer to those that a child cannot modify. Younger children are unable to adjust neighborhood safety, family financial circumstances, parental relationship discord, physical disabilities, or natural disasters. Similarly, exposure to misguided parenting practices and/or the presence of parental mental illness are not under a child's control. In contrast, *controllable stressors* refer to those youth can modify. Although a child may not initially believe it to be the case, many internal stressors (e.g., unrealistic personal standards, catastrophic fears) are within his or her power to change. Similarly, children and adolescents can exert control over their exposure to many external stressors. For example, although children and adolescents cannot pick their parents or their neighborhoods, they typically do have considerable agency in choosing the friends with whom they associate and the extracurricular activities in which they participate. Accordingly, stressors that arise in children's social circles or extracurricular activities can often be considered modifiable.

Acute versus Chronic Stressors

Stressors in the lives of children and adolescents can also be organized by duration. *Acute stressors* are single-event occurrences or transitory circumstances. Examples might include a major car accident or a single high-stakes standardized test. In contrast, *chronic stressors* refer to enduring circumstances—such as poverty, parental discord, or a physical disability—that can provoke significant and compounding stress responses.

SIGNS AND SYMPTOMS OF STRESS IN YOUTH

Professionals working with children must be cautious not to directly equate exposure to stressors with the experience of stress and must similarly be cautious not to equate the absence of obvious stressors with the absence of subjective stress. Indeed, the presence of stress in youth cannot simply be inferred based on what a child has experienced. Most youth who face adversity or experience major traumatic events show great resilience and endure remarkably well (Copeland et al., 2007; Furr et al., 2018; McLaughlin et al., 2013). On a related note, youth who have not faced significant adversity or experienced a major traumatic event can nonetheless experience extraordinary stress as a consequence of unrealistic personal standards, self-imposed pressures, or catastrophic thinking.

Identifying stress in earlier stages of development can be particularly difficult, given unique obstacles in the assessment of youth internalizing psychopathology (see also Comer & Kendall, 2004). Children rarely refer themselves for treatment, and stress itself can compromise the validity of children's self-reports of their well-being. Younger children are rarely familiar with the concept of stress or with the notion that somatic experiences, emotions, thoughts, and actions are intertwined. Social desirability concerns can further limit children's reports. Moreover, although parents are critical informants in the assessment of child mental health problems, stress is largely an internal phenomenon, with many symptoms manifesting beyond parents' awareness (Comer & Kendall, 2004).

A number of observable signals can indicate that a child is having difficulty coping and may warrant clinical attention, but the specific signs and symptoms of stress differ greatly across individuals. Somatic complaints, such as headaches or stomachaches, are common in children experiencing stress. Many stressed children show behavioral changes and challenges, such as disrupted sleep, fatigue, shifts in eating patterns, excessive clinginess or reassurance seeking, inattention and daydreaming, increased nightmares, bedwetting, irritability, mood swings, and/or conduct problems. It is not uncommon for children and adolescents experiencing stress to increasingly withdraw from social situations or previously enjoyed activities, and they may show significant difficulties relaxing or having fun. Academic performance often declines with stress, as well, although a strong academic record cannot be interpreted as an indication that a child is not experiencing stress.

In the most extreme form of stress—known as *posttraumatic stress*—children can exhibit profound alternations in mood or cognition, such as memory gaps, feelings of dissociation, a sense of foreshortened future, and flashbacks (i.e., reliving major adverse events or feeling as though they are happening all over again; Furr et al., 2018). These posttraumatic stress symptoms are often accompanied by feelings of shame and guilt, hypervigilance, and/or an exaggerated startle response (Furr et al., 2018). Exposure to extreme events and stressors, such as when a child experiences or witnesses actual or threatened death or serious injury, often precede the onset of posttraumatic stress symptoms (Furr, Comer, Edmunds, & Kendall, 2010; Furr et al., 2018). Similarly, threatened death to a loved one can also be associated with posttraumatic stress symptoms (Kritikos, Comer, He, Curren, & Tompson, 2019). There is continued controversy in the field as to whether "direct" exposure to a major traumatic event is essential for formal posttraumatic stress disorder (PTSD) to develop (see Chou, Carpenter, et al., 2017; McNally, 2009), but accumulating evidence does suggest that indirect exposure (such as media-based exposure to a disaster and other mass casualty event) can trigger posttraumatic stress responses among children located even thousands of miles out of harm's way (Comer, Bry, Poznanski, & Golik, 2016; Comer et al., 2014; Comer, DeSerisy, & Green, 2016; Comer & Kendall, 2007; Pfefferbaum et al., 2003).

CLINICAL STRESS MANAGEMENT TECHNIQUES FOR YOUTH

Decades of research on youth risk and resilience processes have clarified how the ability to effectively cope with stress plays a primary and powerful role in fostering positive youth well-being and reducing youth psychopathology (Compas et al., 2017; Weisz, McCabe, & Dennig, 1994). Processes of coping entail the purposeful monitoring, coordination, mobilization, guidance, and modification (as needed) of behavior, emotions, physiology, and/or circumstances when responding to stress (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001; Zimmer-Gembeck & Skinner, 2016).

Compas and colleagues' (Compas et al., 2001; Compas, Jaser, Dunn, & Rodriguez, 2012) control-based model of child and adolescent coping distinguishes *primary control coping* (modifying stressful circumstances or emotions) from *secondary control coping* (adapting or adjusting to stressful circumstances or emotions without attempting to change them) and *disengagement coping* (orienting away from stressful circumstances or emotions, without attempting to change them or adapt to them). Table 5.2 presents examples of primary control coping, secondary control coping, and disengagement coping.

Importantly, although much research has focused on distinct coping "styles" and *between*-child coping differences (e.g., Ayers, Sandler, West, & Roosa, 1996; Abela, Brozina, & Haigh, 2002; Hilt, McLaughlin, & Nolen-Hoeksema, 2010), no specific coping "style" is universally effective across circumstances. Research highlights the importance of *within*-child variations in coping across situations. Different stressors call for different forms of coping, and flexibility in coping approaches across situations is adaptive. For example, when facing stressors that are controllable, primary control coping efforts that focus on modifying the situation may be optimal. A child experiencing stress about an upcoming academic test may do well to cope by developing and carrying through a strategic study plan in order to better learn the material and feel better prepared. In contrast, when facing stressors that are relatively uncontrollable, secondary control coping efforts that focus on adapting to the situation without trying to change it may be optimal (see Weisz et al., 1994). A girl anticipating an upcoming major medical procedure may do well to remind herself how brave she was throughout a past medical procedure or to focus on less obvious positive aspects of the situation, such as the fact that she may miss a week of school (see Weisz et al., 1994).

Accordingly, rather than classifying alternative coping strategies as either categorically "adaptive" or "maladaptive," research has increasingly underscored the importance of youth having access to a range of coping strategies for flexibly confronting the different demands and features of varied circumstances (Aldao, 2013; Aldao, Sheppes, & Gross, 2015; Bonanno & Burton, 2013). Adaptive youth functioning and resilience in the face of stress are constrained by access to only a limited repertoire of coping strategies and/or rigidity in the selection of specific coping strategies across alternative situations

TABLE 5.2. Examples of Primary Control Coping, Secondary Control Coping, and Disengagement Coping

Coping Domain	Definition	Examples
Primary control coping	Efforts to influence or modify stressful events, circumstances, or emotions	• Problem solving • Emotional expression • Emotional modulation
Secondary control coping	Efforts to adapt or adjust to stressful circumstances, or emotions (without attempting to change them)	• Acceptance • Reappraisal
Disengagement coping	Efforts to orient away from stressful circumstances, events, or emotions (without attempting to change them or adapt to them)	• Avoidance • Denial • Wishful thinking

Note. See Compas and colleagues' control-based model of youth coping (2001, 2017) for additional information.

with diverse demands. Indeed, children and adolescents are well served by a broad coping toolkit.

In therapeutic practice with children and adolescents, *stress management* entails the purposeful utilization of targeted clinical techniques to promote effective youth coping with stress. Consistent with the notion that the most effective youth coping strategies will vary across stressful circumstances, practitioners working with youth are best served by a broad portfolio of diverse stress management techniques and strategies that are differentially applicable depending on the nature, source, and symptoms of a presenting patient's stress. In this section, we present leading clinical stress management strategies for helping youth effectively cope with stress. Throughout, we include case illustrations and brief vignettes, and we focus on flexible implementation strategies in order to bring the material to life. Our presentation of clinical stress management strategies for youth is organized around (1) problem-focused strategies, (2) emotion-focused strategies, and (3) lifestyle-focused strategies.

Problem-Focused Stress Management Strategies for Youth

On an almost daily basis, youth are confronted with a variety of controllable, acute, external stressors—from facing conflicts with peers to dealing with a heavy academic workload. Knowing how to generate effective strategies for dealing directly with everyday demands is a critical skill for healthy functioning (Stark, Spirito, Williams, & Guevremont, 1989; Rutter, 1994; Gelhaar et al., 2007; Mayordomo-Rodríguez, Meléndez-Moral, Viguer-Segui, & Sales-Galan, 2015). In fact, some studies have indicated that children's ability to cope with everyday problems is a better predictor of concurrent and subsequent psychological symptoms than the ability to cope with major life events (DiCorcia, Sravish, & Tronick, 2013).

Problem-focused stress management strategies refer to efforts to directly change or master the source of the youth's stress by either altering the environment, changing external pressures, or finding resources so that the distressing situation is made less threatening (Lazarus & Folkman, 1984). These strategies are particularly effective when situations are controllable and when stress-related environmental challenge can be altered.

Teaching skills to actively generate effective solutions to stressful, but controllable, situations is linked with positive youth adjustment (Compas et al. 2017). Broadly speaking, "problem-focused" or "problem-solving" interventions focus on teaching youth to directly alter the objective circumstances of a stressful situation by: (1) identifying or defining the stressful problem and one's ultimate goal, (2) generating multiple solutions for a given problem, (3) evaluating the various potential consequences of each solution, and (4) choosing the solution or solutions that are most likely to result in the most positive outcomes and help achieve one's goal.

Many current problem-solving training programs are based on concepts from Spivack and Shure's seminal work (1974, 1982) on the assessment and training of interpersonal cognitive problem-solving skills in children. These programs have been linked with significant improvements in children's generation of multiple solutions, "means–end thinking" (i.e., ability to organize a sequence of steps to achieve an outcome), knowledge and performance of problem-solving skills, and overall behavioral adjustment (e.g., Cowen, Wyman, Work, & Parker, 1990; Weissberg & Elias, 1993; Boyle & Hassett-Walker, 2008).

Dubow and colleagues' (Dubow, 1993; Dubow, Schmidt, McBride, Edwards, & Merk, 1993) problem-solving intervention uses the mnemonic *I CAN DO* to remind children and adolescents of five key problem-solving steps when encountering a stressful situation: (1) **I**dentify the problem, (2) think of **C**hoices, (3) pay **A**ttention to each choice, (4) **N**arrow the choices to one choice, (5) **DO** the choice and praise yourself. Numerous studies have successfully incorporated the I CAN DO steps (along with other complementary skills) into brief problem-solving skills trainings in both school and clinical settings (Dubow, 1993; Dubow et al., 1993; Spence, Sheffield, & Donovan, 2003; Pincus & Friedman, 2004). Teaching children and adolescents concrete problem solving skills with discrete steps can help them think flexibly when encountering a stressful situation. Furthermore, the I CAN DO problem-solving approach can be taught relatively quickly in an individual or group therapy session or in a classroom setting, and has been found to improve youth's ability to generate multiple solutions to everyday stressors (Pincus & Friedman, 2004).

Consider the case of 12-year-old "Marisa," who had been avoiding going to the cafeteria during lunch at school, was spending her lunch period loitering in the school restroom, and, as a result, was repeatedly getting detention. Following is an excerpt from an individual therapy session with Marisa, during which the therapist used the I CAN DO problem-solving steps with Marisa to help her clarify the exact problem, generate alternative strategies for dealing with it, and evaluate the likely effectiveness of her solutions:

THERAPIST: Let's talk about what it is exactly about lunch that is causing you to avoid it by staying in the bathroom?

MARISA: I guess I'm afraid that I won't get a seat with my friends. The tables in the lunchroom are very crowded.

THERAPIST: I understand—so it sounds like lunchtime can be very stressful if you don't find a seat at the table you want. Has the strategy of staying in the bathroom worked for you?

MARISA: No, it isn't working because I keep getting detention and I'd much rather be eating lunch with my friends.

THERAPIST: Let's try to think together of all the things you might do to make this situation work better for you. Sometimes it's helpful to solve a problem or stressful situation like this using steps. The first step is figuring out the problem, and I think you already did that!

MARISA: Yes, the problem is I really want to sit at lunch with my friends but I'm afraid I won't get a seat at their table.

THERAPIST: The next step is figuring out all of your options and then choosing the best one. Rather than just jumping to do the first thing that comes to your mind, you want to think of all of your choices. It's kind of like if you were shopping for something you really wanted—you might want to first look at all the options, and then decide which was best before making a decision. Now let's consider that there could be many ways to solve this problem—hiding in the bathroom was only one option. What are ALL the things you might do to make things better in this situation?

MARISA: Well, I could talk with a friend ahead of time to save me a seat. But I really can't be sure that my friend will be able to save me a seat, since the kids in the lunchroom can be very pushy.

THERAPIST: You did a great job of thinking of another way to problem solve this—how many other ideas can you come up with? At this point it's OK to just list all the options you can think of. Then, together, we can talk about which one might work best for you to meet your goal of sitting with your friends at lunch.

MARISA: Well, I guess I could talk with a teacher about this. . . . I could also bring my lunch so I'm not waiting in a long lunch line—that would get me to the table faster. I could also try to get to lunch more quickly by taking the side staircase next to my math class. . . . I could share a seat with a friend at the lunch table if worse came to worse. I could always think of a "plan B" if I don't get a seat—maybe a friend and I could make a pact that if one of us doesn't get a seat we'll both move together to a different table. Oh! I could also volunteer to help a teacher during lunch if I don't get a seat.

THERAPIST: Wow—you just thought of so many other ways you could change this situation! I just wrote them all down. It can be really helpful to consider whether any of these options will work better than others. It's also possible that several of these ideas could work and that you may not need to choose just one!

MARISA [*considering her options*]: Well, I think it might not meet my goal of hanging out with my friends at lunch if I just volunteer to help a teacher during lunch. I might still feel left out and sad if I choose that one. Maybe I'll do a bunch of these—I'll bring my lunch, use the side staircase, and ask my friend to save me a seat, and have a "plan B" in case my first plan doesn't work out.

THERAPIST: I can't wait to hear how this plan works out. The ideas you came up with have a good chance of helping you meet your goal!

In addition to the I CAN DO solution-generation approach, other coping skills-training programs work to improve youth's problem-focused coping by teaching them organization skills, time management, and planning skills. These programs help youth set goals, think flexibly, organize and prioritize tasks, access working memory, and learn how to self-monitor and check their work and progress (e.g., SMARTS curriculum; Meltzer, 2018). Consider the case of 17-year-old "Nathan," who had attention difficulties and felt tremendous stress about whether he would pass his summer calculus class. Nathan had already failed calculus, and if he did not pass this summer class, he could not begin his major in meteorology in college. Upon assessment, it was clear that Nathan had not developed effective study habits. He was studying only late in the evening when he was already tired and would cram for exams. Using components of various programs that emphasize helping youth with planning, organization, problem solving, and study skills, Nathan was taught to pace his studying during the day. He also created a list of ways he could reward himself after he focused on his work for specific time intervals. Nathan was taught to use reminder notes, which he posted on the bulletin board in front of his desk, to refocus him on his goals for the summer if he began to daydream. Nathan also learned to use a variety of study skills to help him prepare more efficiently for tests. Further, he was also coached to regulate his sleep schedule so that he was going to bed at a consistent time each night and was more alert during the day. At the end of the summer, Nathan passed his class, and he noted to his therapist that he believed he was successful because he "actively changed many behaviors" and learned how to "take control of the situation by making a strategic study plan and developing better habits." Nathan also later reported that these problem-solving strategies he learned generalized to other classes.

Emotion-Focused Stress Management Strategies for Youth

Whereas problem-focused stress management strategies work to change the environments, circumstances, or situational contexts of youth stress, *emotion-focused* stress management strategies target experiential processes that underlie stress—including key physiological, somatic, and cognitive components.

Breathing Retraining

The diaphragm is an important muscle situated between the abdomen and chest that plays a critical role in respiration. During inhalation, the diaphragm flattens, creating a vacuum that draws air into the lungs. During exhalation, the diaphragm relaxes, helping air escape from the lungs.

With the diaphragm functioning as the primary muscle of respiration, *breathing retraining* focuses on strengthening a child's diaphragm, which in turn can improve the availability of oxygen and carbon dioxide in the blood and reduce physiological symptoms associated with stress. Specifically, stress-related "overbreathing" (or *hyperventilation*) is characterized by short and rapid breaths at about three times the normal rate, which in turn have the effect of exhaling more carbon dioxide than the body can actually produce. The result is a reduced concentration of carbon dioxide in the bloodstream, which produces symptoms of dizziness, weakness, tingling sensations, headache, and sometimes fainting. In breathing retraining, the therapist works to (1) bring breathing into the child's awareness and under the child's voluntary control and (2) slow down and deepen the child's breath so as to better moderate the amount of oxygen inhaled and carbon dioxide exhaled (see Pincus, Madigan, Kerns, Hardway, & Comer, 2014).

Before beginning to teach a child to engage in deeper "diaphragmatic breathing," it can be helpful to engage the child in an *overbreathing exercise* in order to highlight for the child the physical sensations that accompany stress-related breathing and lowered carbon dioxide levels (Pincus et al., 2014). In developmentally sensitive terms, the therapist would first explain that breathing too quickly reduces the carbon dioxide in the blood, which in turn can cause a number of uncomfortable feelings in the body. The therapist would then join the child in intentionally taking quick, short breaths together, exhaling very hard and very rapidly. After a round of short, quick breaths, the therapist and child would share with one another all of the different physical sensations they were having, and the therapist would emphasize how these physical sensations are many of the very same physical sensations people feel when they are stressed, anxious, worried, or feeling otherwise panicky.

Importantly, if the child or adolescent has panic disorder, a history of panic disorder in the family, high distress intolerance, or elevated "anxiety sensitivity" (i.e., fear of bodily symptoms of anxiety), it is not uncommon for an overbreathing exercise to actually trigger a panic attack. Accordingly, prior to engaging such a child in an overbreathing exercise, the therapist should prepare the child (and his or her parents) for the possibility that the exercise could trigger a panic attack. Alternatively, when working with a child with such a panic symptom presentation or history, the therapist might elect to omit overbreathing exercises and move directly to training the child in deeper diaphragmatic breathing.

Before retraining the child in how to take deeper, slower breaths, it is important to present a clear rationale for learning this skill. A therapist might introduce a diaphragmatic breathing exercise to the child in the following manner:

"Our bodies breathe automatically, and we usually don't pay much attention to it. It's sort of happening in the background so we can focus on other things. But when we want, we can actually take over voluntary control of our breathing—like when we're holding our breath, or when we're swimming. Stress can speed up our breathing, and we learned how breathing too quickly will cause all sorts of uncomfortable feelings in our bodies. I'm going to teach you how to become more aware of your breathing, and how you can change your breathing patterns.

"All right—first, I want you to watch my body while I breathe way too quickly. [*The therapist should model rapid overbreathing, accompanied by quick and exaggerated chest and shoulder movements with each breath. Exaggerating the movements helps make clear to the child what overbreathing looks like. It can also look silly, which can make the exercise a bit more fun and help engage the child.*] You can see I'm breathing way too quickly. What else did you notice about my body when I was breathing too quickly? [*Hopefully the child was able to observe the therapist's chest moving rapidly.*] That's right! My chest was moving rapidly with each breath. Well, when we're stressed, we breathe too quickly, and we tend to use the muscles in our chest to breathe instead of the muscles in our belly. This makes our chest feel tight, makes our whole body feel tight, and the extra annoying thing is it makes us feel even more stressed. So that's why it's so important to learn how to bring our breathing under our voluntary control, to try to breathe less from our chests, and to slow our breathing down."

After ensuring that the child understands the rationale for retraining his or her breathing, the next steps can be broken down into three pieces: (1) teaching the child to recruit different muscles to breathe (i.e., switching from chest breathing to abdominal breathing), (2) teaching the child to change the rate of his or her breathing (i.e., slowing down his or her breathing so as to exhale less carbon dioxide per minute), and (3) having the child practice this new breathing approach in, and outside of, session.

When teaching the child to recruit abdominal muscles (instead of chest muscles) as he or she breathes, the therapist should first model appropriate diaphragmatic breathing for the child. The therapist can do this by placing one hand on his or her stomach (just above the navel) and the other hand on his or her chest. As the therapist breathes, it should be clear to the child that the hand on the stomach moves with each breath while the hand on the chest stays relatively still. The therapist might engage the child by saying something like:

"What are you noticing about my hands while I breathe? [*If the child does not respond, or if the child comments on something unrelated to hand movements, the therapist can follow up with 'Are either of them moving? Are they both moving or only one of them?'*] That's right! The hand on my belly moves with each breath, but the hand on my chest is staying pretty still. That's because I'm now using the muscles in my belly to breathe, and I'm not using the muscles in my chest to breathe.

"OK, now I want you to try—I want you to place one hand above your belly button, like I'm doing here, and I want you to place your other hand on your chest, like I'm doing with my other hand. With each breath, I want to you to try to make your belly hand move out, but not your chest hand. That's how you know you're using the right muscles as you breathe—the belly hand moves, but the chest hand stays pretty still."

After the child successfully learns how to voluntarily breathe at his or her normal breathing pace from the abdomen instead of the chest, the therapist moves to focus on slowing the rate of this diaphragmatic breathing. If the child takes music lessons, a metronome metaphor can work very well (e.g., "OK, now we're going to work on doing the same thing, but now slowing down the metronome a bit"). The therapist should emphasize the importance of big, deep, slow breaths. The therapist might use a fun name for these types of breaths, like "pizza breaths" or "birthday cake breaths":

> "OK, whenever you breathe in, I want you to breathe in through your nose, super slow, like you are smelling a delicious pizza on the table in front of you, for 3 seconds. [*The therapist needs to make sure that the child is continuously inhaling for 3 seconds, and not quickly inhaling for just the 1st second and holding his or her breath for 2 additional seconds.*] As you breathe in, I want you to count to 3, taking the delicious smell of the pizza in through your nose throughout the entire 3 seconds . . . 1 . . . 2 . . . 3. . . . [*The therapist models.*] OK, now whenever you breathe out, I want you to breathe out slowly, through your mouth, like you are blowing out birthday candles in front of you, for 3 seconds. [*The therapist needs to make sure that the child is continuously exhaling for 3 seconds, and not just quickly exhaling on the 1st second and holding his or her breath for 2 seconds.*] As you breathe out, I want you to count to 3, making sure to continue to blow out those candles throughout the entire 3 seconds . . . 1 . . . 2 . . . 3 . . . [*The therapist models.*]"

When the child switches from focusing on abdominal breathing to slowing his or her breathing rate, it is not uncommon for him or her to have some initial difficulty bringing the two skills together. The goal is for the child to accomplish both at the same time (i.e., slow breaths, from his or her abdomen), but this takes practice. For the frustrated child, the therapist will do well to comment jokingly on how long he or she also took to learn this type of breathing as an adult, and how much more quickly the child seems to be already mastering it. Once the child is able to both breathe from the abdomen and slow his or her breathing, the goal is to continue to slow the rate of this diaphragmatic breathing down to roughly eight breaths per minute (i.e., 4 seconds for each inhalation and 4 seconds for each exhalation; Pincus et al., 2014).

Simply learning *how* to engage in slower, diaphragmatic breathing is not sufficient—the child must engage in daily practice in order to be able to voluntarily recruit these skills to actively reduce tension during stressful moments and in order to actually shift automatic breathing processes so as to reduce the child's general stress level. The therapist should have the child practice slow, diaphragmatic breathing out of session for at least 5 minutes each day. Initially, the child should be completing his or her out-of-session breathing exercises in a calm, comfortable, and relaxing atmosphere (e.g., perhaps in his or her room with all distractions turned off). As the child masters the breathing exercises in relaxing settings, he or she should be encouraged to practice them in increasingly challenging and distracting environments, such as in common spaces in the home, while the television is on, or while at school.

As with all therapeutic homework in child psychotherapy, it is not uncommon for a child's compliance with out-of-session breathing practices to be poor initially. Parents should be recruited to remind the child to complete daily breathing exercises, and parents often need to sit and do the breathing exercises with the child to ensure that they are being completed properly. This can even be turned into a fun family activity. A sticker chart or reinforcement system can further improve child compliance with out-of-session breathing exercises.

Progressive Muscle Relaxation

Given that stress is associated with an array of somatic symptoms and increased muscle tension throughout the body, it can be productive to bring stress-related bodily feelings into the child's awareness and to provide tools for bringing muscle tension under the child's control. *Progressive muscle relaxation* (PMR) refers to a series of exercises involving the systematic tensing and relaxing of major muscle groups throughout the child's body in order to reduce overall body tension and bring about a state of bodily relaxation (Pincus et al., 2014). Many children experiencing stress cannot even recognize what a relaxed body feels like. PMR gives such children the skills to distinguish the feelings of a stressed body from a relaxed body and to release muscle tension, as needed, in order to better regulate the somatic components of stress.

In clinical practice, PMR is one of the most widely employed youth stress management strategies, given its straightforward techniques and its positive effects on a range of child and adolescent problems. For example, PMR in youth has been found to reduce insomnia, acute and chronic pain, anger and aggression, depressive and related internalizing symptoms, and chronic medical conditions such as asthma (Higa-McMillan, Francis, Rith-Najarian, & Chorpita, 2016; Nickel, Lahmann, et al., 2005; Nickel, Kettler, et al., 2005; Pincus et al., 2014). In broader terms, PMR can help children and adolescents feel calmer and less nervous, can foster better concentration and focus, can increase enjoyment of pleasurable activities, and can promote an improved sense of peace and tranquility (Rapee, Wignall, Hudson, & Schniering, 2000).

That said, PMR by itself is not sufficient in the treatment of formal pediatric anxiety disorders. When treating these diagnoses, the inclusion of exposure-based techniques that have children systematically confront feared situations and stimuli is essential for adequate treatment response (Comer, Hong, Poznanski, Silva, & Wilson, 2019; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008). In fact, randomized trials evaluating treatment methods for youth anxiety and obsessive–compulsive disorders have increasingly employed muscle relaxation treatments as control/comparison conditions when evaluating exposure-based treatment methods (e.g., Freeman et al., 2014). Moreover, some modern evidence-based treatments for youth anxiety and related disorders do not even include relaxation strategies in their protocols (e.g., Ehrenreich-May et al., 2017).

Despite the relatively limited success of PMR (by itself) for achieving adequate treatment response in the management of youth anxiety disorders, bodily tension is a key component of youth stress, and, in the absence of a formal anxiety disorder diagnosis, PMR can be powerfully effective in managing and reducing somatic symptoms of stress. In the treatment of youth anxiety disorders, PMR *combined* with exposure-based treatment techniques can produce very positive treatment gains (Cornacchio, Sanchez, Chou, & Comer, 2017; Rapee et al., 2000). Accordingly, PMR remains an essential, transdiagnostic stress management technique when treating a range of stress-related problems in youth.

The first step of PMR is to increase the child's awareness of physical signs of stress in the body. The therapist might consider having the child lie down on a stretch of butcher paper and trace around his or her body. Within the drawn outline of the child's traced body, the therapist and the child can then use markers and stickers to depict the bodily symptoms of stress. It is helpful to be as vivid and silly as possible during such a "draw-a-body" activity. For example, when drawing a "pounding heart," it can be fun to draw a hammer where the chest would be located within the body outline. For an upset stomach, several butterflies can be drawn in the body outline's stomach region. When the child is unable to generate stress symptoms (or is otherwise quiet), the therapist can chime in with his or her own suggestions (e.g., "I know whenever I'm stressed, I start sweating a lot, and my knees feel weak and shaky. That happens to one of my friends, too. How about we draw a faucet, or some raindrops, in the armpits, and some wavy lines near the knees, to show sweating weak knees?"). Alternatively, the therapist can invite a parent to talk about what the parent feels in his or her body when stressed, and these symptoms can be incorporated into the drawing.

To further increase awareness of physical signs of stress in the body and to help the child distinguish the feelings of a stressed body from a relaxed body, the therapist might play a "robot versus rag doll" game. The therapist asks the child, "How might a robot walk around the room here?" Typically, the child can demonstrate very stiff and tight movements around the room, but if not, the therapist can demonstrate by moving around the room with exaggerated, nonfluid stiffness. While moving around like a pretend robot, the therapist might also vocalize (in his or her best robotic voice, with awkward and flat prosody) "I . . . am a . . . robot. My body . . . is so . . . stiff and . . . tight. . . . Nothing in . . . my body feels . . . smooth. . . . This is so . . . uncomfortable." The therapist leads the child in marching around the room as robots in a rigid manner for a bit longer, talking to each other in stiff robot voices. After some time passes, the therapist shifts to ask the child how a rag doll might instead move around the room. Typically, the child can demonstrate very flexible and fluid movements around the room, but if not, the therapist can demonstrate by moving around the room with exaggerated, fluid flexibility. While moving the way a relaxed rag doll might, the therapist can also vocalize in a laid-back voice with smooth prosody, "I'm a rag doll and my body is sooo relaxed and fluid. It's sooo easy to smoothly move any part of my body." The therapist and the child glide around the room as rag dolls in a free-flowing manner for a bit longer, talking to each other in voices that exaggerate how laid back they are. Once the child understands both modes ("robot" mode and "rag doll" mode), the therapist and child continue moving around the room, but must quickly transition to "robot" mode every time the therapist (or parent) yells out "robot," and must quickly transition to "rag doll" mode every time the therapist (or parent) yells out "rag doll."

Variations of the robot-versus-ragdoll game include "cooked versus uncooked spaghetti," "statue versus Jell-o," "tin man versus jellyfish," and "soldier standing at attention versus at ease." The important component of this approach is to facilitate a conversation with the child contrasting how it feels in his or her body when he or she is in the mode of stiff robot (or uncooked spaghetti, statue, tin man, soldier at attention, etc.) versus how it feels in his or her body when he or she is in the mode of relaxed rag doll (or cooked spaghetti, Jell-o, jellyfish, or soldier at ease). The child should be able to recognize that it is more comfortable in his or her body to move like a rag doll (or cooked spaghetti, Jell-o, jellyfish, or soldier at ease).

The therapist transitions to letting the child know that the therapist will teach the child skills to actually control how stiff his or her body is and how much tension he or she feels. The therapist then collects a "stress rating" from the child as a baseline indication of his or her pre-PMR body stress. This can be a 0–10 scale, or, for the younger child, breaking things down into three imagery-based levels (e.g., green/yellow/red, or jellyfish/palm tree/statue) can be helpful.

The therapist then instructs the child to get in a comfortable position and take a series of big, deep breaths, similar to the slow diaphragmatic breaths presented in the "breathing retraining" section above. To maximize comfort and minimize the impact of distractions in the office, it can be helpful to have the child close his or her eyes and think of a place he or she finds relaxing (e.g., on the beach). The therapist can reinforce this imagery by asking the child to think of the smells and sounds of this relaxing place (e.g., "Breathe in the smell of that sea air," "listen and you can hear the sound of the waves gently crashing") while he or she is taking deep, slow breaths.

The first muscle group to target is the hands, as most children are already relatively familiar with the idea of squeezing the hand into a fist. Focusing on one hand at a time, the therapist guides the child to make a fist and to squeeze that fist tightly for a count of 3. Guided imagery works well for younger children (e.g., "I want you to imagine you have a lemon in your right hand. Now squeeze that lemon as hard as you can in your hand, squeezing all of the juice out"). After a count of 3, the therapist has the child release the fist and relax the hand (e.g., "OK, now I want you to drop the lemon and relax your hand"). The therapist guides the child to alternate between a tight fist for 3 seconds and a loose hand for at least 15 seconds, for a total of two more times. Each time that the child is tensing the hand muscles to make a fist, the therapist calls attention to how tense and uncomfortable the child's hand is (e.g., "Feel all of that tension in your hand"), and each time that the child is releasing the tension, the therapist calls attention to how relaxed the child's hand is (e.g., "Feel all of that tightness flowing out of your hand. Feel how loose and warm your hand is, and how much better that feels").

The therapist systematically guides the child in tensing and relaxing each of the muscle groups throughout his or her body, focusing on one muscle group at a time, for three repetitions each. As with the hand-muscle-tensing exercise, guided imagery can be used to engage the child in tensing each of the other key muscle groups (Ollendick & Cerny, 1981). Turtle imagery can be used to target neck muscles (e.g., "Pretend you're a turtle hiding in your shell—bring your shoulders all the way to your ears and push your head down into your body"). Imagery of a fly on the child's nose can be used to target facial muscles (e.g., "Without using your hands, try to get rid of that annoying fly—wrinkle and scrunch up your nose super hard"). Imagery about walking barefoot through thick mud can be used to target leg and foot muscles (e.g., "Push your foot down deep into the thick squishy mud, and spread your toes apart to feel the mud squish up between your toes"). Imagery about a baby elephant stepping on the child's belly can be used to target stomach muscles (e.g., "Imagine you're lying in the grass, and a cute baby elephant is walking around but not looking where he is going. He's about to step on your belly and you don't have time to get out of the way. You just need to get ready for him—tighten up your belly muscles and make your stomach super hard").

To avoid pain or discomfort resulting from PMR, a given muscle group should not be tensed for longer than 3 seconds at a time and for no more than three times before moving on to target another muscle group. The therapist must also make sure that at least 15 seconds have passed before tensing the same muscle group again. Between sets of muscle tension, the therapist returns the child back to focus on deep, slow, diaphragmatic breaths. If a child is reporting pain from PMR exercises, it is usually an indication that a muscle group is being tensed too often or for too long. On a related note, children and adolescents with various injuries or who are recovering from a medical procedure should not work on affected muscle groups until they have fully healed.

At the conclusion of a PMR session, the therapist should solicit another "stress rating" from the child, along the same scale used at the beginning of the session. If done correctly, and if the child is able to sense somatic variations in the body, the child's rating should likely decline from pre- to post-PMR. If the child's stress rating does indeed decline from the beginning to the end of the session, it is important to call this to the child's explicit attention to help him or her appreciate how the bodily tension he or she experiences can be brought under his or her control. And if the child's stress rating does not decline from the beginning to the end of the session, this is helpful information for troubleshooting on the part of the therapist and to inform him or her whether any adjustments are needed.

As with breathing retraining, simply learning *how* to release muscle tension is not sufficient—the child must engage in daily practice in order to be able to actively reduce muscle tension during stressful moments and in order to actually shift the general level of tension in his or her body across time. The therapist should have the child practice PMR out of session for at least 10–15 minutes each day. The therapist can make an audio recording for the child to follow when completing PMR activities at home. Alternatively, there are a number of commercially available audio recordings that guide children through PMR using guided imagery (e.g., Pincus & Otis, 2001).

Importantly, the child does not need to feel stressed at the time he or she practices. It should simply become a daily routine, like brushing teeth. Recruiting parents to remind the child to complete daily PMR exercises can improve compliance with out-of-session PMR assignments. Similarly, a sticker chart or reinforcement system can further improve child compliance. Moreover, as with daily breathing exercises, the family can make PMR a daily family activity, which can have added benefits of reducing any problematic bodily tension among other members of the family and increasing shared family activity time.

Cognitive Reappraisal Strategies

Whereas breathing retraining and PMR target *somatic* components linked to experiential aspects of youth stress, cognitive reappraisal strategies target *thought* processes that underlie experiential aspects of youth stress. Thought patterns of children and adolescents experiencing elevated stress are often characterized by cognitive "biases" or "distortions," in which ambiguous situations are processed, interpreted, and/or recalled in an overly negative manner (Dearing & Gotlib, 2009; Taghavi, Moradi, Neshat-Doost, Yule, & Dalgleish, 2000). Such distorted, biased, or overly negative thought patterns have cascading effects on mood and behavior, which in turn trigger or perpetuate maladaptive cycles of stress and negativity.

Cognitive reappraisal strategies work to disrupt negative thought patterns by helping children and adolescents shift maladaptive interpretations of neutral or stress- and anxiety-provoking situations to be more balanced, realistic, or helpful. This clinical technique is commonly referred to as "cognitive restructuring," in which children and adolescents learn to identify and adjust maladaptive thoughts and distortions that negatively affect quality of life (see Cornacchio et al., 2017). With practice, cognitive restructuring enhances children's awareness of their own thought patterns and fosters their ability to think more flexibly in stressful situations.

When engaging a child in cognitive reappraisal techniques, it is important to help the child understand how his or her thoughts can affect his or her mood and behaviors. To demonstrate this concept, the therapist might begin with a developmentally appropriate example of a situation that the child enjoys (or feels neutral toward). In this initial stage, the goal is for the child to understand how thoughts, feelings, and behaviors are connected, without yet applying this understanding to situations or circumstances the child personally finds stressful. To introduce this concept to the child, the therapist could use the example of a child who has never tasted ice cream before:

> "Today, we're going to talk about how the thoughts in our heads can actually affect our feelings and behavior. Here's a picture of a kid in an ice cream shop who has never tasted ice cream before. [*The therapist shows drawing or cartoon of a child in an ice cream shop; child's face should be somewhat ambiguous, neither smiling nor frowning.*] Now imagine this kid is thinking to him-/herself, 'I think this ice cream will taste disgusting. It might even make me sick to my stomach!' How do you think the child would feel about trying ice cream for the first time? Do you think the child would want to try the ice cream? [*The therapist and child might even do role-play exercises, with the child pretending to work at an ice cream counter and the therapist pretending to be a boy who has never eaten ice cream before and thinks it will be disgusting. When the child asks the therapist (playing the boy) if he would like any ice cream, the therapist can act out how much he doesn't want ice cream—the more exaggerated and silly, the better.*]
>
> "OK, now let's imagine the kid had different thoughts in his head when going into the ice cream shop. Maybe he was thinking, 'Lots of people love ice cream. I bet this ice cream will taste delicious. It might become my new favorite dessert!' Now how do you think the boy would feel about trying the ice cream now? Do you think he would be more likely or less likely to try the ice cream? [*Hopefully, the child is able to anticipate that the boy would be more likely to try the ice cream.*] That's right! [*The therapist and child might again role-play the boy in the ice cream store, but this time when the child (playing the ice cream store worker) asks if the therapist (playing the boy) if he would like any ice cream, the therapist can act out how much he does want ice cream—again, the more exaggerated and silly, the better.*]
>
> "So, when the boy was thinking the ice cream is going to be disgusting, he felt nervous and did not want to try it. But when the boy was thinking the ice cream might be delicious, he felt excited and wanted to try it. See how the way the child thought about the situation changed how he felt, and what he might do?"

If the child has difficulty understanding the concept of thoughts or cognitive activity, it can be useful to use cartoon thought bubbles to illustrate. If the child has a favorite character, superhero, or athlete, the therapist might print out a series of images from the Internet of that individual in various scenarios and could, additionally, print and cut out a large, blank "thought bubble." The therapist can place this blank thought bubble on top of each of the printed images of the child's favorite character or hero in different situations and, for each one, ask the child, "What do you think is in her thought bubble now?"; "If that's in her thought bubble, what do you think she's feeling? What's her mood like?"; and "If that's in her thought bubble, what do you think she'll do next?" If the therapist and child take a break to play a game, the therapist might subtly bring the concept of thought bubbles into their game (e.g., "When you moved your guy to that spot on the board, my thought bubble was thinking 'That's a great move. I bet he's going to win now' ").

After introducing the concept that different thoughts have different connections to mood and behavior, the next step is to help the child learn how to identify different types of maladaptive thoughts. The therapist can use the concept of "thinking traps" to help the child understand that specific patterns of negative or inaccurate thoughts can get kids stuck thinking about stressful circumstances in unnecessarily distressing and interfering ways. These thinking traps include "catastrophizing" (i.e., thinking the worst; assuming the worst possible sequence of events will occur, no matter how little the chance), "fortune telling"/"jumping to conclusions" (i.e., assuming the outcome of a situation before it happens or getting all of the facts), "walking with blinders"/"mental filtering" (i.e., focusing on the negative aspects of a situation and filtering out the positive aspects), "mind reading" (i.e., assuming someone is thinking bad things about you, without concrete evidence to support it), "emotional reasoning" (i.e., believing one's feelings about a situation represent reality), as well as "black-and-white" or "all-or-nothing" thinking (i.e., overly rigid, dichotomous thought patterns that fail to recognize the nuances of a situation).

When introducing these thinking traps, it can be helpful to present a handout that summarizes them and includes examples (and images if possible). For children it can be helpful to come up with fun characters for each (e.g., "Frank the Fortune Teller," "Blinders Bill," "Joey the Jumps-to-Conclusions Kangaroo"). To bring the material to life and promote engagement, the therapist and child can role-play these characters in various situations, each time falling into that character's specific thinking trap. The therapist and child can make props to assist in these role plays (e.g., making a silly crystal ball for Frank the Fortune Teller, making horse blinders for Blinders Bill to wear). The more exaggerated and silly the role play (e.g., actually hopping around the room when playing Joey the Jumps-to-Conclusions Kangaroo), the more likely the child will be able to remember these characters and their associated thinking traps at later points in time.

After helping the child learn how to identify different patterns of maladaptive thoughts, the next step is to help the child assess the reality, impact, or helpfulness of his or her *own* thoughts about stressful situations or circumstances. The therapist's role is to (1) support the child in identifying the cognitive distortions that are influencing his or her interpretation of a situation, (2) weigh the evidence for and against that interpretation, and (3) help the child generate more realistic alternative thoughts. When evaluating the evidence for and against a negative interpretation, the therapist should emphasize the importance of considering evidence that is objective and/or observable.

Importantly, the therapist should be cautious not to simply tell the child whether his or her various thoughts are helpful or whether he or she is falling into a specific thinking trap. Instead, the therapist should use Socratic methods to help the child identify for him- or herself how realistic or productive his or her thoughts seem to be. To facilitate this process, the therapist can ask the following questions, as appropriate:

- "What are your thoughts about [situation]? What do you think will happen?"
- "How likely do you think [the feared/negative outcome] is?"
- "Has [the feared/negative outcome] ever happened to anyone you know?"
- "When [the feared event] happened to your friend, did [the feared outcome] actually happen?"
- "How bad would it be if [the feared/negative outcome] occurred?"
- "Are there any of the thinking traps you might be falling into? [e.g., In what ways are you possibly thinking like Blinders Bill?]"

For children facing noncatastrophic stressors, this line of questioning can help reveal possible distortions and biases in the child's processing of the situation. The responses to the above questions can be used to help the child generate more realistic or optimistic outlooks about the situations and circumstances that are stressing him or her. Needless to say, questions such as "What's the worst that could happen?" and "If that did happen, how bad would that really be?" are not appropriate when children are indeed facing catastrophic circumstances (e.g., chronic illness, family violence, the death of a loved one). In these situations, the child may still benefit from exploring coping mechanisms, considering whether others have experienced that situation, and evaluating whether his or her distressing feelings would last forever.

While the child is still learning how to produce more realistic and adaptive thoughts, the therapist will typically need to supply several to start. Throughout this process, the therapist should validate the child's feelings about the situation, but refrain from endorsing his or her distorted beliefs. For example:

> "It sounds like you're worried that your friend is mad at you because he/she didn't say hi to you in the hallway. It makes sense that you would feel upset if you thought that your closest friend was mad at you! Let's take a look at the evidence there is for and against this belief. [*The therapist should assist the child in identifying objective evidence, using Socratic probing and running through the different thought traps*.] Now, let's think of some alternatives to the thought that he/she is mad at you. Remember, these thoughts are meant to be realistic and helpful! What are some other possible explanations for [him/her] not saying hi? [*The therapist allows the child to list any alternative thoughts that immediately come to mind*.] Nice work using your flexible thinking skills! I also thought of some other possibilities to add to our list. [*The therapist should help compile a list of several realistic alternatives; e.g., he/she may not have seen you, he/she may have been in a rush, he/she may have thought that you didn't see him/her! If the child is having difficulty identifying alternative thoughts, the therapist could role-play the friend and act out different alternatives*.]"

After proceeding through this restructuring process, the therapist should ask the child to rate again the likelihood of his or her original thought. If his or her original thought is the "worst case scenario," the therapist might guide the child through exploring how he or she could cope if this scenario came true (i.e., "decatastrophizing"). Although it can be difficult for some children to confront their most feared outcomes, children often find their initial predictions more distressing than they would be in reality.

When implementing cognitive strategies with youth populations, the therapist must be sensitive to the child's verbal and developmental level. Particularly for highly abstract or verbally demanding therapy techniques (e.g., cognitive restructuring), the ability to comprehend and encode verbal information and consider hypothetical scenarios is critical. Because the skills required to monitor, articulate, and alter one's own thoughts continue to mature into adolescence and early adulthood, complex cognitive strategies may be less appropriate for developmentally younger children. Encouragingly, research suggests that cognitive techniques can be modified (e.g., using simplified language and active parent involvement) for use with young children with promising results. As with the other techniques described in this chapter, regular practice enhances the likelihood that children will be able to access and successfully apply cognitive restructuring skills under stress.

Distraction

Whereas active primary and secondary control coping strategies (Compas et al., 2017) remain the preferred approach for remediating most youth stressors, distraction techniques can offer an effective strategy for managing stress, anxiety, and pain associated with unavoidable medical procedures, particularly in younger children. Distraction techniques have been successfully implemented with youth undergoing a variety of procedures, including dental extractions (Singh, Samadi, Jaiswal, & Tripathi, 2014), injections (Fowler-Kerry & Ramsay Lander, 1987), and chemotherapy (Redd et al., 1987).

Some hospitals have dedicated "child life specialists" who are equipped with activities, games, and toys to reduce medical distress and improve pain management; however, caregivers can also assemble customized "distraction kits" that cater to their child's unique preferences and interests. Useful distraction tools include social songs (e.g., "Patty Cake," "The Itsy Bitsy Spider"), stimulating sensory activities (e.g., bubbles, pinwheels), interactive games (e.g., puzzles, electronic games), and preferred film clips. Distraction techniques that engage multiple senses may be more immersive and, thus, more effective than less engaging techniques. Some medical centers offer canine therapy or pet visitation for inpatient and outpatient procedures. Canine therapy and visitation have been shown to reduce perceived pain (Sobo, Eng, & Kassity-Krich, 2006) and distress (Vagnoli et al., 2015) in child inpatients.

Mindfulness

Mindfulness describes a state of attending to the present moment, in a nonjudgmental manner (Brown & Ryan, 2003; Kabat-Zinn, 1994). Western psychosocial treatments have increasingly integrated Eastern traditions that use meditation techniques to intentionally achieve mindful states, and recent years have witnessed increasing applications of mindfulness-based interventions applied in youth settings (Barnert, Himelstein, Herbert, Garcia-Romeu, & Chamberlain, 2014; Burke, 2010; Kallapiran, Koo, Kirubakaran, & Hanock, 2015; Mendelson et al., 2010). Increasingly, studies are showing that mindfulness is an effective technique for reducing stress in youth populations (e.g., Biegel, Brown, Shapiro, & Schubert, 2009), with researchers theorizing that mindfulness leads to improved psychological well-being through sustained exposure to internal cues (e.g., heart rate, respiration), enhanced self-awareness, and cognitive changes associated with adopting a nonjudgmental, accepting attitude (Baer, 2006). Notably, the present-focused framework of mindfulness directly contrasts with thought patterns that characterize future-oriented worries and past-oriented rumination. With regular mindfulness practice, children may be better able to curb the cognitive and emotional processes that perpetuate stress and associated psychological problems. Although mindfulness is distinct from relaxation strategies, children may find that this practice has a secondary benefit of making them feel more relaxed and at ease.

Although mindfulness is often practiced as its own meditative session, a mindful approach can be applied to a variety of youth activities, from practicing the violin to eating a snack. As a child is first learning mindfulness, setting aside time for dedicated, guided mindfulness meditation is recommended. Whenever possible, initial mindfulness sessions should be conducted in a comfortable, quiet room, and the child should be allowed to sit or lie down, as preferred. During these sessions, the therapist assists the child to (1) anchor his or her attention in the present moment using the natural rhythm of his or her breath (or another observation target), (2) observe his or her thoughts, feelings, and physical sensations without judgment, and (3) bring his or her attention back to the present if it is carried away by thoughts, feelings, or other distractions.

Before beginning a mindfulness exercise, a target of observation is selected to help "anchor" the child in the present moment. The natural rhythm of a child's own breathing cycle serves as a familiar and accessible target; however, other potential cues include the paced, gentle tone of a bell or the child's own footsteps (e.g., during a walking meditation). A child who has already learned diaphragmatic breathing will be familiar with the process of attending to his or her breath and may transition more easily into mindfulness meditation practice.

When practicing mindfulness, children are instructed to notice their thoughts, feelings, and physical sensations as they arise in an accepting, nonjudgmental fashion. For some children, this practice may be accompanied by increased attention to physical sensations that are perceived as aversive or distressing (e.g., natural fluctuations in heart rate). Particularly for children with panic disorder or elevated anxiety sensitivity, it is important to practice observing physical sensations nonjudgmentally prior to engaging in mindfulness meditation. A therapist might facilitate this process in the following way:

> "Sometimes, even when we are not in a frightening situation, our bodies send us messages that can make us feel nervous or afraid—like our heart might race or we might feel out of breath. But just because our body is sending us these messages doesn't mean we are in actual danger. What other kinds of situations might make your heart beat really fast, even though you are not feeling worried or afraid? [*The therapist should elicit positive and/or neutral situations, such as feeling excited or engaging in physical activity.*] Exactly! Our bodies can send us the same kinds of messages in lots of different situations, and how we accept these messages can change how we feel about them. If every time you felt excited about something, you thought, 'my heart is pounding—I must be afraid!' how do you think you might feel? [*The therapist should help the child to understand how the extent to which he or she accepts his or her interpretation of physical sensations influences how he or she feels about a situation.*] Today, we are going to work on observing the physical feelings in our bodies. Instead of judging those feelings as good or bad, we are simply going to notice and describe them. Another way of describing the feeling 'My heart is pounding!' in a nonjudgmental way is by simply noticing 'My heart is beating faster' or 'My heart rate increased.' [*The therapist should guide the child through reframing different physical sensations using objective/neutral language, as necessary.*] Sometimes, when a physical sensation—like a fast heartbeat—makes us feel nervous or afraid, we try to avoid the feeling or make it go away. For example, we might distract ourselves by focusing on something else. If those feelings come up during our mindfulness practice today, instead of judging the feeling as good or bad, or distracting yourself from it, I want you to notice it in the same way we just practiced."

Once the child has demonstrated that he or she can observe his or her physical sensations nonjudgmentally, the therapist can introduce the concept of mindfulness and initiate a mindfulness meditation session. The therapist might introduce mindfulness in the following way:

> "We are going to practice something called mindfulness. Mindfulness is a way of paying attention to your thoughts and feelings in the present moment, without judging your experience as good or bad. Beginning to practice mindfulness is like learning any new skill. The more you do it, the easier it will become. During your practice, you might get distracted by sounds, feelings, or thoughts that enter your mind—and that's okay! When you find that you've become distracted, notice the thought or

> feeling that drew your attention away, and bring your attention back to the present moment using your breath [or other target of observation].
>
> "Start by getting into a comfortable position in your chair [or on the floor] and gently close your eyes. Notice the feeling of the chair [or floor] against your legs, back, shoulders, and arms. Start by taking a long, slow breath in through your nose . . . and breathe out through your mouth. [*The therapist should demonstrate slow inhales and exhales audibly along with the child, repeating this instruction several times as the child tunes into the flow of his or her own breathing cycle*.] Feel your belly as it rises and falls with your breath. Just notice the rhythm created by your breath, without trying to change anything. Notice when you get distracted by sounds, thoughts, or feelings . . . and return to the rhythm of your breath, in through your nose . . . and out through your mouth. [*The therapist should periodically repeat these phrases, as needed, throughout the mindfulness session*.]"

Particularly when first initiating mindfulness meditation, the child may find it difficult to sustain present-focused attention beyond 3–5 minutes. The therapist may choose to have initial exercises last only a few minutes and increase the duration as the child becomes more familiar with mindfulness practice. It is not uncommon for the child to become distracted by memories, worries, future plans, daydreams, and stimuli in the external environment. Some children may be critical of their initial inability to maintain a nonjudgmental, present-focused awareness. The therapist should reassure the child that it is natural and expected for him or her to become distracted by thoughts and feelings during the session and should instruct the child to observe the thought/feeling/sensation that drew his or her attention away and to gently return to the present moment by attending to his or her breath cycle. The therapist can facilitate this process by periodically instructing the child to bring his or her attention to the present moment.

To strengthen mindfulness skills, mindfulness meditation should be incorporated into out-of-session practices multiple times throughout the week. Therapists may choose to make an audio recording of the mindfulness session for at-home use or provide a caregiver with a mindfulness script to read to the child. There are also commercially available audio mindfulness recordings tailored for children. As the child becomes more adept at mindfulness meditation, mindfulness principles can be applied to any number of activities, including eating, walking, exercising, and spending time with family and friends.

Lifestyle-Focused Strategies for Youth

Lifestyle-focused stress management strategies work to foster consistent and enduring positive shifts in children's daily routines and interactions with others. Engaging in regular exercise, obtaining adequate sleep, and optimizing interpersonal effectiveness, for example, confer additive benefits and can enhance other stress-reduction strategies. Fortunately, these "prescriptions" can be seamlessly and naturally incorporated into children's daily lives.

Exercise

The roles of exercise in enhancing psychological outcomes and promoting youth wellbeing have underpinnings in biological processes. Increased cardiac vagal activity appears to mediate youth stress-reduction effects of exercise (e.g., Field, 2012), and brain-derived neurotrophic factor (BDNF), a growth protein that supports long-term memory and synaptic plasticity, is thought to underlie exercise-related enhancements in cognition and mood (Szuhany, Bugatti, & Otto, 2015). Exercise can lead to quantifiable changes in biological indices of stress. For example, in a sample of adolescents with depression, Nabkasorn and colleagues (2006) found that 50-minute group jogging sessions were associated with significant decreases in depressive symptoms and the stress hormones cortisol and epinephrine. Exercise may also be effective at reducing sensitivity to physical sensations associated with anxiety (i.e., anxiety sensitivity, discussed earlier; Otto et al., 2016). Research specifically focusing on children, adolescents, and young adults has found that physical exercise (particularly short bouts of intense physical activity) can enhance a variety of outcomes, including executive functioning skills (for a review, see Verburgh, Königs, Scherder, & Oosterlaan, 2014), cognitive performance (for a review, see Sibley & Etnier, 2003), and mood (e.g., Field, 2012; Motta, McWilliams, Schwartz, & Cavera, 2012; Taylor, Sallis, & Needle, 1985). Although these effects are relatively modest (Larun, Nordheim, Ekeland, Hagen, & Heian, 2006), they also provide an affective "edge" without the use of medications, which may be desirable for many families. Thus therapists should encourage children to engage in regular physical exercise to enhance their psychological well-being outside of treatment sessions. Children can elect to participate in organized team-based sports (e.g., soccer, basketball), individual sports (e.g., swimming, ballet), or informal exercise practices (e.g., jogging, yoga).

Sleep

Like physical activity, sleep is intimately linked to cognitive and emotional health. Pediatric sleep disturbance frequently co-occurs with youth stress symptoms (Gregory & Sadeh, 2012), and problems with sleep and mood can be mutually exacerbating. For example, the loss of sleep-related emotion regulatory functions increases emotional reactivity to daily challenges and setbacks (Baum et al., 2014; Reddy, Palmer, Jackson, Farris, & Alfano, 2017). In contrast, children who get healthy amounts of sleep experience less distress when recalling emotionally salient events later on. Sleep processes, specifically during the rapid eye movement (REM) stage associated with dreaming, have been shown to reduce the "affective tone" of memories over time. For children who get adequate sleep in the days following an emotionally distressing event, such as the loss of a pet or an argument with a friend, REM sleep appears to help shed layers of emotional intensity associated with this event when it first occurred.

Given the critical roles of sleep in memory encoding, consolidation, and emotion regulation, researchers have even suggested remediating sleep disturbances prior to engaging in other psychosocial treatment in order to fully reap the benefits (e.g., Zalta et al., 2013). Indeed, research on individuals undergoing CBT treatment for social anxiety disorder found that poor sleep quality was associated with slower symptom improvements and poorer posttreatment outcomes (Zalta et al., 2013). For children with heightened stress presenting for treatment, completing a brief intervention to improve sleep hygiene and bedtime habits, such as cognitive-behavioral therapy for insomnia (CBT-I), may optimize the child's ability to engage with and benefit from treatment.

A therapist working to treat a child with significant sleep difficulties should begin by helping the parents establish a comfortable, sleep-promoting bedroom environment. If possible, families should be instructed to keep their child's bedroom cool and dark. For rooms with exposure to sunlight or light pollution, the family may choose to hang blackout curtains to block excess light from entering the room. Similarly, noise can be counteracted by using a fan or white noise machine. The therapist should stress to the family that, to the extent that it is possible, the child's bedroom should be reserved for sleep; other activities, such as homework, eating, time on a tablet, and other playing, should be done elsewhere. Importantly, the therapist should instruct the family to remove electronics such as televisions, computers, tablets, and phones from the child's bedroom.

The therapist should work with the child and family to improve lifestyle habits that may be affecting the child's sleep. Important targets include avoiding stimulants in the late afternoon (e.g., tea, hot chocolate, coffee, medications), avoiding spicy or rich foods before bed to prevent indigestion, and engaging in regular daily exercise. It is also critical to implement a consistent bedtime and wake time. If children cannot fall asleep within 20 minutes of their bedtime, it is recommended that they leave their bedrooms and engage in a quiet, technology-free activity (e.g., reading) and only return to bed once they're sleepy. Although children who have difficulty falling or staying asleep may complain of tiredness during the day, it is important that they arise at a consistent time each day and avoid napping, regardless of the time that they fell asleep. This will maximize the chance that they will be able to fall asleep at bedtime the following evenings.

Interpersonal Effectiveness

Childhood and adolescence are characterized by unique social stressors, including rapidly changing relationships, social cliques, and interpersonal/relational aggression. As youths learn to independently navigate social relationships, it becomes increasingly crucial for them to become equipped with the necessary skills for effectively communicating with others and asserting personal boundaries.

Assertiveness describes the act of standing up for the rights of oneself or others without experiencing significant distress or utilizing excessive aggression. Research suggests that assertiveness training is effective at reducing symptoms of stress and mood and improving various indices of psychological well-being (see Speed, Goldstein, & Goldfried, 2018). Children who are stressed and who have anxious and inhibited temperaments, in particular, may benefit from assertiveness training to improve their comfort and willingness to advocate for their own needs, interests, and boundaries. Inhibited children may have concerns about the consequences of communicating assertively (e.g., that they will inconvenience someone or that others will think they are being "pushy" or "demanding"). Cognitive reappraisal strategies (as detailed earlier) can help such a child assess the actual likelihood of feared interpersonal outcomes occurring and can help the child consider alternative thoughts (e.g., that others might respect them for standing up for themselves).

The therapist can further support a child's communication skills through role plays and exposures. As effective communication is a fairly sophisticated skill, the therapist may first choose to model assertive communication. During this practice, the therapist should demonstrate using "I" statements that express how he or she feels and by making clear requests. Additionally, the therapist should present a confident yet respectful tone (e.g., eye contact, a firm but pleasant tone) and empathic understanding, when appropriate. The following example is from a hypothetical case of a child who was experiencing a conflict with a friend's online behaviors:

> "Let's practice assertiveness with an example that you provided earlier. You told me that your friend posted an embarrassing comment about you online, and that you want him/her to take it down. You might feel like yelling at him/her to take the

comment down or avoiding the situation altogether. Let me show you how you could ask for what you want using the skills we discussed earlier: using 'I' statements, telling your friend how you feel, and making a clear request. [*The therapist demonstrates*.] 'I know you probably didn't mean for it to come off this way, but I felt embarrassed by the comment you posted about me online yesterday. I wanted that story to remain between the two of us. I would appreciate it if you would take it down.' "

For illustrative purposes, the therapist may choose to also perform passive and aggressive communication styles in order to demonstrate ineffective forms of communication (i.e., to show the child what not to do). Once the therapist has demonstrated various examples of assertiveness, he or she can provide the child with the opportunity to practice assertive communication using role plays.

Additional Stress Management Strategies for Youth

Although we have presented the array of clinical stress management strategies for youth that have received the most extensive research support, there are certainly a number of other strategies that are used in clinical practice with children and adolescents. The omission of many clinical stress management practices—such as biofeedback, hypnosis, gratitude journaling, and yoga—from this child-focused chapter reflects the fact that rigorous evaluations in youth samples have not been conducted on these clinical practices. Although illustrative case studies and retrospective chart reviews of these clinical methods can be found throughout the child literature, at this stage, biofeedback, hypnosis, gratitude journaling, and yoga should still be considered "experimental" strategies for the management of stress in children and adolescents. This designation is not to suggest that these clinical methods cannot work or do not work; rather, this designation reflects the fact that these clinical methods, when applied to youth, have not been scrutinized in adequately powered controlled trials. Accordingly, these are considered to be clinical methods of unknown efficacy when applied to youth.

CONCLUDING THOUGHTS

Although stress in youth constitutes a very serious public health concern, a substantial evidence base now supports a broad portfolio of clinical strategies that can be effective in reducing stress and stress-related impairments. In this chapter, we presented leading clinical stress management strategies for youth, including problem-focused strategies that work to resolve the circumstances of stress, emotion-based strategies that work to alter the experiential aspects of stress (e.g., breathing retraining, progressive muscle relaxation, cognitive reappraisal, distraction, and mindfulness), and lifestyle-based strategies that work to foster general shifts in children's daily routines and interactions with others (e.g., exercise scheduling, maintaining good sleep hygiene, training in interpersonal effectiveness).

Increasingly, research has highlighted the fact that there is no universally optimal strategy for coping with stress. Rather, the most effective stress management techniques and targets vary across situations, circumstances, and goals. Children and adolescents facing relatively controllable stressors often benefit from problem-solving stress management strategies, whereas children and adolescents facing more uncontrollable stressors (e.g., those facing a major chronic illness) may benefit more from emotion-focused and/or lifestyle-focused stress management strategies. Youth are best served by broadly informed practitioners who can flexibly apply different stress management techniques across various situations. This is highly consistent with larger trends in the mental health care literature that increasingly embrace flexibility, as illustrated in modularized treatments, sequenced treatment options, and personalized care strategies (e.g., Barlow & Comer, 2013; Hong, Cornacchio, Pettit, & Comer, 2019; Weisz et al., 2012).

Despite the development of effective techniques for managing youth stress, problems persist in the availability, accessibility, and acceptability of quality care options (see Comer & Barlow, 2014). Geographic disparities in quality providers, person-power problems in the mental health workforce, overburdened community mental health centers operating with long wait lists, and stigma-related concerns interfere with needed service utilization. Accordingly, in recent years, practitioners have increasingly adopted innovative delivery methods, such as treating children in natural settings (e.g., Carpenter, Pincus, Furr, & Comer, 2018; Sanchez et al., 2018), offering intensive destination treatment options (e.g., Cornacchio et al., 2019), and leveraging digital technologies to overcome traditional barriers to care (Bry, Chou, Miguel, & Comer, 2018; Comer et al., 2017; Doss, Feinberg, Rothman, Roddy, & Comer, 2017).

Indeed, the past decade has seen a vast proliferation of digital behavioral intervention technologies (e.g., mobile apps) targeting youth stress and anxiety-related problems (e.g., Bry et al., 2018). Moreover, it has been envisioned that in the not too far-off future, much of stress management and wellness promotion will entail sophisticated personalized interventions drawing directly on wearable (and even implanted) computing devices with sensing technologies that can detect optimal "moments" for intervention (Comer, Conroy, & Timmons, 2019; Hunkin, King, & Zajac, 2019). Emerging wearable devices are already using sensing technologies to collect people's physiological data in everyday life in relatively unobtrusive manners, and machine learning developments are promising to afford advanced opportunities to deliver strategic and responsive in situ supports and guidance throughout the day upon detection of dynamic shifts in physiological states (Comer, Conroy, & Timmons, 2019; Nahum-Shani et al., 2018). Given continued digital divides in society across income and race, there is limited support for the majority of technology-based treatments in the consumer marketplace that target youth anxiety and stress (Bry et al., 2018; Comer, Conroy, & Timmons, 2019). With our ability now to use technology to collect more data than we know what to do with and the fact that technology-based opportunities are rapidly outpacing the development of relevant standards for guiding them, we must proceed in these exciting directions with caution (Chou, Bry, & Comer, 2017).

REFERENCES

Abela, J. R. Z., Brozina, K., & Haigh, E. P. (2002). An examination of the Response Styles Theory of Depression in third- and seventh-grade children: A short-term longitudinal study. *Journal of Abnormal Child Psychology, 30,* 515–527.

Aldao, A. (2013). The future of emotion regulation research: Capturing context. *Perspectives on Psychological Science, 8,* 155–172.

Aldao, A., Sheppes, G., & Gross, J. J. (2015). Emotion regulation flexibility. *Cognitive Therapy and Research, 39,* 263–278.

Ayers, T. S., Sandler, I. N., West, S. G., & Roosa, M. W. (1996). A dispositional and situational assessment of children's coping: Testing alternative models of coping. *Journal of Personality, 64*(4), 923–958.

Baer, R. A. (2006). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice, 10*(2), 125–143.

Barlow, D. H., & Comer, J. S. (2013). What are the optimal treatment courses for geriatric anxiety, and how do we find out? *American Journal of Psychiatry, 170,* 707–711.

Barnert, E. S., Himelstein, S., Herbert, S., Garcia-Romeu, A., & Chamberlain, L. J. (2014). Exploring an intensive meditation intervention for incarcerated youth. *Child and Adolescent Mental Health, 19*(1), 69–73.

Baum, K. T., Desai, A., Field, J., Miller, L. E., Rausch, J., & Beebe, D. W. (2014). Sleep restriction worsens mood and emotion regulation in adolescents. *Journal of Child Psychology and Psychiatry, 55*(2), 180–190.

Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*(2), 183–214.

Berntson, G. G., Bigger, J. T., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34*(6), 623–648.

Biegel, G. M., Brown, K. W., Shapiro, S. L., & Schubert, C. M. (2009). Mindfulness-based stress reduction for the treatment of adolescent psychiatric outpatients: A randomized clinical trial. *Journal of Consulting and Clinical Psychology, 77*(5), 855–866.

Bonanno, G. A., & Burton, C. L. (2013). Regulatory flexibility: An individual differences perspective on coping and emotion regulation. *Perspectives on Psychological Science, 8*(6), 591–612.

Boyle, D., & Hassett-Walker, C. (2008). Reducing overt and relational aggression among young children. *Journal of School Violence, 7*(1), 27–42.

Brenner, S. L., & Beauchaine, T. P. (2011). Pre-ejection period reactivity and psychiatric comorbidity prospectively predict substance use initiation among middle-schoolers: A pilot study. *Psychophysiology, 48*(11), 1588–1596.

Brenner, S. L., Beauchaine, T. P., & Sylvers, P. D. (2005). A comparison of psychophysiological and self-report measures of BAS and BIS activation. *Psychophysiology, 42*(1), 108–115.

Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology, 84*(4), 822–848.

Bry, L. J., Chou, T., Miguel, E., & Comer, J. S. (2018). Consumer smartphone apps marketed for child and adolescent anxiety: A systematic review and content analysis. *Behavior Therapy, 49,* 249–261.

Burke, C. A. (2010). Mindfulness-based approaches with children and adolescents: A preliminary review of current research in an emergent field. *Journal of Child and Family Studies, 19*(2), 133–144.

Calkins, S. D. (2007). The emergence of self-regulation: Biological and behavioral control mechanisms supporting toddler competencies. In C. A. Brownell & C. B. Kopp (Eds.), *Socioemotional development in the toddler years: Transitions and transformations* (pp. 261–284). New York: Guilford Press.

Carpenter, A. L., Pincus, D. B., Furr, J. M., & Comer, J. S. (2018). Working from home: An initial pilot examination of videoconferencing-based cognitive-behavioral therapy for anxious youth delivered to the home setting. *Behavior Therapy, 49,* 917–930.

Centers for Disease Control and Prevention & Kaiser Permanente. (2016). The ACE Study survey data. Retrieved from *www.cdc.gov/violenceprevention/acestudy/about.html.*

Chou, T., Bry, L. J., & Comer, J. S. (2017). Overcoming traditional barriers only to encounter new ones: Doses of caution and direction as technology-enhanced treatments begin to "go live." *Clinical Psychology: Science and Practice, 24,* 241–244.

Chou, T., Carpenter, A., Kerns, C. E., Elkins, R. M., Green, J. G., & Comer, J. S. (2017). Disqualified qualifiers: Evaluating the utility of the revised DSM-5 definition of potentially traumatic events among area youth following the Boston Marathon bombing. *Depression and Anxiety, 34*(4), 367–373.

Cohen, S., Gianaros, P. J., & Manuck, S. B. (2016). A stage model of stress and disease. *Perspectives on Psychological Science, 11*(4), 456–463.

Comer, J. S., & Barlow, D. H. (2014). The occasional case against broad dissemination and implementation: Retaining a role for specialty care in the delivery of psychological treatments. *American Psychologist, 69,* 1–18.

Comer, J. S., Bry, L., Poznanski, B., & Golik, A. M. (2016). Children's mental health in the context of terrorist attacks, ongoing threats, and possibilities of future terrorism. *Current Psychiatry Reports, 18*(9), 79.

Comer, J. S., Conroy, K., & Timmons, A. (2019). Ensuring wearable devices don't wear out their welcome: Cautions for the mental health care road ahead. *Clinical Psychology: Science and Practice, 26*(3), e12297.

Comer, J. S., Dantowitz, A., Chou, T., Edson, A. L., Elkins, R. M., Kerns, C., et al. (2014). Adjustment among area youth after the Boston Marathon bombing and subsequent manhunt. *Pediatrics, 134,* 7–14.

Comer, J. S., DeSerisy, M., & Green, J. G. (2016). Caregiver reports of Internet exposure and posttraumatic stress symptoms among Boston-area youth following the 2013 Marathon bombing. *Evidence-Based Practice in Child and Adolescent Mental Health, 1*(2–3), 86–102.

Comer, J. S., Furr, J. M., Miguel, E., Cooper-Vince, C. E., Carpenter, A. L., Elkins, R. M., et al. (2017). Remotely delivering real-time parent training to the home: An initial randomized trial of Internet-delivered parent–child interaction therapy (I-PCIT). *Journal of Consulting and Clinical Psychology, 85,* 909–917.

Comer, J. S., Hong, N., Poznanski, B., Silva, K., & Wilson, M. (2019). Evidence base update on the treatment of early childhood anxiety and related problems. *Journal of Clinical Child and Adolescent Psychology, 48*(1), 1–15.

Comer, J. S., & Kendall, P. C. (2004). A symptom-level examination of parent–child agreement in the diagnosis of anxious youths. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(7), 878–886.

Comer, J. S., & Kendall, P. C. (2007). Terrorism: The psychological impact on youth. *Clinical Psychology: Science and Practice, 14,* 179–212.

Comer, J. S., Roy, A. K., Furr, J. M., Gotimer, K., Beidas, R. S., Dugas, M. J., et al. (2009). The Intolerance of Uncertainty Scale for Children: A psychometric evaluation. *Psychological Assessment, 21*(3), 402–411.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127,* 87–127.

Compas, B. E., Jaser, S. S., Bettis, A. H., Watson, K. H., Gruhn, M. A., Dunbar, J. P., et al. (2017). Coping, emotion regulation, and psychopathology in childhood and adolescence: A meta-analysis and narrative review. *Psychological Bulletin, 143,* 939–991.

Compas, B. E., Jaser, S. S., Dunn, M. J., & Rodriguez, E. M. (2012). Coping with chronic illness in childhood and adolescence. *Annual Review of Clinical Psychology, 8,* 455–480.

Copeland, W. E., Keeler, G., Angold, A., & Costello, E. J. (2007). Traumatic events and posttraumatic stress in childhood. *Archives of General Psychiatry, 64*(5), 577–584.

Cornacchio, D., Furr, J. M., Sanchez, A., Hong, N., Feinberg, L., Tenenbaum, R., et al. (2019). Intensive group behavioral treatment (IGBT) for children with selective mutism: A preliminary randomized controlled trial. *Journal of Consulting and Clinical Psychology, 87*(8), 720–733.

Cornacchio, D., Sanchez, A. L., Chou, T., & Comer, J. S. (2017). Cognitive-behavioral therapy for children and adolescents. In S. G. Hofmann & G. Asmundson (Eds.), *The science of cognitive behavioral therapy* (pp. 257–288). New York: Elsevier.

Cornacchio, D., Sanchez, A. L., Roy, A. K., Pincus, D. B., Read, K. L., Holaway, R. M., et al. (2018). Factor structure of the Intolerance of Uncertainty Scale for Children. *Journal of Anxiety Disorders, 53,* 100–107.

Cowen, E. L., Wyman, P. A., Work, W. C., & Parker, G. R. (1990). The Rochester Child Resilience Project (RCRP): Overview and summary of first year findings. *Development and Psychopathology, 2,* 193–212.

Dearing, K. F., & Gotlib, I. H. (2009). Interpretation of ambiguous information in girls at risk for depression. *Journal of Abnormal Child Psychology, 37*(1), 79–91.

DiCorcia, J. A., Sravish, A. V., & Tronick, E. (2013). The Everyday Stress Resilience Hypothesis: Unfolding resilience from a perspective of everyday stress and coping. In G. Laviola & S. Macrì (Eds.), *Current topics in neurotoxicity: Adaptive and maladaptive aspects of developmental stress* (pp. 67–93). New York: Springer.

Doss, B. D., Feinberg, L. K., Rothman, K., Roddy, M. K., & Comer, J. S. (2017). Using technology to enhance and expand interventions for couples and families: Conceptual and methodological considerations. *Journal of Family Psychology, 31,* 983–993.

Dubow, E. F. (1993). *I CAN DO problem-solving program training manual.* Bowling Green, OH: Bowling Green State University.

Dubow, E. F., Schmidt, D., McBride, J., Edwards, S., & Merk, F. L. (1993). Teaching children to cope with stressful experiences: Initial implementation and evaluation of a primary prevention program. *Journal of Clinical Child Psychology, 22*(4), 428–440.

Ehrenreich-May, J., Kennedy, S. M., Sherman, J. A., Bilek, E. L., Buzzella, B. A., Bennett, S. M., et al. (2017). *Unified protocols for transdiagnostic treatment of emotional disorders in children and adolescents: Therapist guide*. New York: Oxford University Press.

Fairchild, G., van Goozen, S. H. M., Stollery, S. J., Brown, J., Gardiner, J., Herbert, J., et al. (2008). Cortisol diurnal rhythm and stress reactivity in male adolescents with early-onset or adolescence-onset conduct disorder. *Biological Psychiatry, 64*(7), 599–606.

Field, T. (2012). Exercise research on children and adolescents. *Complementary Therapies in Clinical Practice, 18*(1), 54–59.

Finkelhor, D., Shattuck, A., Turner, H., & Hamby, S. (2013). Improving the Adverse Childhood Experiences Study Scale. *JAMA Pediatrics, 167*(1), 70–75.

Fowler-Kerry, S., & Ramsay Lander, J. (1987). Management of injection pain in children. *Pain, 30*(2), 169–175.

Fowles, D. C. (1986). The eccrine system and electrodermal activity. In M. G. H. Coles, E. Donchin, & S. W. Porges (Eds.), *Psychophysiology: Systems, processes, and applications* (pp. 51–96). New York: Guilford Press.

Freeman, J., Sapyta, J., Garcia, A., Compton, S., Khanna, M., Flessner, C., et al. (2014). Family-based treatment of early childhood obsessive–compulsive disorder: The Pediatric Obsessive–Compulsive Disorder Treatment Study for Young Children (POTS Jr)—A randomized clinical trial. *JAMA Psychiatry, 71*(6), 689–698.

Furr, J. M., Comer, J. S., Edmunds, J., & Kendall, P. C. (2010). Disasters and youth: A meta-analytic examination of posttraumatic stress. *Journal of Consulting and Clinical Psychology, 78*(6), 765–780.

Furr, J. M., Comer, J. S., Villodas, M. T., Poznanski, B., & Gurwitch, R. (2018). Trauma and child psychopathology: From risk and resilience to evidence-based intervention. In J. N. Butcher & P. C. Kendall (Eds.), *APA handbook of psychopathology: Child and adolescent psychopathology* (pp. 187–211). Washington, DC: American Psychological Association.

Gaither, J. R., Shabanova, V., & Leventhal, J. M. (2018). US national trends in pediatric deaths from prescription and illicit opioids, 1999–2016. *JAMA Network Open, 1*(8), e186558.

Gelhaar, T., Seiffge-Krenke, I., Borge, A., Cicognani, E., Cunha, M., Loncaric, D., et al. (2007). Adolescent coping with everyday stressors: A seven-nation study of youth from central, eastern, southern, and northern Europe. *European Journal of Developmental Psychology, 4*(2), 129–156.

Graziano, P., & Derefinko, K. (2013). Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology, 94*(1), 22–37.

Gregory, A. M., & Sadeh, A. (2012). Sleep, emotional and behavioral difficulties in children and adolescents. *Sleep Medicine Reviews, 16*(2), 129–136.

Gunnar, M. R. (2003). Integrating neuroscience and psychological approaches in the study of early experiences. *Annals of the New York Academy of Sciences, 1008*(1), 238–247.

Gunnar, M. R., & Adam, E. K. (2012). The hypothalamic–pituitary–adrenocortical system and emotion: Current wisdom and future directions. *Monographs of the Society for Research in Child Development, 77*(2), 109–119.

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology, 34*(7), 953–967.

Heron, M. (2018). Deaths: Leading causes for 2016 (DHHS Publication No. 2018-1120). *National Vital Statistics Reports, 67*(6), 1–76. Retrieved from *www.cdc.gov/nchs/data/nvsr/nvsr67/nvsr67_06.pdf*.

Higa-McMillan, C. K., Francis, S. E., Rith-Najarian, L., & Chorpita, B. F. (2016). Evidence base update: 50 years of research on treatment for child and adolescent anxiety. *Journal of Clinical Child and Adolescent Psychology, 45*(2), 91–113.

Hilt, L. M., McLaughlin, K. A., & Nolen-Hoeksema, S. (2010). Examination of the response styles theory in a community sample of young adolescents. *Journal of Abnormal Child Psychology, 38*(4), 545–556.

Hong, N., Cornacchio, D., Pettit, J., & Comer, J. S. (2019). Coal mine canaries in clinical psychology: Getting better at identifying early signals of treatment nonresponse. *Clinical Psychological Science, 7*(6), 1207–1221.

Hunkin, H., King, D. L., & Zajac, I. T. (2019). Wearable devices as adjuncts in the treatment of anxiety-related symptoms: A narrative review of five device modalities and implications for clinical practice. *Clinical Psychology: Science and Practice, 26*(3), e12290.

Kabat-Zinn, J. (1994). *Wherever you go, there you are: Mindfulness meditation in everyday life*. New York: Hyperion.

Kallapiran, K., Koo, S., Kirubakaran, R., & Hanock, K. (2015). Effectiveness of mindfulness in improving mental health symptoms of children and adolescents: A meta-analysis. *Child and Adolescent Mental Health, 20*(4), 182–194.

Keller, A. E., Langer, D. A., Pincus, D. B., Kerns, C. E., Elkins, R. M., & Comer, J. S. (2019). A psychometric evaluation of the Distress Intolerance Index for Youth. *Journal of Psychopathology and Behavioral Assessment, 41,* 447–455.

Kelsey, R. M., Ornduff, S. R., & Alpert, B. S. (2007). Reliability of cardiovascular reactivity to stress: Internal consistency. *Psychophysiology, 44*(2), 216–225.

Kendall, P. C., Hudson, J. L., Gosch, E., Flannery-Schroeder, E., & Suveg, C. (2008). Cognitive-behavioral therapy for anxiety disordered youth: A randomized clinical trial evaluating child and family modalities. *Journal of Consulting and Clinical Psychology, 76*(2), 282–297.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology, 84*(3), 394–421.

Kritikos, T. K., Comer, J. S., He, M., Curren, L., & Tompson, M. C. (2019). Combat experiences and PTSD among military-serving parents: A meta-analytic examination of associated offspring and family outcomes. *Journal of Abnormal Child Psychology, 47,* 131–148.

Larun, L., Nordheim, L. V., Ekeland, E., Hagen, K. B., & Heian, F. (2006). Exercise in prevention and treatment of anxiety and depression among children and young people. *Cochrane Database Systems Review, 3.*

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping.* New York: Springer.

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences, 35*(3), 121–143.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds?: Coherence among emotion experience, behavior, and physiology. *Emotion, 5*(2), 175–190.

Mayordomo-Rodríguez, T., Meléndez-Moral, J. C., Viguer-Segui, P., & Sales-Galan, A. (2015). Coping strategies as predictors of well being in youth adult. *Social Indicators Research, 122*(2), 479–489.

McLaughlin, K. A., Koenen, K. C., Hill, E. D., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., et al. (2013). Trauma exposure and posttraumatic stress disorder in a national sample of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(8), 815–830.

McNally, R. J. (2009). Can we fix PTSD in DSM-V? *Depression and Anxiety, 26*(7), 597–600.

Meltzer, L. (2018). *Executive function in education: From theory to practice* (2nd ed.). New York: Guilford Press.

Mendelson, T., Greenberg, M. T., Dariotis, J. K., Gould, L. F., Rhoades, B. L., & Leaf, P. J. (2010). Feasibility and preliminary outcomes of a school-based mindfulness intervention for urban youth. *Journal of Abnormal Child Psychology, 38*(7), 985–994.

Merikangas, K. R., He, J. P., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., et al. (2010). Lifetime prevalence of mental disorders in US adolescents: Results from the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry, 49*(10), 980–989.

Motta, R. W., McWilliams, M. E., Schwartz, J. T., & Cavera, R. S. (2012). The role of exercise in reducing childhood and adolescent PTSD, anxiety, and depression. *Journal of Applied School Psychology, 28*(3), 224–238.

Nabkasorn, C., Miyai, N., Sootmongkol, A., Junprasert, S., Yamamoto, H., Arita, M., et al. (2006). Effects of physical exercise on depression, neuroendocrine stress hormones and physiological fitness in adolescent females with depressive symptoms. *European Journal of Public Health, 16*(2), 179–184.

Nahum-Shani, I., Smith, S. N., Spring, B. J., Collins, L. M., Witkiewitz, K., Tewari, A., et al. (2018). Just-in-time adaptive interventions (JITAIs) in mobile health: Key components and design principles for ongoing health behavior support. *Annals of Behavioral Medicine, 52,* 446–462.

Nickel, C., Kettler, C., Muehlbacher, M., Lahmann, C., Tritt, K., Fartacek, R., et al. (2005). Effect of progressive muscle relaxation in adolescent female bronchial asthma patients: A randomized, double-blind, controlled study. *Journal of Psychosocial Research, 59*(6), 393–398.

Nickel, C., Lahmann, C., Tritt, K., Loew, T. H., Rother, W. K., & Nickel, M. K. (2005). Stressed aggressive adolescents benefit from progressive muscle relaxation: A random, prospective, controlled trial. *Stress and Health, 21*(3), 169–175.

Ollendick, T. H., & Cerny, J. A. (1981). *Clinical behavior therapy with children*. New York: Plenum Press.

Otto, M. W., Eastman, A., Lo, S., Hearon, B. A., Bickel, W. K., Zvolensky, M., et al. (2016). Anxiety sensitivity and working memory capacity: Risk factors and targets for health behavior promotion. *Clinical Psychology Review, 49,* 67–78.

Pagliaccio, D., Luby, J. L., Bogdan, R., Agrawal, A., Gaffrey, M. S., Belden, A. C., et al. (2015). Amygdala functional connectivity, HPA axis genetic variation, and life stress in children and relations to anxiety and emotion regulation. *Journal of Abnormal Psychology, 124*(4), 817–833.

Pfefferbaum, B., Seale, T. W., Brandt, E. N., Pfefferbaum, R. L., Doughty, D. E., & Rainwater, S. M. (2003). Media exposure in children one hundred miles from a terrorism bombing. *Annals of Clinical Psychiatry, 15,* 1–8.

Pincus, D. B., & Friedman, A. G. (2004). Improving children's coping with everyday stress: Transporting treatment interventions to the school setting. *Clinical Child and Family Psychology Review, 7*(4), 223–240.

Pincus, D. B., Madigan, R. J., Kerns, C. E., Hardway, C., & Comer, J. S. (2014). Managing maladaptive mood and arousal. In E. S. Sburlati, H. J. Lyneham, C. A. Schniering, & R. M. Rapee (Eds.), *Evidence-based CBT for anxiety and depression in children and adolescents: A competencies based approach* (pp. 225–246). New York: Wiley Blackwell.

Pincus, D. B., & Otis, J. D. (2001). *I can relax: A relaxation CD for children*. Boston: Psychzone.

Pinker, S. (2012). *The better angels of our nature: Why violence has declined*. New York: Penguin Group.

Polanczyk, G. V., Salum, G. A., Sugaya, L., Caye, A., & Rohde, L. A. (2015). Annual research review: A meta-analysis of the worldwide prevalence of mental disorders in children and adolescents. *Journal of Child Psychology and Psychiatry, 56*(3), 345–365.

Porges, S. W., Doussard-Roosevelt, J., Portales, A. L., & Greenspan, S. I. (1996). Infant regulation of the vagal "brake" predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology, 29*(8), 697–712.

Pruessner, J. C., Hellhammer, D. H., & Kirschbaum, C. (1999). Burnout, perceived stress, and cortisol responses to awakening. *Psychosomatic Medicine, 61*(2), 197–204.

Rapee, R., Wignall, A., Hudson, J., & Schniering, C. A. (2000). *Treating anxious children and adolescents: An evidence-based approach.* Oakland, CA: New Harbinger.

Redd, W. H., Jacobsen, P. B., Die-Trill, M., Dermatis, H., McEvoy, M., & Holland, J. C. (1987). Cognitive/attentional distraction in the control of conditioned nausea in pediatric cancer patients receiving chemotherapy. *Journal of Consulting and Clinical Psychology, 55*(3), 391–395.

Reddy, R., Palmer, C. A., Jackson, C., Farris, S. G., & Alfano, C. A. (2017). Impact of sleep restriction versus idealized sleep on emotional experience, reactivity and regulation in healthy adolescents. *Journal of Sleep Research, 26*(4), 516–525.

Richter, M., & Gendolla, G. H. E. (2009). The heart contracts to reward: Monetary incentives and preejection period. *Psychophysiology, 46*(3), 451–457.

Rith-Najarian, L. R., McLaughlin, K. A., Sheridan, M. A., & Nock, M. K. (2014). The biopsychosocial model of stress in adolescence: Self-awareness of performance versus stress reactivity. *Stress, 17*(2), 193–203.

Roser, M., Ortiz-Ospina, E., Ritchie, H., Hassell, J., & Gavrilov, D. (2019). Our world in data. Retrieved June 15, 2019, from *OurWorldInData.org.*

Rutter, M. (1994). Stress research: Accomplishments and the tasks ahead. In R. J. Haggerty, L. R. Sherrod, N. Garmezy, & M. Rutter (Eds.), *Stress, risk, and resilience in children and adolescents: Processes, mechanisms, and interventions* (pp. 354–385). New York: Cambridge University Press.

Salminen, M., Ravaja, N., Kallinen, K., & Saari, T. (2013). Mediated cues of group emotion during knowledge-work tasks: Effects on subjective and physiological responses. *Interacting with Computers, 25*(1), 60–73.

Sanchez, A. L., Cornacchio, D., Poznanski, B., Golik, A., Chou, T., & Comer, J. S. (2018). The effectiveness of school-based mental health services for elementary-aged children: A meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry, 57,* 153–165.

Sherwood, A., Turner, J. R., Light, K. C., & Blumenthal, J. A. (1990). Temporal stability of the hemodynamics of cardiovascular reactivity. *International Journal of Psychophysiology, 10*(1), 95–98.

Sibley, B. A., & Etnier, J. L. (2003). The relationship between physical activity and cognition in children: A meta-analysis. *Pediatric Exercise Science, 15*(3), 243–256.

Singh, D., Samadi, F., Jaiswal, J., & Tripathi, A. M. (2014). Stress reduction through audio distraction in anxious pediatric dental patients: An adjunctive clinical study. *International Journal of Clinical Pediatric Dentistry, 7*(3), 149–152.

Sobo, E. J., Eng, B., & Kassity-Krich, N. (2006). Canine visitation (pet) therapy: Pilot data on decreases in child pain perception. *Journal of Holistic Nursing, 24*(1), 51–57.

Speed, B. C., Goldstein, B. L., & Goldfried, M. R. (2018). Assertiveness training: A forgotten evidence-based treatment. *Clinical Psychology: Science and Practice, 25*(1), e12216.

Spence, S. H., Sheffield, J. K., & Donovan, C. L. (2003). Preventing adolescent depression: An evaluation of the Problem Solving for Life program. *Journal of Consulting and Clinical Psychology, 1(1),* 3–13.

Spivack, G., & Shure, M. B. (1974). *Social adjustment of young children: A cognitive approach to solving real-life problems*. London: Jossey Bass.

Spivack, G., & Shure, M. B. (1982). The cognition of social adjustment: Interpersonal cognitive problem-solving thinking. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 5., pp. 323–372). New York: Plenum Press.

Stark, L. J., Spirito, A., Williams, C. A., & Guevremont, D. C. (1989). Common problems and coping strategies: I. Findings with normal adolescents. *Journal of Abnormal Child Psychology, 17*(2), 203–212.

Szuhany, K. L., Bugatti, M., & Otto, M. W. (2015). A meta-analytic review of the effects of exercise on brain-derived neurotrophic factor. *Journal of Psychiatric Research, 60,* 56–64.

Taghavi, M. R., Moradi, A. R., Neshat-Doost, H. T., Yule, W., & Dalgleish, T. (2000). Interpretation of ambiguous emotional information in clinically anxious children and adolescents. *Cognition and Emotion, 14*(6), 809–822.

Taylor, C. B., Sallis, J. F., & Needle, R. (1985). The relation of physical activity and exercise to mental health. *Public Health Reports, 100*(2), 195–202.

Vagnoli, L., Caprilli, S., Vernucci, C., Zagni, S., Mugnai, F., & Messeri, A. (2015). Can presence of a dog reduce pain and distress in children during venipuncture? *Pain Management Nursing, 16*(2), 89–95.

Van Eck, M., Berkhof, H., Nicolson, N., & Sulon, J. (1996). The effects of perceived stress, traits, mood states, and stressful daily events on salivary cortisol. *Psychosomatic Medicine, 58*(5), 447–458.

Verburgh, L., Königs, M., Scherder, E. J. A., & Oosterlaan, J. (2014). Physical exercise and executive functions in preadolescent children, adolescents and young adults: A meta-analysis. *British Journal of Sports Medicine, 48*(12), 973–979.

Weissberg, R. P., & Elias, M. J. (1993). Enhancing young people's social competence and health behavior: An important challenge for educators, scientists, policy makers and funders. *Applied and Preventive Psychology, 2,* 179–190.

Weisz, J. R., Chorpita, B. F., Palinkas, L. A., Schoenwald, S. K., Miranda, J., Bearman, S. K., et al. (2012). Testing standard and modular designs for psychotherapy treating depression, anxiety, and conduct problems in youth: A randomized effectiveness trial. *Archives of General Psychiatry, 69*(3), 274–282.

Weisz, J. R., McCabe, M. A., & Dennig, M. D. (1994). Primary and secondary control among children undergoing medical procedures: Adjustment as a function of coping style. *Journal of Consulting and Clinical Psychology, 62*(2), 324–332.

Zalta, A. K., Dowd, S., Rosenfield, D., Smits, J. A. J., Otto, M. W., Simon, N. M., et al. (2013). Sleep quality predicts treatment outcome in CBT for social anxiety disorder. *Depression and Anxiety, 30*(11), 1114–1120.

Zimmer-Gembeck, M. J., & Skinner, E. A. (2016). The development of coping: Implications for psychopathology and resilience. In D. Cicchetti (Ed.), *Developmental psychopathology* (pp. 485–545). Oxford, UK: Wiley.

PART II

STRESS MANAGEMENT METHODS

SECTION A

PSYCHOPHYSIOLOGICAL METHODS

CHAPTER 6

Psychophysiological Interventions for Stress-Related, Medically Unexplained, and Anxiety Disorders

Richard N. Gevirtz

Increasingly, individuals with so-called stress-related disorders represent a significant proportion of patients seeking medical care. Treating these patients is expensive and difficult, and they are not generally satisfied with traditional Western medical care (Andrade, Walters, Gentil, & Laurenti, 2002; Cassidy et al., 2004; Cummings & VandenBos, 1981; Issakidis & Andrews, 2003; Katon, Von Korff, & Lin, 1992; Sharpe & Carson, 2001; Yates, 1984). For this reason, presentation of symptoms that may represent anxiety or the effects of stress are often mishandled in medical settings. One manifestation of this phenomenon has been labeled "medically unexplained physical symptoms" (MUPS). But this problem is present in many other syndromes (e.g., fibromyalgia, chronic pain, irritable bowel syndrome or other functional gastrointestinal disorders). Stress is believed to play an important role in patient reports of physical symptoms. These symptoms burden patients in their well-being and functioning and have a prevalence of approximately 25–50% in primary and specialist care. Medical specialists often find patients with unexplained symptoms or with the syndromes mentioned above difficult to deal with. In fact, some researchers have reported gains in patient outcomes for an evidence-based MUPS-focused communication training program for medical specialists (Weiland et al., 2016; Haller, Cramer, Lauche, & Dobos, 2015).

Furthermore, many population studies have identified large numbers of these patients (including those with anxiety disorders) who have not sought or have given up on medical care (Drossman et al., 1999; Katon, 1996; Katon, Hart, & Montano, 1997). In looking at this problem, den Boeft and colleagues (2016) concluded, "Our results show that MUPS were positively associated with HCU [health care use] over 2 years, even after adjusting for depressive and anxiety disorders and personality traits" (p. 100).

This chapter lays out a heuristic model that integrates psychophysiological measures and biofeedback into a framework that may be useful in assessment and treatment planning. This approach is in sharp contrast to more psychodynamic models that assume that emotional factors operate through some central process to create symptom perceptions where no pathophysiology actually exists. Rather, "functional" symptoms are conceptualized as representing changes in physiology, such that the symptoms are "real," that is, not "all in one's head." This approach has several advantages:

1. It is not in the least pejorative so as to be stigmatizing. As with any medical disorder, symptoms are presented in a scientific causal pathway with as neutral a value valence as possible. Thus patients can go public with their new "diagnosis" without shame or concealment.
2. It appears to produce symptom reduction over long-term follow-ups (Goessl, Curtiss, & Hofmann, 2017; Gevirtz, 2013; Lehrer & Gevirtz, 2014; DeGuire, Gevirtz, Hawkinson, & Dixon, 1996; DeGuire, Gevirtz, Kawahara, & Maguire, 1992; Humphreys & Gevirtz, 2000; Ryan & Gevirtz, 2001; Sharpe & Carson, 2001).
3. It can be incorporated into medical settings, corporate stress management settings, or mental health settings with only small adjustments.

The model works by making a credible case for physiological mediators (between psychological factors and symptoms). For this reason, the following sections describe a case for various autonomic, respiratory, and endocrine pathways that can be used to describe symptoms of many of these syndromes.

POTENTIAL PHYSIOLOGICAL MEDIATORS

Four general physiological systems are candidates as causes for most of the complaints or symptoms we encounter in this realm: the sympathetic branch of the autonomic nervous system (SNS), including the sympathetic adrenal medullary system; the parasympathetic branch of the autonomic nervous system (PNS); the respiratory system; and the hypothalamic–pituitary–adrenal system (HPA). Each has been described in many places (see Woolfolk & Lehrer, Chapter 1, Kusnecov, Norton, & Nissenbaum, Chapter 4, and Sime & Phillips, Chapter 21, all in this volume; Gevirtz, 2017; Gevirtz & Schwartz, 2003; Guyton & Hall, 1995), so I highlight only features that are often overlooked.

The perspective I am advocating involves first assessing the nature of the stress-related complaint, creating a "mediational model" based on multimodal, multimethod analyses, and thereby creating the basis for a treatment plan. To do this we must first review some physiology.

Sympathetic Branch of the Autonomic Nervous System

The SNS is a complex system with multiple pathways synapsing on an intermediate ganglionic plexus and terminating at the target organs. Though named *sympathetic* by Cannon (1929), based on the belief that it operated as a mass action system, modern physiology has rejected this idea in favor of much more specificity (Porges, 1995a).

Morrison (2001) summarized the situation nicely:

> With advances in experimental techniques, the early views of the sympathetic nervous system as a monolithic effector activated globally in situations requiring a rapid and aggressive

> response to life-threatening danger have been eclipsed by an organizational model featuring an extensive array of functionally specific output channels that can be simultaneously activated or inhibited in combinations that result in the patterns of autonomic activity supporting behavior and mediating homeostatic reflexes. With this perspective, the defense response is but one of the many activational states of the central autonomic network. (p. 683)

From the earliest decades of the 20th century, physiologists have postulated the SNS as the primary mediator of the human stress response.

Cannon's "fight or flight" concept (Cannon, 1929) has become a part of everyday language and has heavily influenced both medicine and psychology. This is certainly understandable, because the system seems to act to mobilize the organism for emergency situations. In everyday life, however, this mechanism becomes less clear. Most of us do not experience fight-or-flight types of challenges on an hourly or even daily basis. Rather, modern stress is more likely to stem from issues of social hierarchies, ruminative self-deprecating thoughts, general anxiety, worry, or boredom. Indeed, stress management training with firefighters or police officers almost always centers on issues of bureaucracy, paperwork, relationships, and so forth, not on the dangers of the job. Thus clients with stress-related symptoms cannot usually identify stressors that most would characterize as fight-or-flight triggers unless we move to metaphorical models. This would seem to present a problem for models that postulate stress as primarily an SNS phenomenon.

Those readers familiar with clinical psychophysiological measurement know all too well that supposed indicators of SNS arousal (increased heart rate [HR], skin conductance [SC], and cooler fingertips) rarely cooperate in the clinic and work as a team. Rather, every combination of pattern is seen. This is in contrast to situations that do produce a dramatic response, such as parachuting out of a plane for the first time or being in combat (Biondi & Picardi, 1999).

Nevertheless, subtler SNS pathways are undoubtedly involved in many disorders. These pathways may be unique to a specific organ system, but nonetheless they are potential mediators of symptoms when maintained for longer periods of time (as the stimuli mentioned earlier often are). For example, our group (Gevirtz, Hubbard, & Harpin, 1996; Hubbard, 1996, 1998; Hubbard & Berkoff, 1993; McNulty, Gevirtz, Hubbard, & Berkoff, 1994) has shown that nodules in muscles called *trigger points* are (alpha) sympathetically mediated and are responsive to very mild stressors, such as worry, performance anxiety, and so forth. This is true even when SC or HR responses show only very subtle changes to the same stimuli. Similarly, a number of researchers (Adeyemi, Desai, Towsey, & Ghista, 1999; Waring, Chui, Japp, Nicol, & Ford, 2004) have shown that a high low-frequency (LF):high-frequency (HF) ratio (thought to reflect SNS influence), calculated from sequential R–R intervals of the electrocardiogram (EKG), characterize many patients with irritable bowel syndrome (IBS), a stress-related gastrointestinal disorder. Again, this finding is often not accompanied by other SNS indicators. As another example, Martinez-Lavin and colleagues (Martinez-Lavin, 2001a, 2001b; Martinez-Lavin & Hermosillo, 2000; Martinez-Lavin et al., 1997; Martinez-Lavin, Hermosillo, Rosas, & Soto, 1998) have presented data that characterize fibromyalgia as at least partly stemming from SNS dominance.

Thus, despite the added complexity in the model, the SNS remains a key candidate for mediation of symptoms. The issue becomes difficult to disentangle because the PNS exerts inhibitory influences on the SNS. This phenomenon has been called *accentuated antagonism* (Olshansky, Sabba, Hauptman, & Colucci, 2008; Yang & Levy, 1984; Schwegler & Jacob, 1975; Levy, 1990; Uijtdehaage & Thayer, 2000).

Parasympathetic Branch of the Autonomic Nervous System

Porges has succinctly summarized a rationale for examining parasympathetic factors in the stress response.

> The neglect of these (*parasympathetic*) concepts and an emphasis on the global construct of *arousal* still abide within the sub-disciplines of psychology, psychiatry and physiology. This outdated view of arousal may restrict an understanding of how the autonomic nervous system interfaces with the environment and the contributions of the autonomic nervous system to psychological and behavioral processes. (Porges, 1995b, p. 302)

Because the SNS has not been sufficient to explain many stress complaints, some physiologists have turned their attention to the PNS (Porges, 1995a, 1995b, 1997; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012), and especially the 10th cranial nerve, the vagus nerve. The vagus presents a *yang* to the *yin* of the SNS for most target organs. The SNS accelerates the heart and increases stroke volume, whereas the PNS brakes the heart. The SNS bronchodilates, the vagus bronchoconstricts. Although this antagonistic relationship is roughly correct, in actuality the interactions between the systems are quite complex and nonlinear (Cacioppo, Uchino, & Berntson, 1994). For our purposes, however, it is useful to conceive of the vagal system as a brake that withdraws when the organism is in any situation that might call for increased attention, defensiveness, premobilization, and so forth. From an evolutionary point of view, it would make sense that the self-maintaining functions of the PNS be "put on hold" at the hint of danger.

Fluctuations in HR or interbeat interval (IBI) with inspiration and expiration often are used as an index of cardiovagal activity (see Lehrer, Chapter 10, this volume). The fluctuations can be divided into three clusters: (1) HF, based on oscillations between 0.15 Hz and 0.4 Hz (almost totally produced by parasympathetic efferents), (2) LF, reflecting baroreceptor feedback to the sinoatrial node based on blood pressure fluctuations (reflecting both sympathetic and parasympathetic pathways), and (3) very low frequency (VLF), probably reflecting vascular or temperature rhythms (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Pagani & Malliani, 2000). In this way, vagal withdrawal can be observed. Especially when prolonged, vagal withdrawal could be an active component of the mediation between symptoms and emotional factors.

Porges, nicely summarized by McEwen (2002), has written extensively about this topic and proposes a theory called the polyvagal theory, based on an evolutionary perspective. It proposes that for everyday human interactions, the PNS control of the ANS is dominant and regulates complex human interpersonal emotions (McEwen, 2002). Porges has labeled this the *vagal social engagement system*. If valid, this theory would have major consequences for stress management. The theory posits that humans have evolved to have three neural circuits involving the autonomic nervous system: immobilization, mobilization, and social communication/engagement. This last system involves myelinated vagal fibers originating in the nucleus ambiguus of the brainstem that control facial expressions, vocalizations, and listening. This system evolved to enable social bonding and attachment to be the foundation of effective human functioning. He believes, for example, that autism illustrates the limits of human function without this developmental stage (Porges, 2011).

Others (e.g., Grossman & Kollai, 1993) have criticized this perspective and have argued that "vagal tone" is a more complex phenomenon, only partially represented by respiratory sinus arrhythmia (RSA). However, researchers on both sides of the issue would agree that the PNS plays a crucial role in stress and stress management.

The implications are that by conceptualizing "stress" as primarily a vagal withdrawal phenomenon, we will shift our interventions and treatment models dramatically. For some disorders (i.e., IBS or recurrent abdominal pain [RAP]) or pervasive anxiety, this may provide a powerful tool for intervention in and of itself.

Respiratory Parameters as Potential Mediators

Another physiological system that has the potential to mediate stress-related symptoms is the respiratory system—more accurately, the acid–base regulation that occurs during relaxed breathing. This topic is covered extensively elsewhere (Chaitow, Bradley, & Gilbert, 2002; Fried, 1987; Fried, Fox, & Carlton, 1990; Gevirtz, Lehrer, & Schwartz, 2016), but here we can note that subtle versions (e.g., sighing) of hyperventilation (producing more tidal flow than necessary to preserve the acid–base balance) can produce symptoms that are often characterized as stress related (e.g., dizziness, palpitations, dyspnea, panic, chest pain, anxiety). Therefore, in our search for links between "mind and body," respiration is often a very good place to start. To do this, one must carefully observe breathing patterns, measure end-tidal carbon dioxide ($ETCO_2$), and note symptom clusters (Nijmegen Questionnaire; van Dixhoorn & Folgering, 2015). No one measure is sufficient, but Nijmegen scores above 22, $ETCO_2$ below 32 mm HG, rapid respiration, and thoracic breathing, in combination, indicate that respiratory-based alkalosis is a prime candidate for symptoms such as those listed previously (van Dixhoorn & Duivenvoorden, 1985). A 2014 study (Davies & Craske, 2014) measured $ETCO_2$ in a group of patients with mixed anxiety who were being treated with one of two evidence-based therapies. Baseline $ETCO_2$ predicted outcome in both groups such that those patients with low $ETCO_2$ had poorer outcomes on anxiety measures. For a subset of these patients, respiratory factors probably play a strong role in symptom permanence and resistance to "talk therapies." Tolin, Billingsley, Hallion, and Diefenbach (2017) recently assessed the predictability of baseline $ETCO_2$ on dropout rate and found that a 5-mm Hg increase in $ETCO_2$ (e.g., 30 mm Hg to 35 mm Hg) predicted a threefold decrease in dropout rate with ethnicity and benzodiazepam use held constant. For some symptoms, simply correcting the overbreathing is quite effective (DeGuire et al., 1996; DeGuire et al., 1992). More often, respiratory factors play a contributory role, along with other mediational systems. Recently, commercial products that feed back $ETCO_2$ and promote slower breath rates have been shown to dramatically improve panic symptoms even at the 1-year follow-up (Meuret et al., 2008; Tolin et al., 2017).

For a more extensive discussion of respiratory factors in stress and stress management, see Meuret and Ritz (Chapter 11), van Dixhoorn (Chapter 12), and Telles, Kala, Gupta, and Balkrishna (Chapter 16) in this volume.

HPA Axis and the Sympathetic Adrenal Catecholamine System

Another potential pathway for mediation of psychological factors to physical states is based on endocrine responses to stress. The HPA axis functions as a systemic energy producer and anti-inflammatory system. When a stressor is detected by cortical and limbic brain systems, the hypothalamus can stimulate the pituitary to secrete adrenocorticotropic hormone (ACTH), which is picked up by adrenal receptor sites. The adrenal gland then secretes cortisol and other chemicals that produce effects in the brain and throughout the body. This is a relatively slow response, but it can have devastating effects if prolonged (McEwen, 2002). Similarly, the SNS stimulates the adrenal medulla to secrete epinephrine and norepinephrine (together called *catecholamines*), which reinforce SNS activation, especially to the cardiovascular system. Measurement of these "stress hormones" is commonplace in stress research. Thus these endocrine-based systems can also be considered as candidates for mediation. Modern analytic systems have brought the cost and invasiveness of these measures down, so clinicians may soon be able to use salivary cortisol (as one example) in clinical practice. Although the HPA and sympathoadrenal systems are quite complex, and although many methodological issues remain, reductions or increases in stress are usually reflected in comparable cortisol levels.

The preceding sections are meant to convey highlights of the current knowledge of autonomic, respiratory, and endocrine systems as they might apply to stress. This is, of course, a cursory description, and the reader is urged to seek other sources to expand his or her knowledge base. For the standard biofeedback modalities, an excellent source is Schwartz and Andrasik (2016). McEwen's (2002) book on stress is intended for the educated lay public but contains accurate and up-to-date material.

TREATMENT MANUAL

Based on the preceding, I now present a biofeedback-based stress management manual. The model is based on a number of basic principles. This particular model is especially relevant to disorders that carry a stigma as being "psychosomatic," "somatoform," "neurotic," or "hysterical"—such as IBS; many anxiety disorders, but especially panic and generalized anxiety disorder; chronic pain without obvious pathology; and similar complaints.

Before the Client Comes: The Trojan Horse Principle

The Trojan horse principle (Wickramasekera, 1994, 1995; Wickramasekera & Price, 1997) is based on the famous Greek myth related by Homer in the *Iliad,* in which the Greeks offered a hollow giant horse statue to Troy but filled it with invading troops. Here the Trojan horse is biofeedback, which appears to carry very little stigma or association with mental health procedures. When the clinician gives the client referral sources that emphasize the biomedical nature of the treatment, the client is less likely to think that the clinician believes the complaint is "all in my head." Thus the first principle is "medicalize, don't mentalize." Start treatment with a model that is as medical as possible and work slowly up to psychological or emotional factors over time.

Session 1: Symptoms

Take an elaborate oral history of symptoms, making sure to cover the *what, when, where,* and *how* of the complaint. Repeat back the history from your notes to obtain confirmation of accuracy. For example, if the complaint is headache, write a detailed description of the time course, the changes in severity, the precipitants, the attempts at self-treatment, and so forth. Symptom checklists can be used, but a face-to-face history is preferable. The Nijmegen Scale can be filled out with the client to clarify hyperventilation (HV) problems. The purpose of this procedure is twofold: to emphasize the legitimate nature of the symptoms and to begin building the mediational model, as described subsequently.

Session 2: Psychophysiological Stress Profile

After a clear picture of the symptom pattern is completed (including any questionnaires such as the Nijmegen), the clinician completes a psychophysiological stress profile (PSP). This procedure utilizes as many of the following modalities as possible: forehead electromyogram (EMG), skin conductance, respiration pattern and rate, $ETCO_2$, HR (EKG if possible), heart rate variability (HRV) measures (see Lehrer, Chapter 10, this volume), pulse amplitude, and finger temperature. A simple model involves a 5-minute baseline, a mental arithmetic or mild exercise stressor, and a recovery period (2–5 minutes). Longer baselines are desirable, but often impractical. Additional stressors are often added, such as personally relevant images. Information obtained from the PSP should be fed back to the client as the beginning of the explanation for the mediational model. We prefer to get a complete picture of the ANS by collecting at least 5 minutes of HRV data and then analyzing time and frequency domain measures (see Lehrer, Chapter 10, this volume).

If, for example, the client presents with a profile of global sympathetic dominance (high SC, cold hands, low HRV, and high facial muscle tension), the clinician might start with more traditional relaxation interventions (progressive muscle relaxation, autogenic training, etc.). On the other hand, the client might present with a profile of normal SNS indicators but low HRV, perhaps indicating predominant vagal withdrawal. The take-home message here is to customize the treatment to the ANS profile and use this information as a psychoeducational tool.

Psychological or Environmental Factors

Finally, the clinician should attempt to assess environmental or ecological factors that might be contributing to the complaint. This should be done as casually as possible so as to keep the medical focus. It can be done by asking questions about the home and work environment, about any unusual recent events, and so forth. It is critical to be listening for hints as to where the critical path will be.

At this juncture, a preliminary mediational model is constructed. Figure 6.1 shows a generic version of a model. As can be seen, the model shows how physical symptoms develop from physiological mediators and psychological and/or emotional factors. The "hysteria" pathway should be saved for frank hypochondriasis or symptom phobia or, in rare cases, true hysteria, such as blindness, paralysis, and so forth. For most problems, the other paths should be descriptive.

Some Common Psychophysiological Configurations

A wide variety of combinations of physiological profiles exists. I now present some common themes that might appear. However, one must be prepared to individualize the profile for each client.

PNS Dominance I: An Exaggerated Freeze Response

In this extreme configuration, the behavior and physiology are consistent with an organism's exhibiting a shutdown or immobilization response. The behavior is withdrawal or lack of engagement, facial rigidity, passive avoidance, and unexpressive voice production. The physiology is characterized by high, flat HR patterns consistent with the unmyelinated vagus taking over. This may occur without significant SNS involvement. Treatment

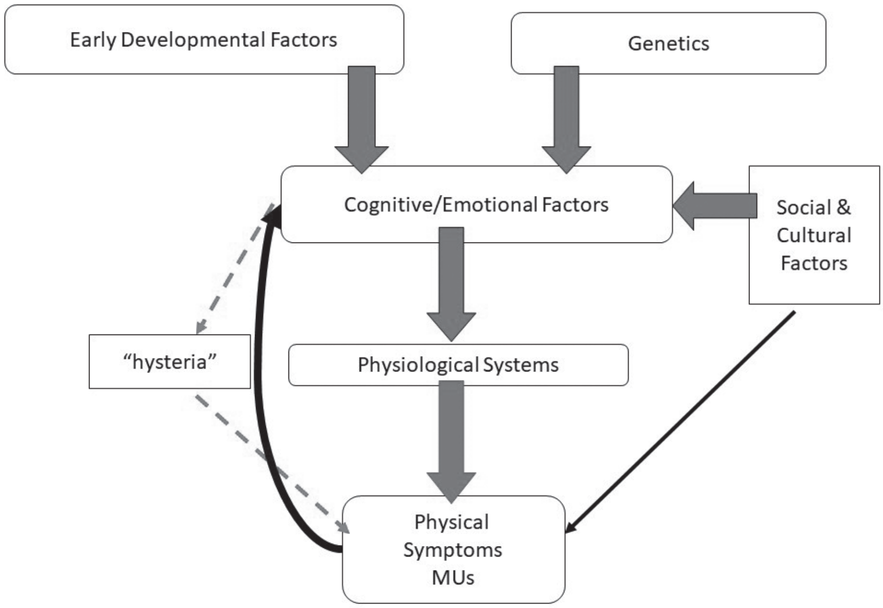


FIGURE 6.1. A generic mediational model.

strategies are complex and should build on the empirical work in trauma (Gevirtz, 2017; Gold, 2017).

PNS Dominance II: Parasympathetic Rebound Response

Another variation that may be harder to spot is presumed to be involved in such disorders as asthma, vasovagal reactions, and perhaps some gastrointestinal problems. It is characterized by a strong parasympathetic response after a strong sympathetic surge. It can be seen when strong RSA occurs after a stressor (presumably a sympathetic surge). Careful observation and measurement may be needed to catch this response, but in cases in which the symptoms are bronchoconstriction, fainting, or constipation, this pattern should be considered (Lehrer, Feldman, Giardino, Song, & Schmaling, 2002).

SNS Dominance

The most obvious configuration (which dominated stress physiology for decades) is one in which the SNS dominates. The organism is in a fight-or-flight mode, and thus HR is elevated, the HR spectrum is solely in the LF bands, SC is elevated, hands are cold, respiration is rapid and thoracic, some HV may be present (low $ETCO_2$), and facial muscle activity is high. During the stress, the levels are even higher, and they do not come down in recovery. The path model is obvious in that the client is in a "driven" state in which, over time, the autonomic and endocrine levels will cause a breakdown in some vulnerable organ system. You might think that this is a common configuration, but it is not. In fact, we rarely see a pattern this obvious. Almost any cultivated low-arousal treatment modality should be helpful (Jacobson's progressive muscle relaxation, autogenic training, breathing retraining, EMG reduction, hand temperature increases, etc.).

PNS Dominance III: Vagal Withdrawal

Much more common is a configuration that is not nearly as obvious. Some traditional SNS pathways may be activated, but no consistent pattern exists. Instead, the profile is characterized by low HF–HR spectral data and episodes of HR that rise and become more monotonic or flat. This is important to notice, as it will greatly affect the way the clinician presents the mediational model to the client. Furthermore, it will guide the discussion toward subtler types of stress. One disorder usually presents in this way: generalized anxiety disorder (GAD). This is an anxiety disorder characterized by states of constant anxiety, inability to relax, and a general sense of dread. Several studies (Ballenger et al., 2001; Borkovec & Costello, 1993; Nutt, 2001; Nutt, Ballenger, Sheehan, & Wittchen, 2002; Thayer, Friedman, & Borkovec, 1996; Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000) have shown that patients with GAD have truncated vagal tone. Levine, Fleming, Piedmont, Cain, and Chen (2016) concluded:

> The results support the notion that GAD is associated with vagal withdrawal during active bouts of idiographic worry and imagery, and question the assumption that GAD is associated with low resting vagal tone. In light of polyvagal theory, these findings provide additional support for the presence of emotion regulation deficits in GAD, and identify specific ANS processes that underlie GAD. (p. 207)

Yet the patient and clinician usually conceive of the problem as SNS overdrive. McLeod and colleagues (McLeod, Hoehn-Saric, Porges, Kowalski, & Clark, 2000; McLeod, Hoehn-Saric, Porges, & Zimmerli, 1992) have shown that imipramine's antianxiety effect in GAD is moderated by the degree to which anticholinergic metabolites reduce cardiac vagal tone. The profile guides treatment and becomes a form of cognitive therapy in itself. Treatment strategies are discussed later.

Respiratory Configuration

Sometimes the respiratory factors are so strong that they trump the ANS–PNS profiles. This would occur when a Nijmegen is high (over 30), $ETCO_2$ is low (under 30), respiration is rapid (> 20 breaths/minute), and the breathing pattern is predominantly thoracic. In these cases, breathing retraining or its equivalent should take precedence. This pattern can accompany many disorders and also can be secondary to severe chronic pain.

Vascular Configuration

A pattern of cold hands, fast HR, and low RSA may occur without other signs of SNS activation. Blood pressure should be checked. A pattern of low pulse amplitude may indicate vascular "clamping," with cold hands that are difficult to warm.

Mixed Configuration

Many other combinations are possible. In fact, most clients will have mixed presentations. In these cases, the clinician should try to build a model that will be credible to the client. For example, a client may present with warm hands and normal HR but with poor recovery and somewhat diminished RSA. This pattern may not present an exclusive path model, but there is sufficient patterning to justify an explanation and a treatment plan.

Again, the change in attribution from vague psychological constructs to a "scientific" or medical type of explanation allows a fast start for cognitive-type therapies.

Other disorders, such as asthma and hypertension, are probably better characterized within an operant model, in which the treatment intervention aims at correcting a dysfunctional homeostatic loop but less stigma or mental focus is associated with the disorder (see Lehrer, Chapter 10, this volume). Educational materials are available on a disorder-by-disorder basis. For example, online information can easily be found for IBS (*www.ibsgroup.org*), fibromyalgia (National Fibromyalgia Association, *www.fmaware.org*), chronic fatigue syndrome (CFS), and anxiety disorders (Anxiety Disorders Association of America, *www.adaa.org*). Evidence for the psychoeducational approach can be found in the literature for brief interventions for many of the disorders listed.

Sessions 3–5: Biofeedback Training

Once the educational nature of the model is communicated and accepted by the client, intervention may begin. It is usually best to start with a psychophysiological intervention such as biofeedback. I explain that we are intervening at the path between physiology and symptoms to try to break the cycle. It is preferable to use a combination of (HRV) training (see Lehrer, Chapter 10, this volume), facial EMG feedback, and finger temperature as an indicator (watching for warming hands). Clients can usually master relaxing jaw, forehead, and other facial muscles fairly quickly. The rationale for this technique is based on the elaborate afferent network involved in the muscles used in emotional expression (Porges, 1995b). See McGuigan and Lehrer, Chapter 7, this volume, for a description of muscle relaxation therapy. Clients must master voluntary relaxation of these muscles to move on to the HRV training. Lehrer (Chapter 10, this volume; Lehrer, Vaschillo, & Vaschillo, 2000) presents a complete manual for HRV biofeedback training, and Lehrer and Gevirtz (2014) try to elucidate mechanisms. After initial mastery, the client must demonstrate a resonance peak without feedback.

Final Sessions

During subsequent sessions, work can proceed up the pathway to tackle emotional problems and dysfunctional cognitions. Most patients can benefit from five to eight sessions, depending on how quickly they master the biofeedback skills and how much cognitive-behavioral type therapy is loaded on toward the end.

The most studied psychological intervention for most of the disorders I have mentioned is cognitive-behavioral therapy (CBT). A recent evolution and revision of CBT principles that is a very good fit with mediational models is acceptance and commitment therapy (ACT), introduced by Hayes, Strosahl, and Wilson (1999). To get to the more psychological aspects of treatment, one must first establish a credible physiological pathway and begin some training in a modality consistent with the model. For example, for GAD, once I have established that low vagal cardiac control is a likely mediator, I would initiate resonant frequency training (RFT; Gevirtz & Lehrer, 2003) with the stated purpose of restoring some vagal control. This might take five or six sessions of mostly biofeedback training, with only a hint of CBT or ACT present. As the symptoms diminish, the time is right to investigate the typical sources of irrational worry, of intolerance of uncertainty, of the usefulness of worry, and so forth. Similarly, many of the ACT strategies ("watching the parade," "deliteralizing language") are appropriate here. As another example, for chronic neck pain, the first stages would deal with elaborate psychoeducational explanations of how trigger points work, followed by RFT and/or surface electromyography (sEMG) feedback, followed by an exploration of what environmental triggers are driving the sympathetically mediated trigger points and how to reduce the activation time to sub–pain threshold levels. Again, CBT or ACT tools will be an obvious fit at this point.

As mentioned earlier, for some disorders, just the attribution shift and a few self-regulatory skills will be sufficient. For IBS, for example, a reformulation of brain–gut interconnection, together with RFT training and homework, often produces very rapid symptom reduction (especially when the problem is less chronic). It has been found (DeGuire et al., 1996; DeGuire et al., 1992) that eight sessions of breathing training alone greatly reduced chest pain in patients with functional cardiac pain, with continued improvement over 3 years. On the other hand, fibromyalgia will most often require more work in shifting perceptions, in establishing a shift in sympathovagal balance, in lifestyle changes (sleep, exercise, breathing, etc.), and in cognitive restructuring. Even here, however, the patients often label their treatment as "biofeedback" and tend to really value the objective physiological changes they see week to week.

INTERVENTIONS

Due to space limitations, treatment protocols for every stress-related disorder cannot be laid out here. Instead, I highlight ways in which the preceding presentations can be used for some disorders in various categories.

The model works very well for disorders that present with medical symptoms but that have a psychophysiological etiology. These are often labeled *somatoform,* or "medically unexplained," symptoms. Patients often pick up on the disapproval or helplessness of medical personnel with regard to these disorders. For this reason, the mediational model with an emphasis on biofeedback is often seen as a positive alternative to either traditional medical treatment (surgery or medications) or traditional psychotherapy.

IBS or Recurrent Abdominal Pain

IBS is the most common gastrointestinal disorder in primary care settings. It occurs in 11–20% of the US population (Drossman et al., 1993) and accounts for 12–19.5% of primary care visits (Longstreth & Wolde-Tsadik, 1993). It is estimated to cost over $30 billion in direct and indirect costs. In light of these numbers, a keen interest in managed care medical groups has developed in the quest to find a cost-effective way of treating these patients. Currently, most gastroenterologists accept that psychological factors such as stress play a key role in IBS. Therefore, presentation of the mediational model or of generic biofeedback is generally accepted (Brun & Kuo, 2010; Gikas & Triantafyllidis, 2014; Klass, Rake, & Williams, 1994; Hyams, Burke, Davis, Rzepski, & Andrulonis, 1996; Abell et al., 2006; Heetun & Quigley, 2012; Horowitz, Su, Rayner, & Jones, 2001) within our framework; the main clinical task is to get the client to see his or her symptoms (usually abdominal pain, diarrhea, etc.) from the psychophysiological point of view. This means showing some pictures of the enteric nervous system and a simple explanation of how stress, worry, and anxiety, even at low levels, could disrupt the way the gut processes food though the gastrointestinal track. In addition, several groups (Mayer, 1999, 2000; Mayer, Chang, & Lembo, 1998; Mayer, Derbyshire, & Naliboff, 2000; Mayer, Naliboff, & Chang, 2001; Mayer, Naliboff, Chang, & Coutinho, 2001; Naliboff, Chang, Munakata, & Mayer, 2000) have shown that this or other disruptions can lead to a growing visceral hypersensitivity. There is certainly a body of literature that supports ANS involvement in IBS and RAP (Gupta, Sheffield, & Verne, 2002; Iovino, Azpiroz, Domingo, & Malagelada, 1995; Waring et al., 2004; Jepson & Gevirtz, 2001). In some recent work of ours (Jepson & Gevirtz, 2001), it is looking as though the physiological mediator might be prolonged vagal or parasympathetic withdrawal rather than excessive sympathetic drive. This type of information is used to convince the patient that his or her very physical symptoms could be related to long-term psychological states. Once that reattribution is made, the interventions follow naturally.

Psychophysiological Interventions

Biofeedback-based relaxation training, HRV biofeedback, mindfulness meditation, and other techniques all make sense within this context. I prefer the HRV biofeedback (see Lehrer, Chapter 10, this volume) because it works to strengthen the autonomic reflexes that may be broken down with long-term stress or worry. The client is instructed to practice at least 10 minutes per day and to use the technique to interrupt prolonged rumination, anxiety, or worry. With children, we have found that this intervention alone is usually sufficient to break the cycle of pain, stress, worry, more pain, and so forth. We presume, but have not yet shown, that the HRV practice restores enough vagal regulation to raise the threshold for abdominal pain. We are currently assessing a noninvasive gastrogram to test this hypothesis (Gharibans et al., 2018).

CBT-Type Interventions

CBT, dialectical behavior therapy (DBT; Clarkin, Levy, Lenzenweger, & Kernberg, 2004; Linehan, 1995; Linehan, Heard, & Armstrong, 1993; Linehan et al., 1999), and ACT (Hayes, 2005; Hayes et al., 1999) are all applicable to IBS. In this case, though, the mediational model creates a nonthreatening and plausible lead-in to the modification of thoughts, attitudes, values, or emotions. Once a client sees that prolonged emotional states are contributing to the symptoms, it is not hard to motivate him or her to consider a cognitive-type intervention.

Case Example

James was a 38-year-old professional in a technical field. He had a PhD in computer sciences and was a linear thinker. He came to treatment with abdominal pain of moderate to severe intensity after exhausting a wide variety of traditional medical paths (in this field, one is sometimes tempted to put up a "Next Stop, Lourdes" sign on the door). He was skeptical about this "psychological stuff" and denied any unusual stress. His stress profile was unremarkable except that his RSA (peak valley HR differences during ordinary breathing in the range of 12–20 breaths per minute and during 6 breaths-per-minute breathing) was low (4 beats/minute) for his age, and he had a prolonged peak in the VLF range of the HRV spectral analyses (see Lehrer, Chapter 10, this volume). I went through the results with him in great detail, showing slides of the ANS and other materials mentioned earlier. He was fascinated and asked many questions.

This case illustrates the Trojan horse approach. By medicalizing his symptoms, I removed the stigma associated with this "unexplained" condition and gave him a reasonable explanation without labeling it an anxiety disorder.

In the second session, we reviewed the hypothesis that his problems might be due to prolonged vagal withdrawal leading to gastrointestinal hyperalgesia. Again, using slides and poring over the physiological traces, he became convinced that this model made sense and began supplying information that was supportive. He was, indeed, a "worrier," spending many early mornings sleeplessly going over various concerns or plans in bed. When not distracted, he often found himself with a wandering mind and run-on thinking. Of course, his main worry was his health, so when a reasonable explanation appeared, he started feeling better immediately. We found his resonant frequency for HRV, set up a home practice schedule, had him buy an inexpensive temperature biofeedback unit as an indicator, and sent him off for 2 weeks of practice. In the next session, we refined his breathing/RSA rate and focused on some ACT principles that seemed especially appropriate for his style of thinking (Harris, 2001; Hayes et al., 1999).

James reduced his symptom severity dramatically and managed to do so with few follow-up visits. At his 1-year follow-up, he was almost symptom-free, but when flares occurred, he would not catastrophize and could soon bring things back to normal. The same traits that made him quite resistant to traditional psychological interventions could be used to his advantage here. Linear thinking, suspicion of emotions, and fear of disclosing to a stranger were all easily used to help him manage his symptoms and, as a bonus, many other aspects of his life.

Fibromyalgia

Fibromyalgia (FM) may represent a somewhat different case, but a version of mediational modeling can also be used here. A case history illustrates how physiological data and biofeedback can be integrated into a mediational model.

Case Example

Beth was a 37-year-old attorney with three children and a husband who was also an attorney. She had been very active in all phases of her life prior to the FM symptoms. She woke up early for a vigorous run each day before work, drove the children to soccer, water polo, and gymnastics, was active with their school and in her church, and was trying to achieve partner status in her law firm. Her husband was supportive but immersed in his own successfully emerging career. All was well until she contracted a cold that turned into a serious case of bronchitis. After a 6-week recovery, she thought she should be ready to resume her active life, but she was exhausted, in pain throughout her body, sleeping poorly, and unable to exercise at all. Again, after an exhaustive medical search, she was desperate enough to try biofeedback. In this case, the educational intervention was aimed at convincing her that her nervous system, both central and ANS, could be responsible for the symptoms, even without an occult viral infection, an autoimmune disorder, or some other "real" disease. I drew on materials from the scientific literature and tried to illustrate to her that her ANS, CNS, and perhaps enteric nervous system were operating in an idiosyncratic manner, consistent with the theories of several researchers (Martinez-Lavin, 2001a, 2001b; Martinez-Lavin, Amigo, Coindreau, & Canoso, 2000; Martinez-Lavin & Hermosillo, 2000; Martinez-Lavin et al., 1997; Martinez-Lavin et al., 1998; Moldofsky, 1993, 1994). Within these frameworks FM is seen as (1) a chronobiological disorder (Moldofsky, 1994) in which a disordered 24-hour body clock causes disturbed sleep, which in turn causes diffuse pain; (2) a CNS substance pain processing disorder

(Russell, 1998, 2000) in which, due to physical or psychological trauma, the CNS gets stuck in a hyperalgesic or defensive stance with disturbance in a number of peptides and neurotransmitters; or (3) chronic sympathetic overdrive (Martinez-Lavin, 2001a, 2001b; Martinez-Lavin et al., 2000; Martinez-Lavin & Hermosillo, 2000; Martinez-Lavin et al., 1997; Martinez-Lavin et al., 1998), in which the CNS and ANS get stuck in a maladaptive high chronic level, flattening circadian rhythms, exhausting fight-or-flight mechanisms, and creating a subsequent HPA response. I used a number of graphs from the preceding material and compared them with Beth's psychophysiological profile. She, for example, had a predominant VLF wave in her HRV, a high LF:HF ratio (indicating sympathetic dominance), cold hands, and high SC levels. Her HR was high for a seasoned athlete, and she was a rapid, shallow breather with slightly low $ETCO_2$. A circadian rhythm of LF:HF ratio has been shown to be flat in patients with FM (Martinez-Lavin et al., 1998).

Over a few sessions, Beth became convinced that this explanation was at least reasonably correct, and she was willing to cooperate and practice the techniques and lifestyle changes that I suggested. In this case, that entailed my version of the SABRE protocol (Nixon, 1989; Nixon & Freeman, 1988):

- *Sleep:* sleep hygiene with a detailed sleep log
- *Arousal:* HRV biofeedback to restore autonomic reflexes and lower sympathetic arousal
- *Breathing:* breathing retraining
- *Rest:* activity management
- *Exercise:* slow, gentle, graded exercise, starting with gentle yoga and slowly working up to aerobic exercise (Jones, Adams, Winters-Stone, & Burckhardt, 2006).

After 4 months of working this system, Beth was about 85% recovered and enjoying a fairly normal life. She never could return to her premorbid stressful life, but she is certainly doing much better. She recently joined a master swim class and is swimming competitively 3 days per week (Hassett & Gevirtz, 2009).

Other Stigmatized Disorders

Other disorders that could be classified in this category are chronic muscle pain, noncardiac chest pain (NCCP), GAD, and some types of panic disorder. In each case, a mediational model can be created with varying degrees of empirical support. For muscle pain, our group (Gevirtz et al., 1996; Hubbard, 1996, 1998; Hubbard & Berkoff, 1993) has described a sympathetically mediated trigger point model for chronic pain. In another set of studies, DeGuire and colleagues (DeGuire et al., 1996; DeGuire et al., 1992) have shown that chronic respiratory factors such as HV may mediate symptoms of NCCP. Borkovec and Costello (1993) have postulated vagal withdrawal as a factor in GAD, and many research groups have emphasized the importance of breathing in panic disorder (Ley, 2005; Meuret, Ritz, Wilhelm, & Roth, 2005; Meuret, Wilhelm, Ritz, & Roth, 2003; Meuret, Wilhelm, & Roth, 2004; Roth, 2005; Roth, Wilhelm, & Trabert, 1998; Wilhelm, Gerlach, & Roth, 2001; Wilhelm, Gevirtz, & Roth, 2001; Wilhelm, Trabert, & Roth, 2001a, 2001b). As mentioned earlier (Ryan, 2001), an application of this model within a primary medical setting provided evidence for symptom reduction and cost savings (in FM, muscle pain, IBS, anxiety, and chest pain).

NEGATIVE SIDE EFFECTS

No specific side effects have been reported from this approach. As is mentioned by Lehrer (Chapter 10, this volume), a relaxation-induced anxiety and some HV are possible during the initial stages of the cultivated low arousal or HRV training. Lehrer provides some useful ideas on how to handle these problems. Otherwise, the general approach usually provides a useful framework for the patient. If, however, the patient remains passive and skeptical, this approach is unlikely to be beneficial.

SUMMARY

This chapter has presented a heuristic model that I and my colleagues have found useful in stress management and treatment of a variety of disorders found commonly in primary care and in stress management groups. By using the physiological mediator model as a foundation, the trainer or therapist can gain acceptance from traditionally skeptical audiences, can introduce skills for specific purposes, and can transition into well-established therapies seamlessly. With some practice and knowledge of the various disorders, this method can greatly enhance the therapist's fulfillment, as well as helping patients find relief from the suffering that dominates their lives.

REFERENCES

Abell, T. L., Bernstein, R. K., Cutts, T., Farrugia, G., Forster, J., Hasler, W. L., et al. (2006). Treatment of gastroparesis: A multidisciplinary clinical review. *Neurogastroenterology and Motility, 18*(4), 263–283.

Adeyemi, E. O., Desai, K. D., Towsey, M., & Ghista, D. (1999). Characterization of autonomic dysfunction in patients with irritable bowel syndrome by means of heart rate variability studies. *American Journal of Gastroenterology, 94*(3), 816–823.

Andrade, L., Walters, E. E., Gentil, V., & Laurenti, R. (2002). Prevalence of ICD-10 mental disorders in a catchment area in the city of Sao Paulo, Brazil. *Social Psychiatry and Psychiatric Epidemiology, 37*(7), 316–325.

Ballenger, J. C., Davidson, J. R., Lecrubier, Y., Nutt, D. J., Borkovec, T. D., Rickels, K., et al. (2001). Consensus statement on generalized anxiety disorder from the International Consensus Group on Depression and Anxiety. *Journal of Clinical Psychiatry, 62*(Suppl. 11), 53–58.

Biondi, M., & Picardi, A. (1999). Psychological stress and neuroendocrine function in humans: The last two decades of research. *Psychotherapy and Psychosomatics, 68*(3), 114–150.

Borkovec, T. D., & Costello, E. (1993). Efficacy of applied relaxation and cognitive-behavioral therapy in the treatment of generalized anxiety disorder. *Journal of Consulting and Clinical Psychology, 61*(4), 611–619.

Brun, R., & Kuo, B. (2010). Review: Functional dyspepsia. *Therapeutic Advances in Gastroenterology, 3,* 145–164.

Cacioppo, J. T., Uchino, B. N., & Berntson, G. G. (1994). Individual differences in the autonomic origins of heart rate reactivity: The psychometrics of respiratory sinus arrhythmia and preejection period. *Psychophysiology, 31*(4), 412–419.

Cannon, W. B. (1929). Organization for physiological homeostasis. *Physiological Reviews, 9,* 399–431.

Cassidy, K., Kotynia-English, R., Acres, J., Flicker, L., Lautenschlager, N. T., & Almeida, O. P. (2004). Association between lifestyle factors and mental health measures among community-dwelling older women. *Australian and New Zealand Journal of Psychiatry, 38*(11–12), 940–947.

Chaitow, L., Bradley, D., & Gilbert, C. (2002). *Multidisciplinary approaches to breathing pattern disorders.* London: Churchill Livingstone.

Clarkin, J. F., Levy, K. N., Lenzenweger, M. F., &

Kernberg, O. F. (2004). The Personality Disorders Institute/Borderline Personality Disorder Research Foundation randomized control trial for borderline personality disorder: Rationale, methods, and patient characteristics. *Journal of Personality Disorders, 18*(1), 52–72.

Cummings, N. A., & VandenBos, G. R. (1981). The twenty-year Kaiser Permanente experience with psychotherapy and medical utilization: Implications for national health policy and national health insurance. *Health Policy Quarterly, 1*(2), 159–175.

Davies, C. D., & Craske, M. G. (2014). Low baseline pCO_2 predicts poorer outcome from behavioral treatment: Evidence from a mixed anxiety disorders sample. *Psychiatry Research, 219*(2), 311–315.

DeGuire, S., Gevirtz, R., Hawkinson, D., & Dixon, K. (1996). Breathing retraining: A three-year follow-up study of treatment for hyperventilation syndrome and associated functional cardiac symptoms. *Biofeedback and Self-Regulation, 21*(2), 191–198.

DeGuire, S., Gevirtz, R., Kawahara, Y., & Maguire, W. (1992). Hyperventilation syndrome and the assessment of treatment for functional cardiac symptoms. *American Journal of Cardiology, 70*(6), 673–677.

den Boeft, M., Twisk, J. W., Terluin, B., Penninx, B. W., van Marwijk, H. W., Numans, M. E., et al. (2016). The association between medically unexplained physical symptoms and health care use over two years and the influence of depressive and anxiety disorders and personality traits: A longitudinal study. *BMC Health Services Research, 16*(1), 100.

Drossman, D. A., Creed, F. H., Olden, K. W., Svedlund, J., Toner, B. B., & Whitehead, W. E. (1999). Psychosocial aspects of the functional gastrointestinal disorders. *Gut, 45*(Suppl. 2), II25–II30.

Drossman, D. A., Li, Z., Andruzzi, E., Temple, R. D., Talley, N. J., Thompson, W. G., et al. (1993). U.S. householder survey of functional gastroenterological disorders: Prevalence, sociodemography, and health impact. *Digestive Disease Science, 38*(9), 1569–1580.

Fried, R. (1987). *The hyperventilation syndrome: Research and clinical treatment.* Baltimore: Johns Hopkins University Press.

Fried, R., Fox, M., & Carlton, R. (1990). Effect of diaphragmatic respiration with end-tidal CO_2 biofeedback on respiration, EEG, and seizure frequency in idiopathic epilepsy. *Annals of the New York Academy of Sciences, 602,* 67–96.

Gevirtz, R. N. (2013). The promise of heart rate variability biofeedback: Evidence based applications. *Biofeedback, 41*(3), 110–120.

Gevirtz, R. N. (2017). The central autonomic network in trauma etiology and treatment. In S. Gold (Ed.), *APA handbook of trauma psychology: Vol. 2. Trauma practice* (pp. 213–226). Washington, DC: American Psychological Association.

Gevirtz, R., Hubbard, D., & Harpin, E. (1996). Psychophysiologic treatment of chronic low back pain. *Professional Psychology: Research and Practice, 27*(6), 561–566.

Gevirtz, R., & Lehrer, P. (2003). Resonant frequency heart rate biofeedback. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (3rd ed., pp. 245–250). New York: Guilford Press.

Gevirtz, R. N., Lehrer, P., & Schwartz, M. S. (2016). Cardiorespiratory measurement and assessment in applied psychophysiology. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (4th ed., pp. 85–97). New York: Guilford Press.

Gevirtz, R. N., & Schwartz, M. S. (2003). The respiratory system. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (3rd ed., pp. 212–244). New York: Guilford Press.

Gharibans, A. A., Smarr, B. L., Kunkel, D. C., Kriegsfeld, L. J., Mousa, H. M., & Coleman, T. P. (2018). Artifact rejection methodology enables continuous, noninvasive measurement of gastric myoelectric activity in ambulatory subjects. *Scientific Reports, 8*(1), 5019.

Gikas, A., & Triantafyllidis, J. K. (2014). The role of primary care physicians in early diagnosis and treatment of chronic gastrointestinal diseases. *International Journal of General Medicine, 7,* 159–173.

Goessl, V. C., Curtiss, J. E., & Hofmann, S. G. (2017). The effect of heart rate variability biofeedback training on stress and anxiety: A meta-analysis. *Psychological Medicine, 47*(15), 2578–2586.

Gold, S. N. (Ed.). (2017). *APA handbook of trauma psychology* (Vols. 1 & 2). Washington, DC: American Psychological Association.

Grossman, P., & Kollai, M. (1993). Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within- and between-individual relations. *Psychophysiology, 30*(5), 486–495.

Gupta, V., Sheffield, D., & Verne, B. N. (2002). Evidence for autonomic dysregulation in the irritable bowel syndrome. *Digestive Disease Science, 47*(8), 1716–1722.

Guyton, A., & Hall, J. (1995). *Textbook of medical physiology* (9th ed.). Philadelphia: Saunders.

Haller, H., Cramer, H., Lauche, R., & Dobos, G. (2015). Somatoform disorders and medically unexplained symptoms in primary care: A systematic review and meta-analysis of prevalence. *Deutsches Ärzteblatt International, 112*(16), 279.

Harris, R. (2011). The happiness trap. Retrieved from *ReadHowYouWant.com*.

Hassett, A., & Gevirtz, R. N. (2009). Nonpharmacological treatment for fibromyalgia: Patient education, cognitive-behavioral therapy, relaxation techniques, and complementary medicine. *Rheumatic Disease Clinics of North America, 35*(2), 393–408.

Hayes, S. (2005). *Get out of your mind and into your life*. Oakland, CA: New Harbinger Press.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy*. New York: Guilford Press.

Heetun, Z. S., & Quigley, E. M. M. (2012). Gastroparesis and Parkinson's disease: A systematic review. *Parkinsonism and Related Disorders, 18,* 433–440.

Horowitz, M., Su, Y. C., Rayner, C. K., & Jones, K. L. (2001). Gastroparesis: Prevalence, clinical significance and treatment. *Canadian Journal of Gastroenterology, 15,* 805–813.

Hubbard, D. (1996). Chronic and recurrent muscle pain: Pathophysiology and treatment, a review of pharmacologic studies. *Journal of Musculoskeletal Pain, 4*(1–2), 123–143.

Hubbard, D. (1998). Persistent muscular pain: Approaches to relieving trigger points. *Journal of Musculoskeletal Medicine, 15*(5), 16–26.

Hubbard, D. R., & Berkoff, G. M. (1993). Myofascial trigger points show spontaneous needle EMG activity. *Spine, 18*(13), 1803–1807.

Humphreys, P. A., & Gevirtz, R. N. (2000). Treatment of recurrent abdominal pain: Components analysis of four treatment protocols. *Journal of Pediatric Gastroenterology and Nutrition, 31*(1), 47–51.

Hyams, J. S., Burke, G., Davis, P. M., Rzepski, B., & Andrulonis, P. A. (1996). Abdominal pain and irritable bowel syndrome in adolescents: A community-based study. *Journal of Pediatrics, 129,* 220–226.

Iovino, P., Azpiroz, F., Domingo, E., & Malagelada, J. R. (1995). The sympathetic nervous system modulates perception and reflex responses to gut. *Gastroenterology, 108*(3), 680–686.

Issakidis, C., & Andrews, G. (2003). Rationing of health care: Clinical decision making in an outpatient clinic for anxiety disorders. *Journal of Anxiety Disorders, 17*(1), 59–74.

Jepson, N., & Gevirtz, R. N. (2001). Cognitive-behavioral and biofeedback group treatments for irritable bowel syndrome. *Applied Psychophysiology and Biofeedback, 26,* 237.

Jones, K. D., Adams, D., Winters-Stone, K., & Burckhardt, C. S. (2006). A comprehensive review of 46 exercise treatment studies in fibromyalgia (1988–2005). *Health and Quality of Life Outcomes, 4*(1), 67.

Katon, W. (1996). Panic disorder: Relationship to high medical utilization, unexplained physical symptoms, and medical costs. *Journal of Clinical Psychiatry, 57*(Suppl. 10), 11–18.

Katon, W., Hart, R., & Montano, B. (1997). The effect of panic disorder in the managed care setting. *Managed Care Interface, 10*(11), 88–94, 98.

Katon, W. J., Von Korff, M., & Lin, E. (1992). Panic disorder: Relationship to high medical utilization. *American Journal of Medicine, 92*(1A), 7S–11S.

Klass, H. J., Rake, M. O., & Williams, J. G. (1994). Prevalence of dyspepsia: The epidemiology of overlapping symptoms. *Postgraduate Medical Journal, 70,* 154–161.

Lehrer, P., Feldman, J., Giardino, N., Song, H. S., & Schmaling, K. (2002). Psychological aspects of asthma. *Journal of Consulting and Clinical Psychology, 70*(3), 691–711.

Lehrer, P. M., & Gevirtz, R. N. (2014). Heart rate variability biofeedback: How and why does it work? *Frontiers in Psychology, 5,* 756.

Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. *Applied Psychophysiology and Biofeedback, 25*(3), 177–191.

Levine, J. C., Fleming, R., Piedmont, J. I., Cain, S. M., & Chen, W. J. (2016). Heart rate variability and generalized anxiety disorder during laboratory-induced worry and aversive imagery. *Journal of Affective Disorders, 205,* 207–215.

Levy, M. N. (1990). Autonomic interactions in cardiac control. *Annals of the New York Academy of Sciences, 601*(1), 209–221.

Ley, R. (2005). Blood, breath, fears redux, and panic attacks: Comment on Roth, Wilhelm, and Pettit (2005). *Psychological Bulletin, 131*(2), 193–198.

Linehan, M. M. (1995). Combining pharmacotherapy with psychotherapy for substance abusers with borderline personality disorder:

Strategies for enhancing compliance. *NIDA Research Monograph, 150,* 129–142.
Linehan, M. M., Heard, H. L., & Armstrong, H. E. (1993). Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. *Archives of General Psychiatry, 50*(12), 971–974.
Linehan, M. M., Schmidt, H., III, Dimeff, L. A., Craft, J. C., Kanter, J., & Comtois, K. A. (1999). Dialectical behavior therapy for patients with borderline personality disorder and drug dependence. *American Journal on Addictions, 8*(4), 279–292.
Longstreth, G. G., & Wolde-Tsadik, G. (1993). Irritable bowel-type symptoms in HMO examinees. *Digestive Diseases and Sciences, 38*(9), 1581–1589.
Martinez-Lavin, M. (2001a). Is fibromyalgia a generalized reflex sympathetic dystrophy? *Clinical and Experimental Rheumatology, 19*(1), 1–3.
Martinez-Lavin, M. (2001b). Overlap of fibromyalgia with other medical conditions. *Current Pain and Headache Reports, 5*(4), 347–350.
Martinez-Lavin, M., Amigo, M. C., Coindreau, J., & Canoso, J. (2000). Fibromyalgia in Frida Kahlo's life and art. *Arthritis and Rheumatism, 43*(3), 708–709.
Martinez-Lavin, M., & Hermosillo, A. G. (2000). Autonomic nervous system dysfunction may explain the multisystem features of fibromyalgia. *Seminars in Arthritis and Rheumatism, 29*(4), 197–199.
Martinez-Lavin, M., Hermosillo, A. G., Mendoza, C., Ortiz, R., Cajigas, J. C., Pineda, C., et al. (1997). Orthostatic sympathetic derangement in subjects with fibromyalgia. *Journal of Rheumatology, 24*(4), 714–718.
Martinez-Lavin, M., Hermosillo, A. G., Rosas, M., & Soto, M. E. (1998). Circadian studies of autonomic nervous balance in patients with fibromyalgia: A heart rate variability analysis. *Arthritis and Rheumatism, 41*(11), 1966–1971.
Mayer, E. A. (1999). Emerging disease model for functional gastrointestinal disorders. *American Journal of Medicine, 107*(5A), 12S–19S.
Mayer, E. A. (2000). Spinal and supraspinal modulation of visceral sensation. *Gut, 47*(Suppl. 4), iv69–iv72.
Mayer, E. A., Chang, L., & Lembo, T. (1998). Brain–gut interactions: Implications for newer therapy. *European Journal of Surgery, 582*(Suppl.), 50–55.
Mayer, E. A., Derbyshire, S., & Naliboff, B. D. (2000). Cerebral activation in irritable bowel syndrome. *Gastroenterology, 119*(5), 1418–1420.
Mayer, E. A., Naliboff, B. D., & Chang, L. (2001). Basic pathophysiologic mechanisms in irritable bowel syndrome. *Digestive Diseases, 19*(3), 212–218.
Mayer, E. A., Naliboff, B. D., Chang, L., & Coutinho, S. V. (2001). Stress and the gastrointestinal tract: V. Stress and irritable bowel syndrome. *American Journal of Physiology: Gastrointestinal and Liver Physiology, 280*(4), G519–G524.
McEwen, B. L. (with Lasley, E. N.). (2002). *The end of stress as we know it.* Washington, DC: Joseph Henry Press.
McLeod, D. R., Hoehn-Saric, R., Porges, S. W., Kowalski, P. A., & Clark, C. M. (2000). Therapeutic effects of imipramine are counteracted by its metabolite, desipramine, in patients with generalized anxiety disorder. *Journal of Clinical Psychopharmacology, 20*(6), 615–621.
McLeod, D. R., Hoehn-Saric, R., Porges, S. W., & Zimmerli, W. D. (1992). Effects of alprazolam and imipramine on parasympathetic cardiac control in patients with generalized anxiety disorder. *Psychopharmacology, 107*(4), 535–540.
McNulty, W. H., Gevirtz, R. N., Hubbard, D. R., & Berkoff, G. M. (1994). Needle electromyographic evaluation of trigger point response to a psychological stressor. *Psychophysiology, 31*(3), 313–316.
Meuret, A. E., Ritz, T., Wilhelm, F. H., & Roth, W. T. (2005). Voluntary hyperventilation in the treatment of panic disorder: Functions of hyperventilation, their implications for breathing training, and recommendations for standardization. *Clinical Psychology Review, 25*(3), 285–306.
Meuret, A. E., Wilhelm, F. H., Ritz, T., & Roth, W. T. (2003). Breathing training for treating panic disorder: Useful intervention or impediment? *Behavior Modification, 27*(5), 731–754.
Meuret, A. E., Wilhelm, F. H., Ritz, T., & Roth, W. T. (2008). Feedback of end-tidal pCO_2 as a therapeutic approach for panic disorder. *Journal of Psychiatric Research, 42*(7), 560–568.
Meuret, A. E., Wilhelm, F. H., & Roth, W. T. (2004). Respiratory feedback for treating panic disorder. *Journal of Clinical Psychology, 60*(2), 197–207.
Moldofsky, H. (1993). Fibromyalgia, sleep disorder and chronic fatigue syndrome. *Ciba Foundation Symposium, 173,* 262–271.

Moldofsky, H. (1994). Chronobiological influences on fibromyalgia syndrome: Theoretical and therapeutic implications. *Baillieres Clinical Rheumatology, 8*(4), 801–810.

Morrison, S. F. (2001). Differential control of sympathetic outflow. *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology, 281*(3), R683–R698.

Naliboff, B. D., Chang, L., Munakata, J., & Mayer, E. A. (2000). Towards an integrative model of irritable bowel syndrome. *Progress in Brain Research, 122,* 413–423.

Nixon, P. G. (1989). Human functions and the heart. In D. S. A. Cribb (Ed.), *Changing ideas in health care* (pp. 31–65). Chichester, UK: Wiley.

Nixon, P. G., & Freeman, L. J. (1988). The "think test": A further technique to elicit hyperventilation. *Journal of the Royal Society of Medicine, 81*(5), 277–279.

Nutt, D. J. (2001). Neurobiological mechanisms in generalized anxiety disorder. *Journal of Clinical Psychiatry, 62*(Suppl. 11), 22–27.

Nutt, D. J., Ballenger, J. C., Sheehan, D., & Wittchen, H. U. (2002). Generalized anxiety disorder: Comorbidity, comparative biology and treatment. *International Journal of Neuropsychopharmacology, 5*(4), 315–325.

Olshansky, B., Sabba, H. N., Hauptman, P. J., & Colucci, W. S. (2008). Parasympathetic nervous system and heart failure: Pathophysiology and implications for therapy. *Circulation, 118*(8), 863–871.

Pagani, M., & Malliani, A. (2000). Interpreting oscillations of muscle sympathetic nerve activity and heart rate variability. *Journal of Hypertension, 18*(12), 1709–1719.

Porges, S. W. (1995a). Cardiac vagal tone: A physiological index of stress. *Neuroscience and Biobehavioral Reviews, 19*(2), 225–233.

Porges, S. W. (1995b). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage: A polyvagal theory. *Psychophysiology, 32*(4), 301–318.

Porges, S. W. (1997). Emotion: An evolutionary by-product of the neural regulation of the autonomic nervous system. *Annals of the New York Academy of Sciences, 807,* 62–77.

Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation.* New York: Norton.

Roth, W. T. (2005). Physiological markers for anxiety: Panic disorder and phobias. *International Journal of Psychophysiology, 58*(2–3), 190–198.

Roth, W. T., Wilhelm, F. H., & Trabert, W. (1998). Autonomic instability during relaxation in panic disorder. *Psychiatry Research, 80*(2), 155–164.

Russell, I. J. (1998). Advances in fibromyalgia: Possible role for central neurochemicals. *American Journal of the Medical Sciences, 315*(6), 377–384.

Russell, I. J. (2000). Fibromyalgia syndrome. In S. Mense, D. Simons, & I. J. Russell (Eds.), *Muscle pain: Understanding its nature, diagnosis and treatment* (pp. 289–337). New York: Lippincott, Williams & Wilkins.

Ryan, M. G., & Gevirtz, R. N. (2001, March). *The effects of biofeedback on cost effectiveness in primary care settings.* Paper presented at the convention of the Association for Applied Psychophysiology and Biofeedback, Raleigh–Durham, NC.

Schwartz, M. S., & Andrasik, F. (Eds.). (2016). *Biofeedback: A practitioner's guide* (4th ed.). New York: Guilford Press.

Schwegler, M., & Jacob, R. (1975). Catecholamine antagonism of acetylcholine and dibutyryl guanosine 3',-5'-monophosphate in the mammalian ventricular myocardium. *Recent Advances in Studies on Cardiac Structure and Metabolism, 7,* 391–399.

Sharpe, M., & Carson, A. (2001). "Unexplained" somatic symptoms, functional syndromes, and somatization: Do we need a paradigm shift? *Annals of Internal Medicine, 134*(9, Pt. 2), 926–930.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal, 17*(3), 354–381.

Thayer, J. F., Ahs, F., Fredrikson, M., Sollers, J. J., III, & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience and Biobehavioral Reviews, 36*(2), 747–756.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39*(4), 255–266.

Thayer, J. F., Friedman, B. H., Borkovec, T. D., Johnsen, B. H., & Molina, S. (2000). Phasic heart period reactions to cued threat and nonthreat stimuli in generalized anxiety disorder. *Psychophysiology, 37*(3), 361–368.

Tolin, D. F., Billingsley, A. L., Hallion, L. S., & Diefenbach, G. J. (2017). Low pre-treatment end-tidal CO_2 predicts dropout from cognitive-behavioral therapy for anxiety and related disorders. *Behaviour Research and Therapy, 90,* 32–40.

Uijtdehaage, S. H., & Thayer, J. F. (2000). Accentuated antagonism in the control of human heart rate. *Clinical Autonomic Research, 10*(3), 107–110.

van Dixhoorn, J., & Duivenvoorden, H. J. (1985). Efficacy of Nijmegen Questionnaire in recognition of the hyperventilation syndrome. *Journal of Psychosomatic Research, 29*(2), 199–206.

van Dixhoorn, J., & Folgering, H. (2015). The Nijmegen Questionnaire and dysfunctional breathing. *ERJ Open Research, 1*(1). Retrieved from *https://openres.ersjournals.com/content/1/1/00001-2015.*

Waring, W. S., Chui, M., Japp, A., Nicol, E. F., & Ford, M. J. (2004). Autonomic cardiovascular responses are impaired in women with irritable bowel syndrome. *Journal of Clinical Gastroenterology, 38*(8), 658–663.

Weiland, A., Blankenstein, A. H., Van Saase, J. L., Van der Molen, H. T., Kosak, D., Vernhout, R. M., et al. (2016). Training medical specialists in communication about medically unexplained physical symptoms: Patient outcomes from a randomized controlled trial. *International Journal of Person Centered Medicine, 6*(1), 50–60.

Wickramasekera, I. (1994). Psychophysiological and clinical implications of the coincidence of high hypnotic ability and high neuroticism during threat perception in somatization disorders. *American Journal of Clinical Hypnosis, 37*(1), 22–33.

Wickramasekera, I. E. (1995). Somatization: Concepts, data, and predictions from the high risk model of threat perception. *Journal of Nervous and Mental Disease, 183*(1), 15–23.

Wickramasekera, I., & Price, D. C. (1997). Morbid obesity, absorption, neuroticism, and the high risk model of threat perception. *American Journal of Clinical Hypnosis, 39*(4), 291–301.

Wilhelm, F. H., Gerlach, A. L., & Roth, W. T. (2001). Slow recovery from voluntary hyperventilation in panic disorder. *Psychosomatic Medicine, 63*(4), 638–649.

Wilhelm, F. H., Gevirtz, R., & Roth, W. T. (2001). Respiratory dysregulation in anxiety, functional cardiac, and pain disorders: Assessment, phenomenology, and treatment. *Behavior Modification, 25*(4), 513–545.

Wilhelm, F. H., Trabert, W., & Roth, W. T. (2001a). Characteristics of sighing in panic disorder. *Biological Psychiatry, 49*(7), 606–614.

Wilhelm, F. H., Trabert, W., & Roth, W. T. (2001b). Physiologic instability in panic disorder and generalized anxiety disorder. *Biological Psychiatry, 49*(7), 596–605.

Yang, T., & Levy, M. N. (1984). The phase-dependency of the cardiac chronotropic responses to vagal stimulation as a factor in sympathetic–vagal interactions. *Circulation Research, 54*(6), 703–710.

Yates, B. T. (1984). How psychology can improve effectiveness and reduce costs of health services. *Psychotherapy, 21,* 439–451.

CHAPTER 7

Progressive Relaxation

Origins, Principles, and Clinical Applications[1]

F. J. McGuigan
Paul M. Lehrer

DESCRIPTION OF THE METHOD

Progressive relaxation therapy is one of the most well-known and widely used stress management therapies. Originated and first described by Edmund Jacobson in the early years of the 20th century, it involves learning to relax the muscles as a mechanism for decreasing stress, anxiety, and their various physiological and psychological concomitants. The method instructs the learner to tense, at very low levels, various muscles in the body, alternately, and then to release the tension. The purpose is to decrease the perceptual threshold for detecting muscle tension to the point where the individual becomes aware of even the slightest tension above zero, and to teach the individual to control muscle tension so it can be voluntarily completely eliminated. Ultimately, progressive relaxation includes instructions just to "let go" and keep muscles completely relaxed. As described below, this method has a cogent physiological rationale, particularly in view of modern research showing an intimate connection between the skeletal muscles and the sympathetic nervous system (Delius, Hagbarth, Hongell, & Wallin, 1972). In its various forms, the method has been found helpful as a component in psychological treatment for a variety of emotional and physical stress-related disorders.

However, despite the fact that almost all recently trained psychotherapists have had some exposure to the method, few are familiar with the method as originally proposed, evaluated, and described in Jacobson's (1938a) classic book *Progressive Relaxation*. This is important not just for the sake of tradition or methodological purity. The "progressive relaxation" methods most widely used differ substantially both in the method of treatment and in the training goals that Jacobson originally proposed. Although there have been no "head to head" comparisons between the currently used methods and Jacobson's, an outline of their differences deserves mention. Despite the common assumptions, taken from Jacobson's original book, that progressive relaxation training by Jacobson's method must be very lengthy, this is actually not the case. Indeed, Jacobson published a description of a brief treatment protocol (Jacobson, 1964a, pp. 161–201). Years ago, we published a paper contrasting Jacobson's method with the ones more currently in use (Lehrer, 1982).

There are a number of contrasts between the two approaches. Some characteristics of the revised progressive relaxation methods that differ from Jacobson's method include the following:

1. In the newer methods, intense muscle contractions are applied in various muscles throughout the body, with the aim of producing a rebound relaxation response stemming from the contrast between muscle tension and release, called a *pendulum effect* in one influential manual (Bernstein & Borkovec, 1973). The theory was that a muscle would relax to a level lower than baseline after being tensed for a number of seconds and then suddenly released. There is, in fact, little empirical evidence for this. In one study from our laboratory, we found an *increase* in baseline muscle tension after 20 seconds of tension (Lehrer, Batey, Woolfolk, Remde, & Garlick, 1988), the time prescribed in most manuals of the time.

In contrast, Jacobson used muscle tension as a didactic method, rather than as a way to *induce* relaxation. He used deliberate specific muscle contractions to teach the trainee both to recognize what muscle tension feels like and then to control residual tension. He emphasized training in perception and control of very low levels of muscle tone through his "method of diminishing tensions," in which he progressively instructed the learner to tense a muscle less and less, in order to decrease the perceptual threshold for muscle sensations to zero levels. Eventually, Jacobson instructed the learner to completely relax the muscle and then look for perception of remaining residual tension in order to sensitize the individual to recognize underlying muscle tone in everyday life, not just sensations of maximal tension. He also used contraction of specific muscles separately, rather than large groups of muscles, in order to teach people control of all muscles in the body. Thus he would instruct a person just to tense the forearm extensor or flexor muscles one at a time, but never to tense the whole arm. He referred to the work of the 19th-century anatomist and physiologist Sir Charles Bell, who systematically described sensations attributable to muscle tension and called them a "sixth sense" (Bell, 1834, p. 195). Development of concepts relating to muscle sensations, both together and independently of sensations involving movement and force, has recently been reviewed (Smith, 2011). Although Jacobson was loath to give too much description of these sensations to his trainees for fear that he might spuriously and inadvertently suggest these sensations to them, so the learner might think that relaxation has occurred even when considerable tension might actually remain. Jacobson preferred to let the learner discover the muscle sensations on his or her own. Nevertheless, he did on occasion describe them to me (P. M. L.) as "squeezing" sensations. These could be differentiated from sensations of a muscle being stretched, which often feels more like a warm sensation and which occurs even when muscle movement occurs passively, as when someone else bends a person's limb at the joint. Jacobson called sensations of muscle contraction "control sensations" because they accompany activity involving control of the body. Relaxation, to him, was exactly the opposite: relinquishing control, utter passivity. If, after adequate training, a muscle tension sensation was perceived, he referred the learner back to the voluntary muscle contraction made when that sensation was perceived more intensely and instructed the person slowly to diminish the tension, exercising voluntary control over the entire process, until absolutely no sensation remained.

As discussed later, Jacobson's method is similar to that used in surface electromyographic biofeedback (sEMG; see Gevirtz, Chapter 6, this volume), a method that Jacobson invented but then abandoned because he did not want his trainees to be dependent on an external machine. Along with collaborators at Bell Laboratories, he was the developer of the sEMG recorder, which he called the "integrating neurovoltmeter," and he used readings on this instrument of less than one microvolt of electrical activity as a criterion for effective relaxation (Jacobson, 1940).

2. "Relaxation patter" and suggestion are used in the newer methods. These could include such comments as "You feel yourself becoming more and more relaxed," or "When I count backwards from 10 to 1, you will find yourself becoming more and more relaxed." An example from an influential and widely adopted instructional chapter includes the following instructions, with the therapist speaking in a slower, softer, deeper voice when the instruction is given to relax the muscles: "[C]lench your right fist, just clench your fist tighter and tighter, and study the tension as you do so. Keep it clenched and feel the tension in your right fist, hand, forearm . . . and now relax . . . " (Wolpe & Lazarus, 1966).

In our previous discussion of this issue, we noted the following (Lehrer, 1982):

> In contrast, Jacobson teaches the technique purely as a muscular skill. He actively tries to avoid suggestion. He leaves the room between instructions so that the trainee can relax completely without relying on outside stimuli. Also, Jacobson does not even tell the [subject] where in the body the sensations of tension should be when a muscle group is tightened—so eager is he to avoid suggestions of sensations when the trainee may not actually be feeling [them]. . . . Jacobson argues that . . . suggestion prevents the trainee from learning the muscular skill. Rather, he says, suggestion encourages the trainee to feel relaxed during the training session, even if levels of physiological arousal are not lowered. He does not try to *induce* a sense of relaxation during a training session. It is immaterial whether or not a person actually feels completely relaxed during training sessions, particularly the initial sessions in which the trainee has only been trained to relax a few muscle groups. Rather, the learning of the muscular skill of relaxation is paramount. (p. 418)

3. Electronically recorded instructions sometimes are substituted for live personal instruction. Although exclusive reliance on media is not characteristic of most relaxation methods, it frequently has been used as a principal component. There is little evidence that prerecorded relaxation instruction, by itself, teaches a relaxation skill that is measurable physiologically outside a training session (Lehrer, 1982). Again, from our previous discussion of this issue (Lehrer, 1982):

> Why is live training better? Paul and Trimble (1970) hypothesize that live training allows the trainer to make instruction contingent on the performance of the trainee and to give the trainee feedback in his progress. There is evidence, however, that simple mechanical repetition of instruction when a [subject] fails to understand or to perform correctly is *not* sufficient to explain the therapeutic advantages of live training. Subjects in Beiman, Israel, & Johnson's (1978) tape-recorded condition had this contingency as part of their training to little avail. Quayle (1979) did find more pronounced physiological effects when the pace of training was contingent on the [subjects] rather than on a set schedule, whether the training was conducted by a live therapist or by a tape recording. Nevertheless, "live contingent" training was still more effective than "taped contingent training," thus indicating other additional advantages of live over taped procedures. Perhaps these include the ability of the therapist to use visual clues to diagnose difficulties in the client, to explain procedures in different ways, to provide

> individually tailored procedures (e.g., by providing pressure or resistance to [subjects'] movements when [subjects] tense a muscle during training, by pointing out especially tense muscles that [subjects] may be unaware of, etc.). (p. 420)

Despite lack of empirical study of differences between the two approaches to progressive relaxation training, research comparing modern progressive relaxation methods with sEMG biofeedback, which Jacobson thought of as a gold standard for measuring muscle control and relaxation, might shed light on whether differences exist. One study (Reinking & Kohl, 1973) compared the modern version of progressive relaxation training with and without simultaneous sEMG biofeedback. Jacobson's approach is more similar to that of EMG biofeedback, in which, for both, the emphasis is on achieving very low levels of muscle tension, objectively defined. In this study, muscle tension levels were found to be lower in the groups receiving biofeedback than in those just given the newer progressive relaxation technique. Since then, other studies have found sEMG biofeedback to have better results than modern progressive relaxation on alleviating headaches (Arena, Bruno, Hannah, & Meador, 1995; Christie, 2008; Kumaraiah, 1980), although some found no differences between the two procedures (Gada, 1984; Han & Son, 1987). In other studies, EMG biofeedback training, either combined with or in contrast to revised progressive relaxation training, produced greater results than the revised progressive relaxation method alone in reducing pain (Corbett, 1982), anxiety (Corbett, 1982; DeBerry, 1979; Feely, 1978), and drug relapse rate among addicts (Feely, 1978).

Nevertheless, one study found that adding progressive relaxation to an sEMG biofeedback protocol increased the anxiolytic effect (Miro, 1981). There is some evidence that progressive relaxation has greater effects than sEMG biofeedback for behavioral aspects of stress coping and management (Murphy, 1983), but some studies found no differences between the methods for treating sleep-onset insomnia (Nicassio, Boylan, & McCabe, 1982) or for anxiety (Leboeuf & Lodge, 1980; Miller, Murphy, & Miller, 1978). One study found no differences between sEMG biofeedback and progressive relaxation for treating test anxiety (Rothman, 1979).

In general, there appears to be some advantage for more detailed muscle training to decrease muscle tension where a specific disorder is closely linked to it. It is possible that the non-muscle-relaxation components of the revised progressive relaxation techniques have more direct therapeutic effects on other symptom patterns, where there is no direct link to a specific muscular pathway that can be specifically treated by muscle relaxation. This is consistent with a specificity theory of relaxation effects, in which the modality of the effect might be greatest on symptoms directly related to the pathway by which the particular method works.

Consistent with this theory of specific progressive relaxation effects, Jacobson himself often used sEMG recordings as a major criterion for effectiveness of training. In one study, he compared sEMG levels and relaxation times of tense patients from his practice, ordinary people recruited for the research study, and the football team from a local university. Relaxation time was fastest in the football team, followed by the ordinary people, and, slowest, the patients, who eventually, after training, developed the fastest relaxation time of all (Jacobson, 1934, 1936). There is some evidence that people who are used to thinking about their muscles and training and utilizing them in activities beyond that of ordinary everyday life, for example, professional athletes or dancers, tend to find muscle relaxation easier and more appealing. The football players were used to thinking about how muscles worked and appeared to have developed an advanced level of muscular control, even for the very subtle amounts of tension measured by Jacobson. It is not uncommon, in those practicing the method, for athletes (Davis, 1991; Hall & Erffmeyer, 1983;

Haney, 2004; Henderson, 1991; Jacobson, 1934, 1936; Wilson & Bird, 1981), dancers (McCloughan, Hanrahan, Anderson, & Halson, 2016), musicians (Grishman, 1989; Oliver, 1997; Wesner, Noyes, & Davis, 1990), and others involved in activities requiring sensitive muscle control to gravitate to this method and to learn it more quickly. We have also found that people with some neural diseases (Anagnostou et al, 2019) and some older people who have experienced subtle neuromuscular or perceptual impairments (Landelle, El Amadi, & Kavounoudias, 2018) have difficulty perceiving sensations related to muscle control. Neuromuscular impairment is a known side effect of some neuroleptic medications (Garver et al., 1976), and, in the author's clinical experience, can lead to impaired proprioception and difficulty learning to detect low levels of muscle tension.

HISTORY OF THE METHOD

Influences on Jacobson

In 1905 Edmund Jacobson was sent to graduate school at Harvard University by Walter Dill Scott, psychologist and president of Northwestern University, to study with four of the great minds of the day: William James, Walter Cannon, Josiah Royce, and Hugo Münsterberg. All four had a considerable influence on him.

James exhorted him to study "the whole man." Also, although Jacobson never mentioned this, undoubtedly James's peripheralist theory of emotion undoubtedly influenced him. James (1884) wrote, "we feel sorry because we cry, angry because we strike, afraid because we tremble . . . "; that is, the bodily responses elicited by emotion-inducing events *are* the emotion, not just a concomitant or effect of the emotion.

Royce nurtured Jacobson's interest in studying mental function. Jacobson devoted his undergraduate thesis to the study of thought, in which he asked people to describe the process of thinking and found, ultimately, that people described various body sensations. He came back to this interest in his last book, *The Human Mind,* in which he cited evidence that thought is inescapably linked to patterns of muscle tension that accompany it (Jacobson, 1982). An implication of this theory is that complete relaxation of the muscles would enable a person to stop mental activity and thus allow an insomniac to sleep (Jacobson, 1938b).

Cannon influenced Jacobson through his pioneering work on physiological arousal and homeostasis and how they manifest in the autonomic nervous system. Münsterberg influenced him in a negative way that was nevertheless beneficial for progressive relaxation. Münsterberg discharged Jacobson as his assistant because, as Jacobson later related, the data he collected were at odds with Münsterberg's theory. Thus freed to work on his own, Jacobson studied the startle reaction to an unexpected loud noise. He found that there was no obvious startle to sudden noise in more relaxed participants. This was the first systematic study of relaxation, and it marked the birth of progressive relaxation.

After graduating from Harvard, Jacobson worked with Edward Bradford Titchener at Cornell University. He probably was influenced by Titchener in two very important ways: through Titchener's expertise in introspection and through his context theory of meaning. Titchener's context theory held that the meanings of words originate, in part, in bodily attitudes (postures) involving the skeletal muscle system. Related to these two avenues of influence, two contemporary applications of progressive relaxation for clinical purposes are (1) detailed observation of ("introspection" on) minute kinesthetic sensations and accompanying mental processes and (2) clinical interpretation of localized bodily tensions as meanings of acts that occur in one's imagination.

Objective Measurement of Tension

After leaving Cornell, Jacobson received his MD and worked in the Department of Physiology at the University of Chicago from 1926 until 1936; he also conducted a private clinical practice. At Chicago, Jacobson, collaborating with A. J. Carlson, discovered an objective measure of tension: They found that the amplitude of knee-jerk reflexes varied directly with the degree of patients' tension. Consequently, as overly tense patients learned to relax, the amplitude of their knee-jerk reflexes decreased. Jacobson's (1938a) further research on several reflexes established that chronic tonus (sustained tension) of the skeletal muscles increased the amplitude of reflexes and decreased their latency; conversely, reflexes diminished in amplitude and increased in latency as patients relaxed. As general skeletal muscle tone decreased, the involuntary startle reflex also was eliminated. (After about 2 months in our own progressive relaxation classes, we sometimes drop a large book onto the floor when the students are well into their relaxation period. Seldom is there even a blink of the eye in these well-relaxed students.)

As successful as it was, measuring the knee-jerk reflex was cumbersome. Through arduous efforts with the aid of scientists at Bell Telephone Laboratories, Jacobson eventually was able to measure tension directly. He recorded electrical muscle action potentials as low as one microvolt, a unit previously unmeasurable by the physiologists of the day. Thus quantitative sEMG was launched. The resultant use of objective measures of degree of relaxation and tension guided Jacobson to develop and validate progressive relaxation.

Measuring Mental Events

With this new instrumentation, Jacobson made important discoveries about how the mind and body function. He found that, in a relaxed person, just the *thought* of moving a limb was accompanied by unique covert sEMG responses in that limb (Jacobson, 1930a, 1930b). For example, if the individual imagined hitting a nail with a hammer three times, there were three unique sEMG bursts in the preferred arm. Through extensive research, he concluded that all thought is accompanied by skeletal muscle activity, though response amplitude may be extremely low. The eye (Jacobson, 1930c, 1930d) and speech (Jacobson, 1931) muscles, he found, were especially important during visual and speech imagery, respectively. Conversely, his data indicated that mental processes diminished and even disappeared as the skeletal musculature relaxed toward zero. As Jacobson concluded, "It might be naive to say that we think with our muscles, but it would be inaccurate to say that we think without them" (cited in McGuigan, 1978, p. iii). Jacobson reported that teaching people to relax particular muscles in these areas helps to block intrusive thoughts, thus facilitating sleep in insomnia (Jacobson, 1938b) and reducing worry symptoms in generalized anxiety (Jacobson, 1964a).

THEORETICAL UNDERPINNINGS

Progressive relaxation begins with the ancient and venerable concept of rest. Physicians have long known the value of rest, frequently prescribing it in the form of "bed rest," a recommendation made from the time of Hippocrates (trans. 1849). Although prolonged bed rest can lead to deconditioning, with devastating effects throughout the body (Sprague, 2004; Stuempfle & Drury, 2007), brief periods during acute stages of infection or injury have long been recommended as a method to conserve body resources needed for recovery, although evidence for its beneficial effects is now in doubt (Brower, 2009).

Nevertheless, the effects of relaxation on decreasing symptoms of allostatic overload and stress are by now incontrovertible, as reviewed below, as are the beneficial effects of better sleep, better mood, and better frame of mind. Although constant rest may be itself maladaptive, so is overload. The deep rest afforded by progressive relaxation provides the healing respite needed for overloaded bodies and minds.

How Stressors Evoke Tension

Each stressful situation ("stressor") that people meet in everyday life reflexively evokes the primitive startle pattern of rising (covertly or overtly) on the balls of the feet and hunching forward, tightening the abdominal muscles causing increased thoracic involvement in breathing and increased ventilation. The entire skeletal musculature reacts immediately. Within a matter of 100 or so milliseconds, people thereby ready themselves for fight or flight, as Walter Cannon (1929) theorized. This startle reaction, followed by complex autonomic and endocrine changes, has had great survival value. However, it is often prolonged beyond the immediate emergency, resulting in a condition of chronic excess tension and continued hyperactivity of the systems of the body. In particular, consistent, excessive covert tightening of the skeletal musculature overdrives the central nervous system and increases activity of the autonomic, cardiovascular, endocrine, and other systems. Elevated muscle tension is a known concomitant of various anxiety conditions (Rothman, 1979). Prolonged, heightened skeletal muscle tension may then result in any of a variety of pathological conditions and contribute to prolonged heightened sympathetic arousal and allostatic overload.

Principles and Physiology of Progressive Relaxation

In learning progressive muscle relaxation, one cultivates the ability to make extremely sensitive observations of the world beneath the skin. To acquire such heightened internal sensory observation, which is a kind of physiological introspection, one first learns to recognize subtle states of tension. When a muscle contracts (tenses), volleys of neural impulses are generated and carried to the brain along afferent neural pathways. This muscle–neural phenomenon, the generation of afferent neural impulses, constitutes the local sign of tension that one learns to observe, the "muscle sense" of Bell (1834). These sensations emanate from specialized cells in the muscles, called muscle spindles. These cells transmit sensations to the central nervous system.

Sensations from the muscles ordinarily tend to be rather subtle and are easily overshadowed by other sensations coming from the joints and the skin. In progressive relaxation, we try to help the trainee to differentiate these signals. Thus, for example, if one bends the hand backwards from the wrist, the most easily perceived sensation comes from receptors in the wrist joint. This sensation is sharp, sometimes even painful; but it has nothing to do with muscle tension. The actual muscle sensations are on the dorsal side of the forearm, about two thirds of the way from the wrist to the elbow. These are dull sensations, sometimes described as squeezing feelings; sometimes they are difficult to perceive in initial training sessions.

Tension is the contraction of skeletal muscle fibers that generates the tension sensation. *Relaxation* is the elongation (lengthening) of those fibers, which then eliminates the tension sensation. After learning to identify the tension sensation, one learns to relax it away. For this, one learns to allow the muscle fibers that generated the tension to elongate. In the learning process, one contrasts the previous tension sensation with the later elimination of tension. This general procedure of identifying a local state of tension, relaxing it away, and marking the contrast between the tension and the ensuing relaxation is then applied to all of the major muscle groups. In progressive relaxation, one learns to control all of the skeletal musculature so that any portion thereof may be systematically relaxed or tensed, as one chooses. Furthermore, training is given in detecting progressively smaller amounts of muscle tension, in order to drive down the perceptual threshold for detecting even the most minute amounts of muscular tone, and in learning to control it by "switching off" all voluntary activity that produces muscle tension. In that way, muscle relaxation involves complete passivity and absence of all voluntary activity, even that of which the individual may have little prior awareness. Those familiar with EMG biofeedback may wish to think of progressive relaxation as a method of "internal biofeedback" in which the learner internally monitors feedback signals from the muscles instead of perceiving their representations on external readout systems.

The Skeletal Muscles Control Other Bodily Systems through Neuromuscular Circuits

In the 19th century, the psychologist Alexander Bain (e.g., Bain, 1855) claimed that the skeletal musculature is the only physiological system over which a person has direct control. Hence, as Bain, Jacobson, and others held, skeletal muscles are "the instrument of the will." They contain the only receptor cells in the body that can be directly shut off, which is accomplished merely by lengthening muscle fibers. A synonym for *skeletal muscles* is *voluntary muscles,* precisely because, when one wishes to perform an act, one systematically contracts and relaxes the voluntary muscles. For instance, a person who decides to walk contracts muscles to put one foot in front of the other. This point is so obvious that it does not need elaboration. What is not so obvious is that the internal (covert) functions of the body are similarly controlled by means of the skeletal muscles. Progressive relaxation is predicated on the principle that covert functions of the body can also be controlled through slight muscle tensions.

Thus the tension sensation (the muscle sense of Bell) is called the *control signal* because it literally controls the body's activities. Muscles exercise such control as they interact with the brain through "neuromuscular circuits" (Jacobson, 1964a). When volleys of neural impulses generated by contracting muscles feed back to the brain, extremely complex events result, following which neural impulses return to the muscles along efferent neural pathways. The muscles then further contract, directing additional neural impulses to and from the brain, and so on. Numerous neuromuscular circuits throughout the body simultaneously reverberate in this way to carry out the body's functions. By learning internal sensory observation, one can become quite proficient in recognizing control signals wherever they may occur throughout the skeletal musculature. Through practice, those controls may be activated or relaxed. Relaxation of the skeletal muscle controls produces a state of rest throughout the neuromuscular circuits, including reduced activity of the brain itself. The long-range goal of progressive relaxation is for the body to instantaneously monitor all of its numerous control signals and to automatically relieve tensions that are not desired. The trained body has an amazing capacity to monitor the many neuromuscular circuits that reverberate in parallel fashion throughout the body. The ultimate goal is to develop "automaticity," wherein one automatically, unconsciously, and effortlessly identifies and relaxes unwanted tensions.

Jacobson (1964a) emphasized the control functions of progressive relaxation when he used *self-operations control* as a synonym for progressive relaxation. *Self-operations control* was a precedent for contemporary use of such terms as *self-regulation* and *stress management*. Its aims are to increase behavioral efficiency by programming oneself to eliminate tensions that interfere with one's primary purposes. A person can thereby control blood pressure, emotional life, digestive processes, mental processes, and the like, as we later illustrate.

The Concept of Neuromuscular Circuits Has a Venerable History

The concept of reverberating neuromuscular circuits driven by muscle controls is ancient. Dating from the period of the early Greeks, its evolution can be impressively traced through the writings of philosophers, through the scientific Renaissance, through the research of later physiologists and psychologists, and into the very forefront of contemporary scientific and clinical thinking (see McGuigan, 1978). Some of our most prominent thinkers have recognized that the human body functions in terms of information generated and transmitted between the muscle systems and the brain. One of the most influential presentations of this concept was provided by Norbert Wiener (1948) in his classic book *Cybernetics*. In greater depth than all others before him, Wiener developed the model that the body functions according to principles of feedback circuits. As he put it:

> The central nervous system no longer appears as a self-contained organ, receiving inputs from the senses and discharging into the muscles. On the contrary, some of its most characteristic activities are explicable only as circular processes, emerging from the nervous system into the muscles, and re-entering the nervous system through the sense organs, whether they be proprioceptors or organs of the special senses. (Wiener, 1948, p. 15)

A similar neuromuscular concept was put forth by Alexander Bain in 1855:

> The organ of mind is not the brain by itself; it is the brain, nerves, muscles, and organs of sense. . . . We must . . . discard forever the notion of the sensorium commune, the cerebral closed, as a central seat of mind, or receptacle of sensation and imagery. (cited in Holt, 1937, pp. 38–39)

More recently, a considerable number of research findings have shown a close connection between skeletal muscle innervation and the sympathetic nervous system (Roatta & Farina, 2010; Wallin, 2006; White, Shoemaker, & Raven, 2015) such that increased muscle tension triggers a burst of sympathetic activity, causing constriction of blood vessels within the muscle tissue. Muscular sympathetic nerve activity is involved in regulation of systems throughout the body (Roatta & Farina, 2010; Wallin, 2006; White et al., 2015), including blood pressure control, through the baroreflexes (Kienbaum, Karlssonn, Sverrisdottir, Elam, & Wallin, 2001), which are important modulators of autonomic stress responses (cf. Lehrer, Chapter 10, this volume). Thus, by modulating muscle tension, sympathetic nervous system activity throughout the body is affected.

Neurophysiology of Relaxation

In various publications, Gellhorn (e.g., Gellhorn, 1958b; Gellhorn & Kiely, 1972) sought to specify the neural mechanisms by which the skeletal musculature leads to relaxation of the body. Gellhorn was especially impressed with Jacobson's method, and Jacobson approved of Gellhorn's theorizing as to those neural mechanisms (Jacobson, 1967). Gellhorn started with the basic fact that progressive relaxation decreases afferent neural impulses from the skeletal musculature. He then noted that the reticular formation receives considerable innervation from those skeletal muscles, so that relaxation reduces activity there. The reticular formation, in turn, functions in circuits with the posterior hypothalamus and thence with the cortex. Consequently, muscular relaxation reduces proprioceptive input to the hypothalamus, with a resulting lessening of hypothalamic–cortical and autonomic discharges. As an animal analogue of progressive relaxation effects, Gellhorn studied the effects of the drug curare, which blocks transmission at the neuromuscular junction and thereby completely eliminates muscle tension. He found that curare produced a state of somnolence in cats, along with decreased sympathetic activity (Gellhorn, 1958a). Gellhorn concluded that lessened emotional reactivity during muscular relaxation is the result of reduced proprioceptive impulses to the hypothalamus, which then decreases excitability of the sympathetic nervous system. Jacobson (1967) summarized research by Bernhaut, Gellhorn, and Rasmussen (1953) as follows:

> These findings suggest that a relaxation of the skeletal musculature is accompanied by a diminution in the state of excitability of the sympathetic division of the hypothalamus and, through a reduction in the hypothalamic–cortical discharges, by a similar reduction in the state of excitability of the cerebral cortex. (Jacobson, 1967, p. 155)

In these ways, then, the skeletal muscles can control other systems of the body, including the reduction and elimination of mental (including emotional) events.

More recent research on muscle relaxation therapy has documented decreases in sympathetic arousal, including a decrease in circulating norepinephrine levels and myocardial contractility (Davidson, Winchester, Taylor, Alderman, & Ingels, 1979), as well as decreased electrodermal activity and heart rate levels and reactivity (Lehrer, 1978; Lehrer, Schoicket, Carrington, & Woolfolk, 1980; McGlynn, Moore, Lawyer, & Karg, 1999; Shapiro & Lehrer, 1980). The close connection between the skeletal muscles and the sympathetic nervous system has received much empirical attention. The muscles are an important element in a complex feedback system that controls physiological arousal. Perception of muscle sensations and afferent feedback from the muscles are provided by active sensory cells called *muscle spindles*. The muscle spindles are active in that they may expand or contract independently of actual muscle tension. Efferents to the muscle spindles may therefore control the amount of afferent feedback provided by muscle tension. Activity in the muscle spindles is strongly influenced by the sympathetic system (Grassi & Passatore, 1988; Roatta, Windhorst, Ljubisavljevic, Johansson, & Passatore, 2002). By controlling this activity, one may directly alter the feedback loop between the muscles and the sympathetic system during progressive relaxation training. Perception and control of muscle spindle activity may be an important mechanism behind the effects of progressive relaxation in diminishing sympathetic arousal.

Differential Relaxation

Differential relaxation is the optimal contraction of only those muscles required to accomplish a given purpose. Those and only those muscles should contract, and they should contract only to the extent required to accomplish the purpose at hand. All other (irrelevant) muscles of the body should be relaxed. In the moment-to-moment monitoring of tensions throughout the day, people can often catch themselves wasting energy. Some needlessly clasp their hands together; others tap their fingers and feet, wrap their legs around the legs of a chair, or needlessly rock back and forth. In learning differential relaxation, while studying a particular tension signal that is to be controlled, the learner recognizes other tensions elsewhere in the body. These are unwanted tensions that can be relaxed away when the learner later practices on that part of the body. By learning to differentially relax 24 hours a day, a person can save considerable energy, so that relevant tensions can be more efficiently directed toward the accomplishment of specific goals. Later we consider some specific applications of the principle of differential relaxation.

The Method of Diminishing Tensions

In developing control over one's muscles, it is necessary (eventually) to detect the most subtle control signals. For this purpose, progressive relaxation starts with relatively obvious control signals generated in the dorsal surface of the forearm by raising the hand at the wrist to nearly a 90° angle. Thus the learner initially perceives a localized sensation of tension in the forearm. With the "method of diminishing tensions," one then studies tensions of ever-decreasing intensity. Thus, after the control signal generated by raising the hand vertically at the wrist is studied, for the next practice the hand is raised only half as much—at a 45° angle from the horizontal. Then the third practice position is to raise the hand only half as high as before (at about a 20° angle); on successive practice positions the hand is raised less and less until movement is imperceptible, but perception of tension persists. The eventual goal is to identify tension signals of perhaps 1/1,000th the intensity of those with which the learner began. Such signals are common in the minute muscles of the tongue and eyes, but they occur in nearly all muscles.

Some practitioners give instructions to generate high-intensity tensions (e.g., to clench the fist tightly). We believe that this practice is counterproductive for learning to perceive and control *low-intensity* tensions. Many covert responses are below 1 μV. To control small tensions, one should study *them* rather than large tensions.

Avoiding Suggestion

In learning progressive relaxation, trainees are never told that they are doing well, that they are getting better, that they are relaxing, that their hands feel heavy, that they are getting sleepy, or the like. No attempt is made to convince the individual that he or she will be "cured" in any sense of the word. Instead, the trainees are aided by instructions, just as in any other learning procedure. Thus a teacher may interrupt a trainee's practice with criticism whenever the individual is failing to relax.

Jacobson (1938a) listed a number of reasons for avoiding suggestion. As with the placebo effect, any method will accomplish something (although usually only temporarily) if it instills into the person the belief that he or she will benefit from its application. Jacobson pointed out that relaxation is a fundamental physiological occurrence that consists of learning to elongate muscle fibers systematically. He specified definitive physiological changes in the body that differ from those occurring during suggestion. The trainee may be skeptical in regard to the procedure, but he or she still can learn very well when presented with objective evidence of progress. Moreover, the person learns to be independent of the therapist; in "suggestion" therapies, by contrast, dependence on therapists is engendered. As Lehrer, Woolfolk, and Goldman (1986) added,

> [Jacobson held that] the danger of suggestion . . . is that it may make the individual feel that relaxation is taking place even when it is not. The *perception* of relaxation is not so important as actual physical relaxation, according to Jacobson. Therefore, suggestion may be deleterious because a person may stop devoting the time and concentration necessary to learn relaxation if he or she [incorrectly] feels relaxed already. (Lehrer et al., 1986, p. 202; italics in original)

Tape-Recorded Relaxation Instructions and Biofeedback

Just as Jacobson eschewed the use of suggestion in relaxation instructions, he also avoided the use of tape-recorded instructions. He did this primarily because he thought that tape-recorded instructions might offer more suggestion than training. In support of this position, a literature review by Lehrer (1982) found that taped training did not produce physiological effects that were measurable outside of training sessions.

Jacobson also recommended against using sEMG biofeedback, even though he was the first to use this technique (see Jacobson, 1978, Figure 25, p. 146). He thought that people should not depend on external sources of biological information but should develop their own powers to sense very low levels of muscle tension and to relax in all situations, even when a biofeedback machine is not available.

However, modern technology has made sEMG biofeedback a much easier and cheaper methodology. People now can afford to have home monitors, which may be used as teaching aids for attaining more sensitive perception and greater control of the muscles. Jacobson's objections to biofeedback may no longer apply.

Relaxation Practice Is Not an Exercise

Many suggestions about how to relax use a lay meaning of the term, which is inappropriate in a scientific/clinical context. For instance, advice to exercise is not advice to relax, because exercise is work. Exercise is very advisable on other grounds. For the same reason, terms such as *relaxation exercises* or *relaxation response* are self-contradictory, because *exercise* and *response* are "work words." The essence of relaxing is to allow the muscle fibers to elongate, which is physiologically impossible when one *tries* (through exercising or responding) to accomplish it. One simply cannot make an *effort* to relax, because an effort to relax is a failure to relax.

Is There a Shortcut?

From a naive learner's point of view, the amount of time required to learn progressive relaxation may seem excessive. Indeed, one needs to learn to control a large mass of muscle that makes up almost half the body weight. Recognizing the desire on the part of the learner for brevity, Jacobson spent many years attempting to shorten the method. However, he abandoned his attempt, because patients did not sufficiently generalize from what they learned in the clinic to everyday life. His conclusion was that there simply is no satisfactory brief method for learning to relax a body that has been producing excessive tension for decades. Nevertheless, Jacobson (1964b, 1970) did offer a "briefer course," reducing the time devoted to each muscle group. For instance, instead of practicing for 3 hours on a single position, one practices three positions in 1 hour, starting with the first three in Table 7.1 (later in the chapter). Similar abridgments have been made for other muscle groups. The complete course can thus be shortened to one-third of the time it ordinarily takes. However, in this world "you get what you pay for," so that you learn considerably less control from a briefer than from a longer course. An appropriate analogy is learning to play the piano: Certainly you can practice for shorter periods, but your competence is thereby reduced. Nevertheless, in clinical practice, the method has been routinely shortened to six or fewer sessions by combining training in several muscle groups in a single session (e.g., muscles of the arms in one session, then the legs in another, the trunk in a third, the face and neck in the fourth, and differential relaxation training in the fifth and sixth).

Jacobson's research in school systems and in clinical work led him to conclude that children learn progressive relaxation quite rapidly. His reasoning was that they have not spent so many years acquiring maladaptive tension habits that must be reversed. Teaching progressive relaxation in elementary school has been done on a large scale in Sweden (Setterlind, 1983).

With this explanation of the principles of progressive relaxation, we now turn to the psychologically important topic of clinical control of mental (cognitive) processes. To establish a basis, we first consider the scientific nature of mind and its component mental events.

A Psychophysiological Model of Mind

Mental (cognitive) events are generated by the selective interaction of reverberating neuromuscular circuits. Various functions of the everyday notion of "mind" are indicated by such terms as *ideas, images, thoughts, dreams, hallucinations, fears, depression,* and *anxieties.* According to the present model, all such mental (cognitive) events are generated when selective systems of the body interact through highly integrated neuromuscular circuits. Most mental processes are generated when muscles of the eyes and speech regions tense, whereupon specialized circuits to and from the brain are activated. Other pathways are activated also, including those involving the somatic musculature and the autonomic system. A detailed presentation of and perhaps the most extensive documentation for this neuromuscular model of the generation of mental events are provided in McGuigan (1978).

1. *Muscular events are present during cognition.* McGuigan's (1978) summary of relevant research over an 80-year period provides a firm basis for the conclusion that muscular contraction in selected regions of the body corresponds to the nature of the mental activity present. During visual imagery, the eyes are uniquely active (e.g., when one is imagining the Eiffel Tower, the eyes move upward in imaginal scanning as detected through electro-oculography). During imagining, somatic activity EMG readings detect localized covert responses (e.g., imagining lighting a cigarette produces a distinct covert response in the active arm). Covert muscular responses have been recorded in the speech musculature during a great variety of thinking tasks; for example, there was heightened tongue EMG while participants were performing a verbal mediation task using Tracy Kendler's paradigm (McGuigan, Culver, & Kendler, 1971). In addition, there is heightened speech muscle activity in both children and adults during silent reading; increased speech muscle activity covertly occurs while individuals are engaged in cursive handwriting; in deaf children, covert responses occur in the fingers, which are the locus of their "speech" region, while they think; rapid, phasic speech muscle activity occurs during night dreams involving auditory content; heightened speech muscle activity occurs in patients with paranoid schizophrenia during auditory hallucinations; and so on for other mentalistic activities (see especially McGuigan, 1978, Chapter 10). Conversely, there is no conscious awareness at all when people are well relaxed, as objectively determined by a lack of tension measured through EMG readings (Jacobson, 1938a).

The reasoning here is that because specific muscle activity occurs during cognitive activity, and because cognitive activity disappears when this muscle activity is reduced to zero, it may be concluded that muscle activity is a critical component of those cognitive events.

2. *Numerous covert reactions during cognition are related by neuromuscular circuits*. Although there are foci of muscular activity in selective regions of the body depending on the nature of the cognitive activity, other covert responses are simultaneously occurring throughout the skeletal musculature. For example, while participants in one study processed a silent answer to a question, events were simultaneously recorded in the arms, lips, neck, and eyes, as well as in the left temporal lobe and left motor area of the brain (McGuigan & Pavek, 1972). The conclusion is that these unique, simultaneously occurring events throughout the body are not independent. Rather, they are related by means of rapidly reverberating neuromuscular circuits between the brain and the extensive skeletal musculature. Because those widespread events occur simultaneously with the silent thought, it is assumed that the neuromuscular circuits generate that thought. We turn now to how such a verbal thought is generated.

3. *There are general linguistic, visual, and somatic components of cognition*. Focusing on linguistic cognition, research has indicated that speech muscles generate a phonetic code, which is presumably transmitted to and from the linguistic regions of the brain (see especially McGuigan & Winstead, 1974; McGuigan & Dollins, 1989). When those speech muscles and linguistic brain regions function in unison, perceptual understanding of linguistic cognitions occurs. No doubt similar processing occurs to generate nonlinguistic cognitive activity. Thus circuits between the eyes and the brain generate visual imagery, and circuits between the nonspeech skeletal musculature and the brain generate somatic components of thoughts (see McGuigan, 1989, 1991b).

Control of Cognitions

From a practical point of view, this model of the mind makes it abundantly clear how people can volitionally control their emotions and other cognitive activities, as well as other bodily functions. That is, if cognitive activities are identical with the energy expended when neuromuscular circuits reverberate, those cognitive events can be eliminated when the neuromuscular circuits cease to be active. They stop reverberating when a person relaxes the skeletal muscle components.

The Meaning and Purpose of Tensions

Recalling Titchener's context theory of meaning, a compatible basic principle of progressive relaxation is that every tension has a purpose—that every tension means something. This point is obvious in many instances. For example, the purpose of the tension in the upper surface of the forearm while bending back the hand at the wrist is simply to raise the hand. Similarly, the purpose of tensions in the muscles of the legs while walking is simply to move the body. What is not so obvious is the interpretation of subtle muscular tensions in the application of clinical progressive relaxation.

Distinguishing between "Meaning" and "Process"

To interpret control signals, one learns that *process* is the way in which meaning is generated—process is the actual tension sensation that one observes within one's body. *Meaning* designates the purpose of the tension, the reason why one tenses. In generating mental events, "process" consists of the muscular contractions within neuromuscular circuits that generate the relevant images, sensations, and so on. "Meaning" is thus the content of those mental processes.

In therapy, a patient is first carefully trained in detecting (proprioceptively introspecting on) subtle tensions throughout the body that constitute process. Then she or he is carefully trained in developing the ability to introspect on and report the content of mental activity in considerable detail. Process usually occurs in unexpected places in the body. The patient first identifies the nature and locality of process. When these are identified, the question to be answered by clinician and patient working together is this: Why do those tensions occur in particular regions and during a given kind of mental activity? Establishing the meaning of the tensions can give the patient better understanding of and control over his or her difficulties. For example, while learning to relax, a man observed subtle tensions throughout his entire right leg. That was process. After some study, the tensions were interpreted as follows: The man was tensing *as if* he were about to fall out of a tree house and crash into a board with the leg. The mental content generated by the covert tensions in the leg was his remembrance of actually having fallen out of a tree house when he was a boy. As the muscles in the leg covertly contracted in the present, he relived that experience in his memory as if it were overtly occurring. Rolfers report similar experiences when muscles are stimulated.

Consider a case of a woman whose complaints included anemia, chronic constipation, nervous tension with inability to sit quietly, slight dizzy spells during excitement, and a slight discharge from the nose (Jacobson, 1938a). After training, she reported the process of sitting stiffly and formally. The meaning of this apparently was that she sought to maintain proper posture in her back because of a fear of developing a habit of faulty posture. That is, the purpose served by maintaining a stiff and formal posture was the prevention of an incorrect everyday posture. To control the tension on the meaning level, she came to understand the reasons why she held herself stiffly and was persuaded to change; on the process level, she learned how to relax the relevant controlling muscles.

In clinical work, it may take a long time to identify tensions characteristic of the "nervous" condition of the patient, to interpret those tensions, and to deal with them effectively. But the history of clinical progressive relaxation is one of considerable success in following this paradigm. For example, anxiety is regarded as a fearful condition represented in the skeletal musculature. Once the clinician can ascertain the meaning of the skeletal muscle representations, it is then possible to relax those critical tensions, whereupon the state of anxiety can be diminished or eliminated. We return to the discussion of anxiety later in this chapter.

ASSESSMENT

Applications of the Method

There are two general purposes of tension control—prophylactic and therapeutic. By learning to relax differentially 24 hours a day, a person can increase the likelihood of preventing a stress or tension disorder. For a person already thus victimized, clinical progressive relaxation can often ease or eliminate the condition.

For over seven decades, Jacobson (e.g., 1938a, 1970) collected an abundance of scientific and clinical data that validated the therapeutic application of progressive relaxation for "psychiatric tension pathologies" (his term). These included nervous hypertension, acute insomnia with nervousness, "anxiety neurosis" (what we now would consider to be panic disorder, generalized anxiety disorder, or one of the other anxiety disorders), cardiac neurosis, chronic insomnia, cyclothymic disorder, obsessive–compulsive disorder, hypochondria, fatigue states, and dysthymia. Somatoform disorders to which he successfully applied progressive relaxation included convulsive tic; esophageal spasm; various bowel disorders including colitis, irritable bowel, and chronic constipation; arterial hypertension; and tension headaches. More recent controlled studies have found improvement in a variety of disorders after at least eight sessions of training in Jacobson's technique, including chronic pain (Gay, Philippot, & Luminet, 2002), headache (Murphy, Lehrer, & Jurish, 1990), anxiety (Lehrer, 1978; McCann, Woolfolk, & Lehrer, 1987), and generalized stress (Carrington et al., 1980; Lehrer, Atthowe, & Weber, 1980; Woolfolk, Lehrer, McCann, & Rooney, 1982). Effects on asthma, although *statistically* significant, tend not to be of *clinically* significant magnitude. Clinical improvement is seen after several months of practice, although the acute effects of relaxation are sometimes of bronchoconstriction due to parasympathetic rebound during relaxation (Lehrer et al., 2004).

Therapy

Cognitive (psychiatric) and somatoform (psychosomatic) disorders, as well as lesser conditions, are characterized by excessive, chronic tension and may be reversed by relaxing the skeletal muscles. To summarize, practice in the gradual lengthening of skeletal muscle fibers can result in a generalized state of relaxation, which in turn can produce a state of relative quietude throughout the central nervous system. Consequently, the viscera can also relax, as evidenced by a lowering of blood pressure, a reduction of pulse rate, and a loosening of the gastrointestinal tract. In this way, numerous somatoform disorders can be alleviated or eliminated.

The rationale for treating cognitive aspects of neurotic and related disorders is to interrupt the reverberation of neuromuscular circuits, preventing undesired thoughts from occurring. Some drugs can interrupt neuromuscular circuits by acting on the brain. However, an alternative way to cause these circuits to be tranquil is to relax the tense muscles that are their peripheral components (a "natural tranquilizer"). Verbal components of undesired thoughts, such as those of phobias and worries, can be eliminated by relaxing the speech muscles (tongue, lips, jaws, throat, and cheeks). The eye muscles are the focus for eliminating the visual imagery of thoughts. When the eye muscles are totally relaxed, one does not visually perceive anything; the eyeballs must move in order for visual perception or visual imagery to occur. Thoroughly relaxing all of the muscles of the body can bring all undesired mental processes to zero.

Developing Emotional Control

Jacobson (1938a) demonstrated that relaxation and the experience of emotions are incompatible—that it is impossible to experience emotions while simultaneously relaxing. The paradigm for controlling emotions, as for controlling other mental events, is to control the skeletal musculature that generates them when neuromuscular circuits are selectively activated.

The goal is to determine rationally when to experience and when not to experience particular emotions. One can thus be wisely emotional by wisely tensing to allow favorable emotions to flow freely and inhibiting negative emotions such as temper tantrums or anxiety states. As has been observed clinically in numerous cases, patients who learn to control the skeletal musculature in both its tonic and phasic activity diminish undesired emotions, such as proneness to anger, resentment, disgust, anxiety, or embarrassment. Conversely, as general tension increases, the proprioceptive impulses thereby generated increase emotionality by exciting the central nervous system, the autonomic system, the endocrine system, and so on, presumably through the pathways specified by Gellhorn (1958b; Gellhorn & Kiely, 1972).

Both increased and decreased emotionality are objectively evidenced by the amplitude and latency of patients' reflexes, as discussed earlier. Everyday examples of this point are obvious, such as when a saucer is accidentally dropped at a tea party. The excessively tense guest will emit an exaggerated startle reflex with heightened emotionality, whereas the well-relaxed person may not even blink or interrupt ongoing conversation.

Individuals who are excessively anxious often continually rehearse their griefs, worries, and difficulties with life. If they can acquire control of the skeletal muscle tensions that key this internal speech, they can consequently control their emotions and other negative mental processes. These controls occur principally in the eye muscles, for visualizing their difficulties, and in the speech muscles, for verbalizing their problems, though the remaining mass of skeletal musculature also helps to control mental processes. Thorough training in progressive relaxation makes it possible to change gradually away from a condition of continually attending to difficulties and to develop a habit of turning attention away from those issues. Anxious individuals thus become better able to verbalize relevant contingencies and to react to problems more rationally. That is to say, instead of reacting reflexively to a difficulty, they can stop and reason about the problem (unless, of course, it is something like an onrushing truck). As they become relaxed, then, they attend less frequently to disturbing issues and can instead focus on other matters and become less emotionally disturbed about problems. A trained person can stop, momentarily relax, and assess a situation—verbalizing, for instance, that "this other person seems to be yelling and screaming at me, and it is to his advantage as well as to mine if I do not yell and scream back."

Specific Applications of Differential Relaxation

The term *tension control* does not mean the same thing as *tension reduction,* because people could not function in life without tensions. The purpose is not to eliminate all tensions but to control them so that they can be wisely used. In other words, the purpose is to relax differentially, which can be prophylactic as well as therapeutic.

For instance, relaxed eating behavior is as appropriate for healthy people as it is for patients with ulcers. Many people exhibit bizarre, often frantic, eating patterns. Such an individual may be hunched over a plate with elbows on the table, eating tools grasped in the hands, tensed legs, and bent shoulders—all as if the eater is ready to leap in animalistic protection of the food should an adversary momentarily appear. The eating process is often a continuous shoveling of food from plate to mouth, with no interruption of the chewing process. Conversation, if any, is through half-ground food, with particles exuded in the direction of the listener. Such excessively tense eating habits most assuredly do not contribute to smooth digestion. People should be differentially relaxed when eating in order to help prevent a variety of gastrointestinal difficulties, as well as to enjoy dining as a pleasant process.

Similarly, a major industry that dispenses a wide variety of products dedicates itself to helping people alleviate their sleep problems. The complaints of such people include not getting to sleep when they first get in bed, as well as waking up during the night and not getting back to sleep. One patient reported that he had only about 2 hours of actual sleep over a period of 4 nights in bed. The consequences of night after night of inadequate sleep can be catastrophic, producing chronic fatigue and inefficient work performance. Nonprescription medicines, opaque blinds for the windows, earplugs, and covers over the eyes are meant to satisfy complaints that the room is "too hot," "too noisy," "too bright," "too cold," and so forth. The effective solution for the insomniac's problems is to learn to practice differential relaxation 24 hours a day, which includes sleeping at night. By applying the principles of differential relaxation, one can carry the habit of automatic relaxation into the sleeping state.

Several other common applications of differential relaxation discussed by McGuigan (1991a) include relaxing while hurrying, conquering the fear of flying by differentially relaxing on an airplane, controlling one's own temper, and learning how to deal with unreasonable people by controlling the tempers of others, too.

Support for Various Applications: Problems in the Literature

Jacobson's clinical applications of progressive relaxation are impressive indeed. However, there apparently are no experimental (vs. clinical) data that validate the method, probably because of the extensive methodological difficulties in conducting an experiment. That is, a true experimental test would require randomly assigning a sufficient number of patients to two or more groups and giving the experimental group(s) extensive training over an extended period with an hour of practice each day. A procedure approximating that of the clinical case study presented at the end of this chapter would have to be employed with a number of experimental participants—a demanding requirement indeed. The problem of comparable activity in a control group (or groups) to contrast with such an extensive treatment presents another difficult issue.

Although the literature on various forms of relaxation therapy is impressive, descriptions of the length and nature of training indicate either that the research has not used Jacobson's progressive relaxation procedure or that this procedure has been confounded with other methods. Several examples should illustrate. Nicassio and Bootzin (1974) gave their participants four 1-hour individual sessions using something of an approximation to progressive relaxation; however, in a short training period, "the entire sequence of muscles was covered at each session" (p. 255). Murphy, Lehrer, and Jurish (1990) taught participants a combination of progressive relaxation training and autogenic training and used a headache diary, a cognitive questionnaire, and an expectancy measure for their dependent variables. Because their participants learned both methods, however, specific conclusions about progressive relaxation are precluded. Schaer and Isom (1988) confused progressive relaxation with hypnosis, stating that "progressive relaxation has some similarities to hypnosis" (p. 513), and used a scale of hypnotic susceptibility to specify participants who were "susceptible to progressive relaxation."

Despite the lack of experimental data on this original version of progressive relaxation, extensive clinical and related data nevertheless lend credence to the effectiveness of the method. A summary of research on clinical applications is included in Table 7.1.

Limitations and Contraindications

We have seen that progressive relaxation can be appropriately applied to the reduction of everyday tensions, as in differential relaxation, and clinically to the elimination of syndromes related to stress and tension. We have discussed a number of cognitive and somatoform disorders, along with other tension-related maladies, that have been shown empirically to benefit from progressive relaxation. One could not expect to use relaxation directly to remove a cancer or to cure a viral infection. At the same time, progressive relaxation can be an adjunctive therapy that can ease discomfort resulting from any malady. There do not appear to be any contraindications to its use.

TABLE 7.1. Significant Clinical Effects of Progressive Relaxation, Including Jacobson's Method and Abbreviated Methods

Insomnia Borkovec & Weerts (1976); Dayapoglu & Tan (2012); Freedman & Papsdorf (1976); Greeff & Conradie (1998); Gustafson (1992); Johnson (1993); Karbandi et al. (2015); Lichstein et al. (1999); Means, Lichstein, Epperson, & Johnson (2000); Nicassio & Bootzin (1974); Nicassio, Boylan, & McCabe (1982); Turner & Ascher (1979); Yilmaz & Kapucu (2017)	*Anxiety and stress* Canter, Kondo, & Knott (1975); De Berry (1981); Dehghan-Nayeri & Adib-Hajbaghery (2011); Leboeuf & Lodge (1980); Lehrer (1978); Manzoni, Pagnini, Castelnuovo, & Molinari (2008); McGlynn, Moore, Lawyer, & Karg (1999); Miller et al. (1978); Ost (1988); Schaer & Isom (1988); Tsitsi, Charalambous, Papastavrou, & Raftopoulos (2017)
Headache Emmen & Passchier (1987); Engel, Rapoff, & Pressman (1992); Kroner-Herwig, Plump, & Pothmann (1992); Tornoe & Skov (2012)	*Stress and anxiety in medical conditions* Akmese & Oran (2014); Alarcon, Jenkins, Heestand, Scott, & Cantor (1982); Baider, Uziely, & Kaplan De-Nour (1994); Cheung, Molassiotis, & Chang (2003); Kim, Na, & Hong (2016); Lowe et al. (2002); Renfroe (1988); Tsitsi et al. (2017); Yu, Lee, & Woo (2007)
Immune system function Chen & Francis (2010); Coppieters et al. (2016); Engel, Jensen, & Schwartz (2004); Kang et al. (2011); Masoudi, Sharifi Faradonbeh, Mobasheri, & Moghadasi (2013); Wardell, Rintala, Duan, & Tan (2006)	*Diabetes control* Jablon, Naliboff, Gilmore, & Rosenthal (1997); Lammers, Naliboff, & Straatmeyer (1984); Surwit & Feinglos (1983)
Seizures Puskarich et al. (1992); Rousseau, Hermann, & Whitman (1985); Whitman, Dell, Legion, Eibhlyn, & Statsinger (1990)	*Dyspnea and respiratory disease* Lehrer, Hochron, McCann, Swartzman, & Reba (1986); Renfroe (1988); Vazquez & Buceta (1993); Yilmaz & Kapucu (2017)
Depression Broota & Dhir (1990); Nicassio et al. (1982)	*Hypertension* Beiman, Graham, & Ciminero (1978); Fey & Lindholm (1978); Walsh, Dale, & Anderson (1977)
Pain Akmese & Oran (2014);"Progressive relaxation eases," (1974); Carroll & Seers (1998); Chen & Francis (2010); Coppieters et al. (2016); de Paula, de Carvalho, & dos Santos (2002); Engel et al. (2004); Graffam & Johnson (1987); Masoudi et al. (2013); Seers, Crichton, Tutton, Smith, & Saunders (2008); Shaw & Ehrlich (1987); Sloman, Brown, Aldana, & Chee (1994); Wardell et al. (2006)	*Hyperactivity* Braud (1978); Chen & Francis (2010); Coppieters et al. (2016); Engel et al. (2004); Klein & Deffenbacher (1977); Masoudi et al. (2013); Wardell et al. (2006)
	Digestion and alimentary problems Blanchard, Greene, Scharff, & Schwarz-McMorris (1993); Jacobson (1925); Sigman & Amit (1982); Turnbull & Ritvo (1992)

Note. This table includes studies with significant clinical effects for progressive relaxation to illustrate the various uses of the technique. It is not meant to be an exhaustive or evaluative review of studies on this topic.

Progressive relaxation does not induce anxiety. Some investigators have reported an adverse effect of relaxation therapy, referring to it as "relaxation-induced anxiety" (e.g., Heide & Borkovec, 1984; Lazarus & Mayne, 1990). It is reported that learners become frightened of sensations, fear losing control, fear the experience of anxiety, engage in worrisome cognitive activity, and the like. "Relaxation-induced anxiety" apparently results from using some of the revised methods of progressive relaxation. Lehrer et al. (1988) specified several differences between what they called "post-Jacobsonian progressive relaxation techniques" and Jacobson's progressive relaxation. "Briefer methods," in which learners engage in large tensions, do not use the method of diminishing tensions and rely heavily on suggestion, clearly departing from progressive relaxation. "Relaxation-induced anxiety" apparently results from such other methods of relaxation, but it rarely if ever occurs using this approach to progressive relaxation and is not a contraindication for progressive relaxation. What sometimes does happen in progressive relaxation during the early stages of learning to observe and control internal tension signals is that patients say such things as, "I think my body is floating." In the initial stages of learning to control anxiety, a learner experiences the world beneath the skin for the first time and lacks words to describe novel sensations adequately. However, these experiences are minor, causing no undue discomfort. In any event, they are forgotten after the first month or two of training, when such ambiguous statements are replaced with more precise reports of process, such as, "Tension in my lower left calf." It also is possible that, among people who chronically hyperventilate, the decrease in metabolic rate accompanying deep relaxation may not be sufficiently accurately or quickly tracked by a commensurate decrease in ventilation, causing hyperventilation symptoms (Ley, 1988). Hyperventilation is discussed in this book by Lehrer (Chapter 2), Meuret and Ritz (Chapter 11), and van Dixhoorn (Chapter 12) and can usually be remedied by instructing the individual to breathe more slowly and shallowly for a few minutes.

Another event that sometimes occurs early in the learning process is the "predormescent start," in which the trunk and limbs may give a convulsive jerk. Apparently, it takes place in individuals who have been hypertense during the day's activities or are experiencing a traumatic event. The physiological mechanism may be similar to that of a nervous start, so that it disappears as relaxation progresses but may appear again after exciting experiences. In any event, some months later the learner usually does not recall having made the predormescent start.[2]

THE METHOD

Introducing the Method to the Client

The basic physiology of neuromuscular circuits and the nature of tension and relaxation are explained to the learner. Muscles, the learner is told, contain muscle fibers that are about the diameter of the human hair and are aligned in parallel. Their action is very simple, in that they can do only two things: by sliding alongside each other, they can either contract (tense) or lengthen (relax). When muscles contract, they generate the control signal that is used within neuromuscular circuits to control the functions of the body. Relaxation of the body is achieved when a person learns how to allow the muscle fibers to elongate.

The learner is provided with a realistic estimate of how far an overly tense individual must go. It is explained that there are some 1,030 striated muscles in the human body, which make up almost half of the body weight. A lifetime of injudicious use of such a mass of muscle simply cannot respond to "quick and easy cures" for tension maladies. Just as the learner has spent a lifetime learning how to misuse the muscles, it is reasonable to expect that prolonged practice is required to reeducate them. It simply takes time and practice to learn to reverse long-standing maladaptive muscular habits. Fortunately, this cultivation of a state of bodily rest can be achieved in much less time than it took to learn deleterious muscle habits in the first place.

A frequently asked question is "How long will it take me to learn to relax?" A reasonable answer is to counter with the question, "How long would it take you to learn to play the piano [or become a good golf, chess, or tennis player]?" The answer, of course, depends on where one starts and on how proficient one wishes to become. An answer more acceptable to the prospective learner is that the basic course specified by Jacobson (1964b) and by McGuigan (1991a) is about 13 weeks in length. In our experience, students who take a university course covering those practice positions become quite proficient by the end of a semester. For those who have a neurotic disorder such as a phobic reaction, 6 months or a year of therapy may be required.

A disadvantage of progressive relaxation is that some people complain that "it takes too much time." People who complain that they "don't have enough time to practice daily" can only suffer the consequences. If they let their bodies get into the painful condition of being phobic, having intense headaches, having bleeding ulcers, or the like, then they might wish that they had learned preventive progressive relaxation. Jacobson's clinical experience led him to conclude that learning progressive relaxation sufficiently early can add 20 years to a person's life. If one totals up all the hours of practice and compares that with an additional 20 years of life, the practice time would seem well worthwhile. Other suggestions include the one that Jacobson gave one of us (P. M. L.)—to awaken an hour earlier each day for practice. The relaxation would give him or her the extra rest he or she needed. Certainly, if one is practicing differential relaxation 24 hours a day, a considerable amount of energy is saved. If one practices differential relaxation in situations that usually evoke stress or tension, suffering from tension-related problems can be dramatically decreased.

These are some of the essential points to get across to the beginning learner. To a large extent, the success or failure of the application depends on the learner's self-discipline—on his or her willingness to practice the method for the prescribed hour each day.

The Physical Environment and Equipment

The physical environment for teaching progressive relaxation can be varied. Groups have been taught in such places as gyms, dance studios, and classrooms. The learners should have something reasonably soft to lie on, such as a thick carpet, gym mats, blankets, or sleeping bags. In clinical treatment, individual rooms in relative quietude with cots, pillows, and blankets are provided. An adept clinician can treat several patients simultaneously, one in each individual room. But whatever the learning situation, no effort is made to eliminate external distractions completely. The goal is to learn to relax in a "normal" environment, which is usually somewhat noisy. Jacobson trained one of us (P. M. L.) in a hotel room on the third floor of a hotel overlooking a major thoroughfare and an exceptionally noisy construction site. The learner should anticipate and eliminate any possible distraction during relaxation practice and should cover the body with a blanket at the start to prevent chilling (with successful relaxation, the body temperature may fall noticeably through decreased metabolic rate).

EMG Confirmation of Progress

Ideally, the clinician obtains objective sEMG tension profiles as treatment progresses over the weeks. This is done to confirm any therapeutic observations of potential progress, especially reports of diminishing complaints from the patient. For illustration, abbreviated tension profiles for a nervous individual and for a relatively relaxed participant are presented in Table 7.2. This table indicates that the goal for a nervous individual is to achieve a tension profile typical of normotensives. One simplistic way of characterizing this application of progressive relaxation is that it may turn a "type A" individual into

TABLE 7.2. Tension Profiles of a "Nervous" versus a Normotensive ("Normal") Participant

Measures	Nervous subject	Normotensive subject
Brow EMG (μV)	8.68	2.95
Left-arm EMG (μV)	2.10	0.58
Tongue EMG (μV)	5.57	2.75
Right-arm EMG (μV)	3.10	0.59
Right-leg EMG (μV)	1.36	0.53
Pulse/minute	70	69
Respirations/minute	17	15
Blood pressure (mm Hg)	117/71	96/70

Note. Adapted from Jacobson (personal communication, 1978). EMG values are based on peak-to-peak measurement.

a "type B"; the person's excessively tense body regions then become relatively relaxed. Observation of the nervous participant with the naked eye would typically yield such traits as the following: wrinkled forehead, frown, darting eyes, exaggerated breathing, rapid pulse and respiration, and habits of fidgeting. But even the experienced clinical eye may not properly diagnose an excessively tense individual when such obvious symptoms are absent. EMG readings are required for proper diagnosis.

The Therapist–Client Relationship

We have emphasized that the clinical application of progressive relaxation minimizes suggestive effects to produce definitive and permanent, rather than fleeting, physiological changes—changes that differ from those that result from suggestion and the placebo effect. Thus the hypochondriac who continually seeks reassurance is not suggestively reassured. Instead, if the patient starts to discuss maladies during the instruction period, he or she is merely told to relax the relevant tensions away. The patient should become as independent of the clinician as possible. The teacher emphasizes that it is the learner who successfully eliminates the control signal, thus putting emphasis on the learner rather than the instructor. The clinician as teacher merely guides the learner. Progressive relaxation is a trial-and-error process that must be learned step by step, with moments of success and failure. Through this process, relaxation, like any other habit, can become permanent.

Description of the Method

Preparation

The psychological set with which the learner starts each practice period is critical. This is the period in which the patient is to do absolutely nothing at all but learn to relax. The session is planned in a room that is free from intrusion, so that practice may be continuous without interruption from telephones, doorbells, or people entering. Any unnecessary movement, such as getting up or fidgeting, is discouraged, because these added tensions retard progress. An hour-long practice period seems optimal because the individual progressively relaxes, achieving lower and lower intensity tensions throughout the body as the hour progresses. One of us (P. M. L.) has successfully taught the technique using half-hour sessions

Program Overview

The 1,030 or so skeletal muscles in the body are studied in groups. Another meaning of "progressive relaxation" is that an individual progressively relaxes regions of the body, progressing from one muscle group to the next in a specific order. The muscle groups progressively studied are first those in the arms, then those in the legs, followed by those in the trunk, neck, and eye region, and finally by those in the speech musculature. There are, for instance, six localized muscle groups in the left arm to be studied. For most people learning to control tension in the arms is easiest, providing the best chance of early success. Training then progresses from easier areas to ones that are more difficult for most people. Table 7.3 describes the practice program for the left and right arms.

Often, the amount of instruction covered can be concentrated, if necessary, with several parts of the body covered in a single session. Lehrer and Carr (1996) have written a manual for relaxation training across only seven sessions of training, in which instructions on several muscle groups are combined into a single session. In this case, the sessions could be as follows:

- Session 1: training the muscles of the arms, with instructions in differential relaxation
- Session 2: training the muscles of the legs, with instructions in differential relaxation
- Session 3: training muscles of the trunk, with instructions in differential relaxation
- Session 4: training muscles in the eye region of the face, with instructions in differential relaxation
- Session 5: training muscles in the speech region of the face, with instructions in differential relaxation
- Sessions 6–7: training in differential relaxation and strategic use in everyday life

TABLE 7.3. Practice Program for the Arms

Day	Left arm	Day	Right arm
1	Bend the hand back.	8	Bend the hand back.
2	Bend the hand forward.	9	Bend the hand forward.
3	Relax only.	10	Relax only.
4	Bend the arm at the elbow.	11	Bend the arm at the elbow.
5	Press the wrist down on books.	12	Press the wrist down on books.
6	Relax only.	13	Relax only.
7	Progressive tension and relaxation of the whole arm (general, residual tension).	14	Progressive tension and relaxation of the whole arm (general, residual tension).

Note. Practice is for one period each day, performing the indicated tension three times at intervals of several minutes. Then go negative for the remainder of each period. Thus, on day 1, bend the left hand back. On day 2, bend the left hand forward, and on day 3 do nothing at all. After 14 days, you are ready to go on to the leg. From McGuigan (1991a, p. 155), as adapted from Jacobson (1964). Copyright 1991 by F. J. McGuigan. Reprinted by permission of the author's estate.

Next we illustrate the use of progressive relaxation in its most intensive form, as described by Jacobson.

The First Practice Session

The learner starts by lying on a couch, bed, floor, or mat with arms alongside the body. Only one position is practiced each hour, the control signal being observed three times in each period (as specified in Table 7.3). The eyes are open for several minutes and then gradually allowed to close. (The specific amount of time that elapses is not important, as the learner should not concentrate on timing or anything else. Nor should the individual actively close the eyes, as that would be work; he or she simply allows the muscle fibers around the eyes to lengthen.) When the eyes have remained closed for another several minutes, the learner raises the hand at the wrist steadily, without fluctuation; there should be no seesawing or wiggling. While the hand is being bent back, the vague sensation in the upper surface of the left forearm, a "tightness," is the signal of tension—the control signal—that the individual is to learn to recognize. The learner holds the position for a minute or two, studying the tension sensation. Then, using the example of turning off the switch of a machine, the learner is instructed to "shut the power off" for a few minutes. Jacobson called this "going negative" (i.e., doing nothing). The individual does not *try* to relax because trying implies doing something and usually generates tension. He also used the "method of diminishing tensions" to help drive down the perceptual threshold. The learner is instructed to tense the muscles by gradually lesser amounts and to perceive the progressively diminishing sensations of tension. Then, after switching off completely, the learner is asked to perceive slight amounts of residual tension that may remain, and gradually to eliminate it by going negative.

Thus the learner studies each major muscle group by (1) identifying the control signal, and thereupon (2) relaxing the control signal away. The learner is not told where the control signal is but should find it for herself or himself. For example, when the instruction is to raise the hand vertically at the wrist, the learner comes to identify the control signal at the dorsal surface of the forearm. As the learner searches for the control signal, there may be some uncertainty about what is being sought. Some people identify the control signal immediately, whereas others have great difficulty. It is very subtle, and only a vague guess as to its location may be sufficient, but with repeated practice the control signal can become as obvious as a loud noise. Even the subtle tension sensations in the small muscles of the tongue and eyes can eventually be identified easily. If the learner cannot recognize tension sensations after repeated trials, the instructor may put resistance on the movement of the hand by pressing it down while that learner tries to bend it back from the wrist. This makes the sensations much more salient, so this method usually need only be used once or twice. People often remark on how subtle the control sensations are and how other sensations, such as strain in the wrist, are much more salient. The therapist reassures the learner that this is often the case and that perception of joint discomfort does tend to be much more easily perceived than muscle sensations.

During this initial learning phase, a number of irrelevant tensions may be identified, whereupon the instructor merely informs the learner that he or she is incorrect and searching must continue. Often the learner will erroneously report that the control signal is at the base of the wrist; this sensation, which is more prominent than the subtle control signal, is *strain*. Strain is the *result* of tension and is generated by receptor cells in the tendons and joints. That is, tension in the muscles pulls on the tendons and joints, so that tension causes strain, which is sometimes easier to perceive than muscle sensations. One cannot learn to run oneself by means of effects (strain), but only by causes (which are tension signals). Consequently, one must learn to control the body through the tension signals.

An effort to relax is a failure to relax. The process of relaxing is one in which an individual gives up the tension—just lets it go and allows the muscle fibers to elongate. Instructions that are synonymous to "relax the tension away" are to "let the power go off" and "go in the negative direction." It is to be emphasized that no work is required to allow "power off." All the learner needs to do is to discontinue working; no effort is required to follow the instruction, "Just don't bother to do anything at all." Untrained people often fail to relax because they work to relax. Typically, at first the learner works the hand down, which is merely adding tension. The key is that one cannot *try* to relax; all one does is to discontinue tensing. Instruction largely consists of preventing the beginner from doing the wrong thing—that is, making an effort to relax. For instance, when the muscle fibers in the dorsal surface of the forearm elongate, the hand simply collapses like a limp dishrag that is released. When successful, all sensations in the relaxed area cease because receptor cells in the muscles "shut off." Although the learner cannot cease activity of the eyes or the ears, the muscle fibers can be instructed to stop generating signals.

In Jacobson's more intensive method, only this single instruction is given in the first instructional session. This sequence is repeated two more times. At the end of the session the therapist reviews progress and other matters of interest to the patient, answering any questions raised. A program of practice can then be scheduled for the patient; for example, following Table 7.3, the individual can be instructed to practice for 14 days on the positions specified. At the end of 2 weeks, the patient may then be asked to return to go on to the next practice area. The precise schedule and length of time between visits to the therapist can vary, depending on the requirements of patient and therapist.

After the First Practice Period

In the second session, the learner bends the left hand forward instead of backward (see Table 7.3), and so on for 14 days. As in Jacobson (1964b) and McGuigan (1991a), the entire practice sequence while lying down is as follows: left arm, 7 days; right arm, 7 days; left leg, 10 days; right leg, 10 days; trunk, 10 days; neck, 6 days; eye region, 12 days; visualization, 9 days; speech region and speech imagery, 19 days. Instructions for these regions are given in Table 7.4.

The last region to be studied, the speech musculature, is critically important. As shown in Table 7.5, specific practice positions for the various parts of the speech musculature are presented in the first 8 days. Then the concluding days are spent in practicing developing speech imagery, so that the individual can learn to control the linguistic components of mental activities. In an analogous way, the individual learns to control the visual components of thoughts through eye control with imaginal practice of visual scenes (the "visualization" practice noted in the preceding paragraph).

After these positions are practiced lying down, they are practiced again in the same order, but in a sitting position. Repetition is the keynote of progressive relaxation.

Generalized and Localized Tension

In Table 7.3, days 7 and 14 call for practice with generalized, residual tensions. In addition to localized tension (that confined to specific muscle groups), the body also generates a more widespread kind of tension that is carried chronically throughout the skeletal

TABLE 7.4. Intermediate Instructions

Body region	Instructions
Legs (each separately)	1. Bend foot forward from the ankle, with toes pointing toward the head. Felt in the side of the shin. 2. Bend foot downward, raising the heel and pointing the toes down. Felt in the calf. 3. Raise your lower leg out, straightening the leg. This can easily be done lying on a couch, dangling your leg over the side of the couch. This is felt on top of the lap. 4. Bring your lower leg back, as if you were going to kick yourself in the rear. This also can most easily be done lying on a couch. This is felt in the thigh. 5. Press the upper leg down. It is sometimes helpful to put a few books under the thigh. This is felt in the buttock. 6. Raise the knee toward your chest. Although felt in the leg, the primary muscle action is in the lower abdomen, near the groin.
Neck	1. Bend the neck back, looking up. This is felt in the back of the neck. 2. Bend the neck forward, with the chin toward the chest. This is felt in the front of the neck, somewhat toward the sides. 3. With the nose pointing forward, bring the right ear toward the right shoulder. This is felt on the right side of the neck (with a stretching sensation sometimes on the left—not a control sensation). 4. Perform the same motion to the left, with control sensations felt on the left side of the neck.
Eye region (thinking becomes impossible with complete relaxation here)	1. Raise the eyebrows and wrinkle up the forehead. Felt throughout the forehead. 2. Frown with the eyebrows. Felt in the forehead above the nose. 3. Close the eyes tightly. Felt in the eyelids. 4. With the eyes closed, look to the left, without turning the head. Felt on the left side of the eyeball, with a faint stretching sensation (not a control sensation) on the right. 5. Do the same maneuver, looking to the right. 6. With the eyes closed, look up. This is felt on top of the eyeball. 7. With the eyes closed, look down. This is felt on the bottom of the eyeball, a little to the right and left side. 8. Imagine being at a tennis game, with eyes closed. Without deliberately moving your eyeballs, vividly imagine the ball going from the right to the left and back. Feel tension shift with the direction of the ball. 9. Imagine looking up at a high building. This is felt at the top of the eyeball. 10. Imagine looking down at the ground. This is felt on the bottom of the eyeball. 11. With eyes open, stare at an object in front of you. Feel tension all over the eyeball.
Trunk	1. Bring the shoulders up toward the ears. This is felt in the back of the neck and on top of the shoulders 2. Bring the shoulders back. This is felt between the shoulder blades. 3. Bring the right arm across the body pointing to the left. This is felt in the right chest. Then try this without moving the arm, just beginning to move the shoulder. 4. Repeat this instruction with the left arm. 5. Pull in the abdomen. This is felt throughout the abdominal area. 6. Breathe quietly. Feel the abdomen expand with each breath, and slight tension in the diaphragm area with each inhalation, totally relaxing with exhalation. 7. Arch the back. Feel tension in the lower back and columns of muscles on the sides of the back.
Relaxed breathing	Relax completely. Observe each breath going into the abdomen, back, groin area, and even the legs.

With each instruction, use the method of diminishing tensions, observe residual tension, and eliminate it by going negative. Note that the relaxed breathing instruction was not part of Jacobson's original instructions, but has been used by me (P. M. L.) to improve awareness of total relaxation of the abdomen, back, and upper legs.

TABLE 7.5. Speech Region Practice

Day	Instruction
1	Close your jaws somewhat firmly.
2	Open the jaws.
3	Relax only.
4	Show your teeth (as if smiling).
5	Pout.
6	Relax only.
7	Push your tongue forward against your teeth.
8	Pull your tongue backward.
9	Relax only.
10	Count out loud.
11	Count half as loudly.
12	Relax only.
13	Count very faintly.
14	Count imperceptibly.
15	Relax only.
16	Imagine that you are counting.
17	Imagine that you are saying the alphabet.
18	Relax only.
19	Imagine saying your name three times.
	Imagine saying your address three times.
	Imagine saying the name of the president three times.

Note. In Jacobson's preferred method, one position is to be practiced on each day, performing the indicated tension three times at intervals of several minutes. Then go negative for the remainder of each period. Thus, on day 1, close jaws somewhat firmly. On day 2, open jaws, and on day 3 do nothing at all. In practice, however, these instructions are often combined in a single session, including practice with the method of diminishing tensions and instructions to become aware of residual tension sensations. From McGuigan (1991a, p. 194), as adapted from Jacobson (1964). Copyright 1991 by F. J. McGuigan. Reprinted by permission of the author's estate.

musculature. This general tension is a residual tension in which there is fine, continuous contraction of the muscle fibers. Localized relaxation allows the individual to relax a particular group of muscles, but a different technique is required for general tension. An effective procedure for controlling this widespread phenomenon is very slightly, gradually, and uniformly to stiffen (tense) an entire limb for perhaps 30 seconds or a minute. The learner then studies this sensation, which is uncomfortable and insidious. General tension differs from local tension in another respect—namely, that it appears to have no useful purpose. After the learner studies the general tension for a brief period, the muscle is then gradually relaxed over an extended period of time. If the general tension is built up within the first 15 minutes or so of the period, the remainder of the hour may be spent reducing the general tension in the limb so that the learner gradually relaxes over an extended period of time. The learner also will usually experience some residual tension sensations after thinking that the muscles are completely relaxed. Becoming aware of this residual tension and allowing it to dissipate while doing *nothing* (because *doing* is a form of tension) is one of the critical components of Jacobson's technique and differentiates it from briefer methods of training. This procedure, with sufficient practice, can allow the individual to relax residual tension down to zero, which can be verified by objective EMG measurements. The untrained person is not directly aware of either localized or generalized tension.

A Learner Can Relax!

Sometimes a learner asserts that he or she *cannot* relax; the point is that the learner *did* not relax. To make the point vividly, the instructor can ask the learner to push down the wall of a room. The learner fails to do so, it can be further explained that such an act violates the laws of physics. On the other hand, elongating the muscle fibers does not violate the laws of physics. The fact that the person *did* not relax does not mean that the person *cannot* relax.

A learner who complains that he or she finds it hard to lie quietly at practice is confused about what is wanted, for there has never been an instruction to hold still *(holding* still is not relaxing). It is never "hard" to relax, for "hard" implies effort. If a learner stiffens when requested to relax, she or he is making a task of it, which is not relaxation but only an unsuccessful attempt. When the learner replaces a statement that "Relaxing makes me nervous" with the statement that "I am beginning to enjoy it," this indicates progress.

If Thoughts Occur

Some learners ask, "What shall I think about when I lie down?" They are instructed not to think, not to bother to *make* the mind blank, not to focus on anything. If they find themselves thinking about something, they should merely "let the power go off" in all the muscle regions on which they have practiced. If the thinking recurs, they should go negative again, no matter how often. But because they do not yet have control of all their muscles, they should not expect perfection; complete elimination of thoughts can come later if they are diligent.

Also, patients should not think frequently about their symptoms as they continue relaxation training. Signs of progress may appear soon after they start, or there may be a delay, depending on such matters as how tense they are and what everyday pressures they experience. The essential point is to practice daily; if they do that, they can expect improvement. Furthermore, they should be aware that learning relaxation skills is like learning any other performance skill: two steps forward and one step back, two steps forward and one step back.

Treatment Resistance, Adherence, and Maintenance of Behavior Change

Perhaps half of those individuals who take the first step in learning progressive relaxation go on to succeed in developing reasonably adequate control over their bodies, including their mental processes. This estimate covers a wide variety of potential learners, including patients who come to clinics; heterogeneous members of the community in evening classes; college, medical, and graduate students; and even professionals who participate in specialized workshops. To explain this difference in behavior would require a complex research project. Short of that, it can be said that some people have the discipline to see themselves through the program, whereas others simply do not. Clearly, the difference cannot be explained according to the need to relax, for many with the greatest need have shunned the opportunity immediately after requesting it. I (F. J. M.) recall the president of a successful company who came for treatment of a peptic ulcer. In our second session, he confessed that he had not practiced a single day since our first instruction period. His explanation was that he had spent his life giving other people ulcers and now he resented having to deal with one for himself. It was agreed that further "treatment" would be a waste of time for all concerned.

Healthy individuals seem to learn more quickly than do patients in distress. Those with neurotic disorders, for instance, are distracted, and their learning is thus prolonged. People who are engaged in cultist activities and fads are especially suggestible and dependent, which makes it more difficult for the teacher to get them to rely on themselves. They have many bizarre ideas that interfere with their learning to relax, and much time can be lost if the teacher chooses to argue with them. Similarly, people who have excessive faith in the clinician also generally fail to observe tensions for themselves and take longer to relax than do average individuals.

Such illustrations of resistance and failure to comply could be enumerated indefinitely, but the problem is not unique to progressive relaxation. In many spheres of life, people behave in a self-destructive fashion, even when they can verbalize the contingencies between their behavioral inadequacies and the consequences thereof. Failure to take prescribed medication for high blood pressure, drug (including alcohol) abuse, and smoking are examples of such self-destructive behaviors. There is often little anyone can do for individuals who engage in denial processes, such as the hospital nurse who asserted that her cigarette smoking was healthy for her. For those individuals who can verbalize the contingencies, perhaps there is some limited hope if they will at least try to discipline themselves in efforts to develop self-operations control—in other words, to enhance their "willpower" (McGuigan, 1991a).

On the other hand, many of those individuals who have dedicated themselves to daily practice and to regular instruction have made amazing progress. A wide variety of symptoms have been alleviated or eliminated, with lasting effects, as shown by follow-up testing (Jacobson, 1938a, 1970). The following is adapted from a classic illustrative case reported by Jacobson (1970).

CASE EXAMPLE: A CASE OF ANXIETY, EXHAUSTION, AND ACROPHOBIA

Complaints

An exceedingly busy middle-level manager, 32 years of age, was referred by a relative, a physician, for an anxiety state that included fears of heights and of dizziness, general uneasiness and irritability, and headache at the vertex and the occiput. He stated that he had been living at high tension all day long and in his opinion was "burned out" for life. Walking down the street or speaking at corporate meetings, he was suddenly beset by fears of dizziness, making him feel very uncomfortable. At times he felt as if he were about to faint, and because he had never fainted, he felt that this was "all ridiculous." "Fighting" the aforementioned symptoms had only made them worse; he had tried to reason matters out and had tried to relax, he had tried to get his mind off of himself, but all had been in vain. He was particularly concerned because when speaking at meetings about his areas of responsibility, he suddenly began to fear becoming dizzy. In consequence, he had been caused to avoid these meetings, delegating speaking responsibilities to a subordinate, feeling, however, that sometime he would return.

Onset

The onset had been 4 or 5 years previously, in the middle of a presentation, when confronted by a challenging and somewhat abusive superior. At the time, he suffered from pain in the left lower portion of his thorax, both subcardially and laterally. He successfully completed his presentation, however, and his recommendations were accepted by the group. The symptoms disappeared after the use of belladonna plasters and the administration of a tonic. But he had begun to feel anxious about one set of matters, and the anxiety turned gradually to others. His relative, the doctor, had suggested that if he would pay no attention to his symptoms, they would disappear. Accordingly, he had said to himself, "Get your mind off it. Forget it." This seemed to bring him relief for about 6 months. Then, however, the symptoms insidiously recurred.

Personal History

Among previous diseases had been a severe case of influenza 9 years previously. Two months prior to symptom recurrence, he suffered from cystitis following cystoscopy, and since then occasional blood cells had appeared in his urine. As a rule, he slept well, but there were exceptions of late when he continued to worry. He had been married for 9 years and had two children, both well. His parents were well, except that his mother had arthritis and worried a lot.

General Examination and Clinical Laboratory Tests

General examination disclosed a well-nourished man looking the age stated. His pulse was regular at the rate of 96 in the sitting posture, and his blood pressure was 130/96. His temperature was 98.6°F. He was 5 feet 7 inches tall and weighed 157 pounds. General examination revealed a somewhat pendulous abdominal wall but otherwise no significant findings, aside from an extremely lively foot-flexion reflex. Laboratory tests supplied by his relative indicated Wasserman and Kahn reactions negative; basal metabolism, –12; blood nonprotein nitrogen, 27 mg%; urea nitrogen, 15 mg%; blood sugar, 84 mg%; red blood count, 4,310,000; white count, 6,500; hemoglobin, 75% with a color index of 0.8. The differential count fell within normal limits. Fluoroscopy of the chest proved negative. Urinalysis was completely negative, aside from a few mucous threads.

Instruction Begun

Instruction in progressive relaxation was begun. At this time, over a 10-year period, the therapist was attempting to rule out any form of suggestive therapy, so far as this might be possible. To this end, I tried to avoid not only reassurance to the patient in any possible form but also unnecessary conversation. The therapist attempted to confine the relationship to instruction in the forms of progressive relaxation appropriate to the patient's needs in my judgment. Accordingly, when the patient asked questions bearing upon the outcome of the instruction, hoping that it might prove favorable, or when he was in doubt that the instruction really applied to his case, The therapist avoided answers that might possibly be interpreted by him suggestively. In reaction to this, the patient lost confidence after a month or two of instruction, suddenly leaving to go elsewhere for "psychiatric treatment." After 3 weeks of absence with little ado or comment on either side, he returned to complete his course.

Second month of treatment. He has been learning. He states that when emotionally disturbed he localizes the tension patterns and lets them go.

Eighth month of treatment. Instruction devoted to review of tension patterns in the eyelids and eyeballs. Practice is given in observing tension patterns and going negative upon looking in various directions with eyelids closed and looking from finger to finger with eyelids open.

Visualization Found Impossible during Complete Eye Muscle Relaxation

Visualization training. He is requested to imagine that he is seeing the fingers held about 10 feet away from his eyes, but at the same time to remain perfectly relaxed. He reports that he finds this instruction impossible to carry out. He finds it necessary to exert effort tensions of the eyes, that is, to look in specific directions, if he is to imagine objects seen. This request and report are typical of our endeavors to secure objective reports from highly trained patients, without leading them to anticipate the answers. Indeed, these autosensory observations with patients have been carried out under strictly controlled methods characteristic of laboratories of experimental psychology.

Autosensory Observation during Emotion

One week later. He mentions fear of being in a high building but fails to give a clear-cut sensory and imaginal report, evidently not yet prepared to describe his experiences, but losing himself in the emotion. He is given further instruction in observing and in reporting matters of visual imagination of indifferent affect.

The following weeks. He continues to be emotionally disturbed from time to time. Training is devoted to the musculature of speech, with particular reference to steady or static tensions.

Two months later. He states that today he has suffered from phobia of high places. Instruction is given in verbal imagination. Yet he fails to recognize tension patterns in the various muscles of speech and to state their locations.

Autosensory Observation during Unemotional Experiences

One week later. He enters the clinic exultant over his personal discovery that attention to the object of fear diminishes the fear. Instruction today begins in the lying posture with the request, "Think of infinity." He fails to report the imaginal, the tension, and other signal patterns, stating only the meaning of his reflection. However, when requested to engage in simple multiplication, as of 14 by 42, he gives a complete report, not only of the meaning but also of the tension pattern. He is given repeated practice in observation of sensory experiences, distinguishing the report thereof from the interpretation or meaning.

Anger Tension Relaxed

He announces that he now relaxes spells of anger and has been gaining in weight; and, as noted by his family physician, he no longer fidgets as he did formerly upon receiving a hypodermic injection. Upon being requested to describe an experience of phobia, he still engages in the “stimulus error” but to a lesser extent than formerly.

One month later. Differential relaxation is begun, with training devoted to the right arm. He has been at home with a fever of 101°F at times, due to an infection of the right kidney and of the bladder.

Instruction during the 4 following weeks. Instruction on the limbs is continued in the sitting posture. At times during the period while sitting, he is on the verge of sleep.

Efficiency

At the end of this time, he asserts that now he does his work effectively and without nervous excitement in a manner which had never previously been possible.

One week later. He complains of tension in the right side of the scalp, mistaking pull or strain on the scalp for muscular tension. After drill in wrinkling of the forehead, he reports relief.

One week later. Instruction is devoted to the abdomen and back in the sitting posture.

One month later. He affirms that his nerves are greatly improved. At times a little phobia persists, but he is becoming accustomed to observing tension patterns at such moments and to relax them. His relative, the doctor, recently was amazed at his calmness in public speaking, in contrast with his extreme excitability in former years. Friends who do not know of the instruction which he has been receiving spontaneously say to him that he is a changed man.

Phobic Tension Patterns Now Recognized and Relaxed

The following 2 weeks. He states that he has been in very good nervous condition and has been efficient. He makes it a rule to relax any phobia or disturbing thought-act. Instruction concerns brow tensions. He often sleeps during the period.

The following 4 weeks, sequentially. Repeated practice is given on imagining falling objects of indifferent affect. The purpose here is to teach him to observe the pattern of mental processes when any object falls. Thus a first step is taken in his learning to observe what he does in high places when he imagines himself jumping and falling. Thus, without telling him, we proceed in the treatment of acrophobia.

Return of Ability to Present at Meetings

Six weekly sessions, beginning 2 months later. For the first time in years, he speaks in public without nervous difficulties. His phobia at meetings has disappeared. Instruction is devoted to musculature of speech.

Return of Ability to Drive and to Go on Trips; No Acrophobia

One week later. He reports that for the first time in about 7 years he has taken a long drive. Previously fearful, but not knowing why, he now finds that he enjoys driving. Before present instruction, he dreaded and avoided speaking outside of his city. Recently, in contrast, he has gone on three long trips with no difficulty. Nowadays, he travels enjoyably, whereas previously he did it with dread. The phobia of jumping from high places has been completely absent. Last month was a very difficult time in his business, because large loans for which he was responsible came due at the height of the panic and bank failures of the Great Recession. Nevertheless, he was relaxed and not nervous. He adds that he relaxes concerns about business meetings. He no longer mentally argues business strategies, but he instead “relaxes the whole thought.” Instruction is devoted to tension signals from the tongue and relaxation of tongue muscles.

The following 3 weeks. Instruction concerns imagining making various statements. He is still slow to relax to the point at which he is ready to observe and report. However, his reports are clear.

One month later. Instruction is begun relaxing the left arm as much as possible while reading.

Nine successive sessions. Instruction on reading is continued, while observing tension patterns and learning to relax muscles not required for reading. He fails to report verbal tensions during reading, claiming that there are none, but only visual images and ocular tension patterns. Obviously, his training is not yet sufficient, but he is not so informed.

One month later. His brother-in-law died suddenly. Instruction is devoted to exposing a single printed stimulus word within the instruction to read and get the meaning. At first his reports are confused: He gives interpretations in place of observations. Finally, he begins to report more accurately, as follows: “Ocular tension in seeing the word. Ocular tension in imagining what the word indicates. Tensions in the tongue as in saying the word.”

Slight Relapse, Yet Patient Not Told What to Look For

One week later. Yesterday he experienced some nervous distress at a business meeting. Upon relaxing, the unnecessary emotions disappeared. He has been through various trying ordeals. For 2 months he has failed to report tensions in the organs of speech while reading. Accordingly, he is requested to count while reading, employing the method of diminishing tensions. Finally, he succeeds in observing the speech tensions. This occurs with no hint from the instructor that he has been omitting anything.

Relaxes Differentially

One week later. There have been four deaths in the family, which ordinarily would have disturbed him much more, he believes. However, in place of engaging in fears and worries each morning, now he goes to work relaxing and preparing himself to relax during the day. He has found that he does not have to postpone relaxation to the weekend but can relax during the work of each day. Accordingly, he is greatly relieved.

Discharged

The following 2 weeks. Instruction is given in reading aloud, relaxing as much as possible during this occupation. He is discharged, apparently free from emotional disturbance. Electrical recording has shown excellent technique.

Freedom from Acrophobia

Following this patient’s discharge, the phobia of high places had so evidently disappeared that, without suggestion from the doctor, he took a position with an office in the tower of a very high office building. In his new office, he felt no fear or phobia. Regarding his nervous condition, he considered himself well. He practices differential relaxation every day. On some days he fails to lie down, but as a rule, after dinner he practices in a lying posture for 45 to 90 minutes. On about half of these occasions he falls asleep. If he awakens at night and finds himself tense, he locates the tension patterns and relaxes accordingly. Uneasiness no longer is experienced, and he no longer shows marked irritability. When engaged in difficult matters, he relaxes to avoid becoming irritated. He has no dizziness, no fears, and no headaches. His technique shows excellent form.

Two years later. Electrical recording is performed with the eyes open. The right thigh averages a little over 0.5 μV. The right biceps averages a little over 0.075 μV with little variability. The right jaw muscles average about 0.2 μV. The eye regions average about 3.5 μV. The values are fair for a trained individual except that variability is a little high. (Note that Jacobson's instrument measured voltage in peak-to-peak values rather than in the lower root mean square (r.m.s.) values used in most current recording equipment.)

Five years later. He reports that for the first time in his life, he has completely lost fear of being in high places. He flew in an airplane and at first was fearful when breathing through tubes of oxygen provided for this purpose, but he used his relaxation technique and enjoyed the flying trip from then on. He relaxes differentially to an extent that is readily appreciated by a professional observer.

Persistent Freedom from Anxiety Tension, Although Difficulties Remain

Five years afterward (10 years after discharge). He relates that he was "wonderfully well" for many years following the instruction. Ten years previously his daughter married, but the next year she became very unhappy, which was a "terrific jolt" to him. "During that whole year I suffered from worry, hoping that the situation would straighten out." His daughter was operated on for appendicitis and recovered. She joined him for a few weeks only, after which she returned home, because her husband needed her. "This," continued the patient, "took quite a bit out of me. Then my son was drafted for the war; usually parents worry about that. That year, loose [bowel] movements had occurred once or twice a year and I didn't know what started them. Certain medication overcame any spell promptly. Also, I could avert the spells by avoiding raw fruits and vegetables. Five years later the spells became more intense and frequent. My brother-in-law got me into a business of his own, in which I sunk time and a small fortune. Two years later I foolishly let him leave the partnership. Thereafter he enticed the plant manager away, which proved irritating and took my time and interfered with my full-time position. Engineers called in to help the business gave bad counsel with resulting further loss. Later that year I sold the business for a pittance."

Irritable Colon

During the period of gastrointestinal distress, a severe spell of diarrhea was followed by X-ray examination and a diagnosis of duodenal ulcer. The symptoms disappeared after a short period of diet restriction and medication. Since then, there have been only two light spells and one severe spell with burning in the rectum.

Practice Neglected

"It was in the attempt to get rid of nervous disturbance that I sold the plant and returned to more or less regular periods of relaxation, which I had been neglecting, and to which

I ascribed alone my lasting improvement. My downfall had been that practice was not regular."

Refresher, 10 years after discharge. A brief refresher course is begun.

First week. Roentgenological examination indicates negative findings as to duodenal ulcer. The colon, however, is found to be spastic. A similar examination made previously suggested that possibly there had been an early ulcer. Currently, however, he has been relaxing a little more frequently than before and has been able to partake once more of solids.

Fourteen weekly sessions. Review practice is given on limb tension patterns and relaxation.

Ten years after the refresher. His wife writes from her observations in retrospect that through relaxation training he had learned "a new way of life."

The following year. He reports that he has been free from anxiety tension, phobias, fears, and other emotional disturbances these many years. He can speak at meetings and public forums where and when he desires and has continued to be generally more relaxed than he had ever been in the decade before he reached the age of 32, when instruction had begun.

COMMENTS

The contemporary scene has been flooded with stress management procedures, varying from thinking certain colors while breathing through one nostril to repetitiously talking to oneself. There are a variety of muscular relaxation procedures, too, that are generally acknowledged as having flowed from progressive relaxation. In the last analysis, which methods are most effective must be empirically determined. The primary criterion for assessing the effectiveness of a stress management procedure that purports to teach a person to relax is the extent to which the individual can selectively allow his or her muscle fibers to elongate on command—that is, to relax differentially in the face of stress. Once the person can thereby exercise self-operations control, he or she can more rationally deal with the stressful situation instead of just reflexively responding to it.

Edmund Jacobson spent over seven decades collecting data documenting the effectiveness of progressive relaxation clinically and scientifically, including the use of EMG. We should all be indebted to him for giving the world progressive relaxation, the grandfather of all relaxation methods. We can think of no better way to close this chapter than to quote Jacobson as follows:

> Until I see proof I incline in the direction of skepticism. Progressive relaxation, as developed in our laboratory and clinic, was to me a matter of skepticism at every step. Thirty years ago, as I went from room to room trying to get individuals with different maladies to relax, I recall saying to myself, "What kind of nonsense is this that you are practicing?" The careful accumulation of data has vindicated the procedure. (1977, p. 123)

ACKNOWLEDGMENTS

We are deeply indebted to Edmund Jacobson for so many things that it would be impossible to list them all here. First and foremost, though, we would specify the innumerable hours and limitless energy that he spent in personally training both of us in progressive relaxation. More specifically for this chapter, we are indebted to him for the ideas, principles, and applications and, in fact, often even for the use of his words, which have become part of our own repertoire.

NOTES

1. This is an update of F. J. McGuigan's chapter for earlier editions of this book. Dr. McGuigan, who passed away in 1998, was a major contributor to research on progressive relaxation and to the relationship between muscular activity and psychological processes. He was Edmund Jacobson's most articulate and longest-term student. He devoted much of his professional life to teaching Jacobson's method and performing research studies on various correlates of Jacobson's theory. As someone who also had the privilege of working under Dr. Jacobson's tutelage, I (P. M. L.) have undertaken a revision of this chapter. This is done with great humility, because the original work was quite definitive. Updates include a review of more recent research literature, as well as some minor editorial changes.

2. In my own (F. J. M.) experience, one day after some years of training, Jacobson instructed me to recall the most terrible event in my life. My reaction, like the predormescent start, was that my legs involuntarily flew directly into the air. My recollection was of a horrible event involving the use of my legs on the brakes of an automobile. (Later, I asked Jacobson whether that was a normal reaction, but apparently, I was being used as a guinea pig, because he said he had never given that instruction before.)

REFERENCES

Akmese, Z. B., & Oran, N. T. (2014). Effects of progressive muscle relaxation exercises accompanied by music on low back pain and quality of life during pregnancy. *Journal of Midwifery and Women's Health, 59*(5), 503–509.

Alarcon, R. D., Jenkins, C. S., Heestand, D. E., Scott, L. K., & Cantor, L. (1982). The effectiveness of progressive relaxation in chronic hemodialysis patients. *Journal of Chronic Diseases, 35*(10), 797–802.

Anagnostou, E., Zachou, A., Breza, M., Kladi, A., Karadima, G., & Koutsis, G. (2019). Disentangling balance impairments in spinal and bulbar muscular atrophy. *Neuroscience Letters, 705,* 94–98.

Arena, J. G., Bruno, G. M., Hannah, S. L., & Meador, K. J. (1995). A comparison of frontal electromyographic biofeedback training, trapezius electromyographic biofeedback training, and progressive muscle relaxation therapy in the treatment of tension headache. *Headache, 35*(7), 411–419.

Baider, L., Uziely, B., & Kaplan De-Nour, A. (1994). Progressive muscle relaxation and guided imagery in cancer patients. *General Hospital Psychiatry, 16*(5), 340–347.

Bain, A. (1855). *The senses and the intellect.* London: Parker.

Beiman, I., Graham, L. E., & Ciminero, A. R. (1978). Self-control progressive relaxation training as an alternative nonpharmacological treatment for essential hypertension: Therapeutic effects in the natural environment. *Behaviour Research and Therapy, 16*(5), 371–375.

Beiman, I., Israel, E., & Johnson, S. A. (1978). During training and post-training effects of live and taped extended progressive relaxation and electromyogram biofeedback. *Jounal of Consulting and Clinical Psychology, 46,* 314–321.

Bell, C. (1834). *The hand: Its mechanism and vital endowments as evincing design* (2nd ed.). London: William Pickering.

Bernhaut, M., Gellhorn, E., & Rasmussen, A. T. (1953). Experimental contributions to the problem of consciousness. *Journal of Neurophysiology, 16,* 21–35.

Bernstein, D. A., & Borkovec, T. D. (1973). *Progressive relaxation training: A manual for the helping professions.* Champaign, IL: Research Press.

Blanchard, E. B., Greene, B., Scharff, L., & Schwarz-McMorris, S. P. (1993). Relaxation training as a treatment for irritable bowel syndrome. *Biofeedback and Self-Regulation, 18*(3), 125–132.

Borkovec, T. D., & Weerts, T. C. (1976). Effects of progressive relaxation on sleep disturbance: An electroencephalographic evaluation. *Psychosomatic Medicine, 38*(3), 173–180.

Braud, L. W. (1978). The effects of frontal EMG biofeedback and progressive relaxation upon hyperactivity and its behavioral concomitants. *Biofeedback and Self-Regulation, 3*(1), 69–89.

Broota, A., & Dhir, R. (1990). Efficacy of two relaxation techniques in depression. *Journal of Personality and Clinical Studies, 6*(1), 83–90.

Brower, R. G. (2009). Consequences of bed rest. *Critical Care Medicine, 37*(Suppl. 10), S422–428.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton-Century.

Canter, A., Kondo, C. Y., & Knott, J. R. (1975). A comparison of EMG feedback and progressive muscle relaxation training in anxiety neurosis. *British Journal of Psychiatry, 127,* 470–477.

Carrington, P., Collings, G. H., Jr., Benson, H., Robinson, H., Wood, L. W., Lehrer, P. M., et al. (1980). The use of meditation-relaxation techniques for the management of stress in a working population. *Journal of Occupational Medicine, 22,* 211–231.

Carroll, D., & Seers, K. (1998). Relaxation for the relief of chronic pain: A systematic review. *Journal of Advanced Nursing, 27*(3), 476–487.

Chen, Y. L., & Francis, A. J. (2010). Relaxation and imagery for chronic, nonmalignant pain: Effects on pain symptoms, quality of life, and mental health. *Pain Management Nursing, 11*(3), 159–168.

Cheung, Y. L., Molassiotis, A., & Chang, A. M. (2003). The effect of progressive muscle relaxation training on anxiety and quality of life after stoma surgery in colorectal cancer patients. *Psychooncology, 12*(3), 254–266.

Christie, N. H. (2008). Surface EMG home trainers or progressive relaxation training: Comparing home practice interventions for the treatment of coexisting migraine and tension-type pediatric headache. *Dissertation Abstracts International: Section B. The Sciences and Engineering, 68*(11-B), 7700.

Coppieters, I., Cagnie, B., Nijs, J., van Oosterwijck, J., Danneels, L., De Pauw, R., et al. (2016). Effects of stress and relaxation on central pain modulation in chronic whiplash and fibromyalgia patients compared to healthy controls. *Pain Physician, 19*(3), 119–130.

Corbett, K. W. (1982). A comparison of EMG biofeedback and progressive relaxation used in the treatment of chronic pain. *Dissertation Abstracts International, 42*(11-B), 4573.

Davidson, D. M., Winchester, M. A., Taylor, C. B., Alderman, E. A., & Ingels, N. B., Jr. (1979). Effects of relaxation therapy on cardiac performance and sympathetic activity in patients with organic heart disease. *Psychosomatic Medicine, 41,* 303–309.

Davis, J. O. (1991). Sports injuries and stress management: An opportunity for research. *Sport Psychologist, 5*(2), 175–182.

Dayapoglu, N., & Tan, M. (2012). Evaluation of the effect of progressive relaxation exercises on fatigue and sleep quality in patients with multiple sclerosis. *Journal of Alternative and Complementary Medicine, 18*(10), 983–987.

De Berry, S. (1979). The effect of EMG biofeedback, progressive muscle relaxation and cognitive style on muscle tension and anxiety. *Dissertation Abstracts International, 40*(3-B), 1357–1358.

De Berry, S. (1981). An evaluation of progressive muscle relaxation on stress-related symptoms in a geriatric population. *International Journal of Aging and Human Development, 14*(4), 255–269.

de Paula, A. A., de Carvalho, E. C., & dos Santos, C. B. (2002). The use of the "progressive muscle relaxation" technique for pain relief in gynecology and obstetrics. *Revista Latino-Americana de Enfermagem, 10*(5), 654–659.

Dehghan-Nayeri, N., & Adib-Hajbaghery, M. (2011). Effects of progressive relaxation on anxiety and quality of life in female students: A non-randomized controlled trial. *Complementary Therapies in Medicine, 19*(4), 194–200.

Delius, W., Hagbarth, K. E., Hongell, A., & Wallin, B. G. (1972). General characteristics of sympathetic activity in human muscle nerves. *Acta Physiologica Scandinavica, 84,* 65B81.

Emmen, H. H., & Passchier, J. (1987). Treatment of headache among children by progressive relaxation. *Cephalalgia, 7*(Suppl. 6), 387–389.

Engel, J. M., Jensen, M. P., & Schwartz, L. (2004). Outcome of biofeedback-assisted relaxation for pain in adults with cerebral palsy: Preliminary findings. *Applied Psychophysiology and Biofeedback, 29*(2), 135–140.

Engel, J. M., Rapoff, M. A., & Pressman, A. R. (1992). Long-term follow-up of relaxation training for pediatric headache disorders. *Headache, 32*(3), 152–156.

Feely, J. M. (1978). The effects of electromyographic biofeedback training and electromyographic biofeedback with progressive relaxation training on narcotic addicts during medical detoxification. *Dissertation Abstracts International, 39*(3-B), 1474–1475.

Fey, S. G., & Lindholm, E. (1978). Biofeedback and progressive relaxation: Effects on systolic and diastolic blood pressure and heart rate. *Psychophysiology, 15*(3), 239–247.

Freedman, R., & Papsdorf, J. D. (1976). Biofeedback and progressive relaxation treatment of sleep-onset insomnia: A controlled, all-night investigation. *Biofeedback and Self-Regulation, 1*(3), 253–271.

Gada, M. (1984). A comparative study of efficacy of EMG bio-feedback and progressive muscular relaxation in tension headache. *Indian Journal of Psychiatry, 26*(2), 121–127.

Garver, D. L., Davis, D. M., Dekirmenjian, H., Ericksen, S., Gosenfeld, L., & Haraszti, J. (1976). Dystonic reactions following neuroleptics: Time course and proposed mechanisms. *Psychopharmacologia, 47*(2), 199–201.

Gay, M.-C., Philippot, P., & Luminet, O. (2002). Differential effectiveness of psychological interventions for reducing osteoarthritis pain: A comparison of Erickson hypnosis and Jacobson relaxation. *European Journal of Pain, 6,* 1–16.

Gellhorn, E. (1958a). The influence of curare on hypothalamic excitability and the electroencephalogram. *Electroencephalography and Clinical Neurophysiology, 10*(4), 697–703.

Gellhorn, E. (1958b). The physiological basis of neuromuscular relaxation. *Archives of Internal Medicine, 102,* 392–399.

Gellhorn, E., & Kiely, W. F. (1972). Mystical states of consciousness: Neurophysiological and clinical aspects. *Journal of Nervous and Mental Disease, 154,* 399–405.

Graffam, S., & Johnson, A. (1987). A comparison of two relaxation strategies for the relief of pain and its distress. *Journal of Pain and Symptom Management, 2*(4), 229–231.

Grassi, C., & Passatore, M. (1988). Action of the sympathetic system on skeletal muscle. *Italian Journal of Neurological Sciences, 9,* 23–28.

Greeff, A. P., & Conradie, W. S. (1998). Use of progressive relaxation training for chronic alcoholics with insomnia. *Psychological Reports, 82*(2), 407–412.

Grishman, A. (1989). Musicians' performance anxiety: The effectiveness of modified progressive muscle relaxation in reducing physiological, cognitive, and behavioral symptoms of anxiety. *Dissertation Abstracts International, 50*(6-B), 2622.

Gustafson, R. (1992). Treating insomnia with a self-administered muscle relaxation training program: A follow-up. *Psychological Reports, 70*(1), 124–126.

Hall, E. G., & Erffmeyer, E. S. (1983). The effect of visuo-motor behavior rehearsal with videotaped modeling on free throw accuracy of intercollegiate female basketball players. *Journal of Sport Psychology, 5*(3), 343–346.

Han, I.-S., & Son, C. (1987). The relative effects of EMG biofeedback and progressive muscle relaxation training on tension headache. *Korean Journal of Psychology, 6*(1), 10–20.

Haney, C. J. (2004). Stress-management interventions for female athletes: Relaxation and cognitive restructuring. *International Journal of Sport Psychology, 35*(2), 109–118.

Heide, F. J., & Borkovec, T. D. (1984). Relaxation-induced anxiety: Mechanisms and theoretical implications. *Behaviour Research and Therapy, 22,* 1–12.

Henderson, J. C. (1991). The effect of progressive relaxation training on stress perception, dispositional optimism, and frequency and severity of running-related injuries. *Dissertation Abstracts International, 52*(4-A), 1253.

Hippocrates. (1849). *The genuine works of Hippocrates* (F. Adams, Ed., & Trans.). London: Sydenham Society.

Holt, E. B. (1937). Materialism and the criterion of the psychic. *Psychological Review, 44,* 33–53.

Jablon, S. L., Naliboff, B. D., Gilmore, S. L., & Rosenthal, M. J. (1997). Effects of relaxation training on glucose tolerance and diabetic control in type II diabetes. *Applied Psychophysiology and Biofeedback, 22*(3), 155–169.

Jacobson, E. (1925). Voluntary relaxation of the esophagus. *American Journal of Physiology, 72,* 387–394.

Jacobson, E. (1930a). Electrical measurements of neuromuscular states during mental activities: I. Imagination of movement involving skeletal muscle. *American Journal of Physiology, 91*(2), 567–608.

Jacobson, E. (1930b). Electrical measurements of neuromuscular states during mental activities: II. Imagination and recollection of various muscular acts. *American Journal of Physiology, 94*(1), 22–34.

Jacobson, E. (1930c). Electrical measurements of neuromuscular states during mental activities: III. Visual imagination and recollection. *American Journal of Physiology, 95*(3), 694–702.

Jacobson, E. (1930d). Electrical measurements of neuromuscular states during mental activities: IV. Evidence of contraction of specific muscles during imagination. *American Journal of Physiology, 95*(3), 703–712.

Jacobson, E. (1931). Electrical measurements of neuromuscular states during mental activities. VII. Imagination, recollection and abstract thinking involving the speech musculature.

American Journal of Physiology, 97(1), 200–209.

Jacobson, E. (1934). Electrical measurements concerning muscular contraction (tonus) and the cultivation of relaxation in man: Relaxation times of individuals. *American Journal of Physiology, 108*(3), 573–580.

Jacobson, E. (1936). The course of relaxation in muscles of athletes. *American Journal of Psychology, 48,* 98–108.

Jacobson, E. (1938a). *Progressive relaxation* (2nd ed.). Chicago: University of Chicago Press.

Jacobson, E. (1938b). *You can sleep well.* New York: Whittlesey House.

Jacobson, E. (1940). The direct measurement of nervous and muscular states with the integrating neurovoltmeter (action potential-integrator). *American Journal of Psychiatry, 97,* 513–523.

Jacobson, E. (1964a). *Anxiety and tension control: A physiologic approach.* Philadelphia: Lippincott.

Jacobson, E. (1964b). *Self-operations control: A manual of tension control.* Chicago: National Foundation for Progressive Relaxation.

Jacobson, E. (1967). *Biology of emotions.* Springfield, IL: Charles C Thomas.

Jacobson, E. (1970). *Modern treatment of tense patients.* Springfield, IL: Charles C Thomas.

Jacobson, E. (1977). The origins and development of progressive relaxation. *Journal of Behavior Therapy and Experimental Psychiatry, 8,* 119–123.

Jacobson, E. (1978). *You must relax* (4th ed.). New York: McGraw-Hill.

Jacobson, E. (1982). *The human mind: A physiological clarification.* Springfield, IL: Charles C Thomas.

James, W. (1884). What is an emotion? *Mind, 9,* 188–205.

Johnson, J. E. (1993). Progressive relaxation and the sleep of older men and women. *Journal of Community Health Nursing, 10*(1), 31–38.

Kang, D. H., McArdle, T., Park, N. J., Weaver, M. T., Smith, B., & Carpenter, J. (2011). Dose effects of relaxation practice on immune responses in women newly diagnosed with breast cancer: An exploratory study. *Oncology Nursing Forum, 38*(3), E240–E252.

Karbandi, S., Hosseini, S. M., Masoudi, R., Hosseini, S. A., Sadeghi, F., & Moghaddam, M. H. (2015). Recognition of the efficacy of relaxation program on sleep quality of mothers with premature infants. *Journal of Education and Health Promotion, 4,* 97.

Kienbaum, P., Karlssonn, T., Sverrisdottir, Y. B., Elam, M., & Wallin, B. G. (2001). Two sites for modulation of human sympathetic activity by arterial baroreceptors? *Journal of Physiology, 531,* 861–869.

Kim, K. J., Na, Y. K., & Hong, H. S. (2016). Effects of progressive muscle relaxation therapy in colorectal cancer patients. *Western Journal of Nursing Research, 38*(8), 959–973.

Klein, S. A., & Deffenbacher, J. L. (1977). Relaxation and exercise for hyperactive impulsive children. *Perceptual and Motor Skills, 45*(3, Pt. 2), 1159–1162.

Kroner-Herwig, B., Plump, U., & Pothmann, R. (1992). [Progressive relaxation and EMG biofeedback in the treatment of chronic headache in children. Results of an explorative study.]. *Der Schmerz, 6*(2), 121–127.

Kumaraiah, V. (1980). EMG biofeedback and progressive muscular relaxation in treatment of tension headache. *Indian Journal of Clinical Psychology, 7*(1), 1–5.

Lammers, C. A., Naliboff, B. D., & Straatmeyer, A. J. (1984). The effects of progressive relaxation on stress and diabetic control. *Behaviour Research and Therapy, 22*(6), 641–650.

Landelle, C., El Amadi, A., & Kavounoudias, A. (2018). Age-related impairment of hand movement perception based on muscle proprioception and touch, *Neuroscience, 381,* 91–104.

Lazarus, A. A., & Mayne, T. J. (1990). Relaxation: Some limitations, side effects, and proposed solutions. *Psychotherapy, 27,* 261–266.

Leboeuf, A., & Lodge, J. (1980). A comparison of frontalis EMG feedback training and progressive relaxation in the treatment of chronic anxiety. *British Journal of Psychiatry, 137,* 279–284.

Lehrer, P. M. (1978). Psychophysiological effects of progressive relaxation in anxiety neurotic patients and of progressive relaxation and alpha feedback in nonpatients. *Journal of Consulting and Clinical Psychology, 46,* 389–404.

Lehrer, P. M. (1982). How to relax and how not to relax: A reevaluation of the work of Edmund Jacobson. *Behaviour Research and Therapy, 20,* 417–428.

Lehrer, P. M., Atthowe, J. M., & Weber, E. S. (1980). Effects of progressive relaxation and autogenic training on anxiety and physiological measures, with some data on hypnotizability. In F. J. McGuigan, W. Sime, & J. M. Wallace (Eds.), *Stress and tension control* (pp. 171–184). New York: Plenum Press.

Lehrer, P. M., Batey, D. M., Woolfolk, R. L., Remde, A., & Garlick, T. (1988). The effect of repeated tense–release sequences on EMG and self-report of muscle tension: An evaluation of Jacobsonian and post-Jacobsonian assumptions about progressive relaxation. *Psychophysiology, 25,* 562–569.

Lehrer, P. M., & Carr, R. (1996). Progressive relaxation. In W. T. Roth (Ed.), *Treating anxiety disorders* (pp. 83–116). San Francisco: Jossey-Bass.

Lehrer, P. M., Hochron, S. M., McCann, B., Swartzman, L., & Reba, P. (1986). Relaxation decreases large-airway but not small-airway asthma. *Journal of Psychosomatic Research, 30*(1), 13–25.

Lehrer, P. M., Schoicket, S., Carrington, P., & Woolfolk, R. L. (1980). Psychophysiological and cognitive responses to stressful stimuli in subjects practicing progressive relaxation and clinically standardized meditation. *Behavior Research and Therapy, 18,* 293–303.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Lu, S.-E., Scardella, A., Siddique, M., et al. (2004). Biofeedback treatment for asthma. *Chest, 126,* 352–361.

Lehrer, P. M., Woolfolk, R. L., & Goldman, N. (1986). Progressive relaxation then and now: Does change always mean progress? In R. J. Davidson, G. E. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation* (Vol. 4, pp. 183–216). New York: Plenum Press.

Ley, R. (1988). Panic attacks during relaxation and relaxation-induced anxiety: A hyperventilation interpretation. *Journal of Behavior Therapy and Experimental Psychiatry, 19*(4), 253–259.

Lichstein, K. L., Peterson, B. A., Riedel, B. W., Means, M. K., Epperson, M. T., & Aguillard, R. N. (1999). Relaxation to assist sleep medication withdrawal. *Behavior Modification, 23*(3), 379–402.

Lowe, B., Breining, K., Wilke, S., Wellmann, R., Zipfel, S., & Eich, W. (2002). Quantitative and qualitative effects of Feldenkrais, progressive muscle relaxation, and standard medical treatment in patients after acute myocardial infarction. *Psychotherapy Research, 12*(2), 179–191.

Manzoni, G. M., Pagnini, F., Castelnuovo, G., & Molinari, E. (2008). Relaxation training for anxiety: A ten-years systematic review with meta-analysis. *BMC Psychiatry, 8,* 41.

Masoudi, R., Sharifi Faradonbeh, A., Mobasheri, M., & Moghadasi, J. (2013). Evaluating the effectiveness of using a progressive muscle relaxation technique in reducing the pain of multiple sclerosis patients. *Journal of Musculoskeletal Pain, 21*(4), 350–357.

McCann, B. S., Woolfolk, R. L., & Lehrer, P. M. (1987). Specificity in response to treatment: A study of interpersonal anxiety. *Behaviour Research and Therapy, 25,* 129–136.

McCloughan, L. J., Hanrahan, S. J., Anderson, R., & Halson, S. R. (2016). Psychological recovery: Progressive muscle relaxation (PMR), anxiety, and sleep in dancers. *Performance Enhancement and Health, 4,* 12–17.

McGlynn, F. D., Moore, P. M., Lawyer, S., & Karg, R. (1999). Relaxation training inhibits fear and arousal during in vivo exposure to phobia-cue stimuli. *Journal of Behavior Therapy and Experimental Psychiatry, 30*(3), 155–168.

McGuigan, F. J. (1978). *Cognitive psychophysiology: Principles of covert behavior.* Englewood Cliffs, NJ: Prentice-Hall.

McGuigan, F. J. (1989). Managing internal cognitive and external environmental stresses through progressive relaxation. In F. J. McGuigan, W. Sime, & J. M. Wallace (Eds.), *Stress and tension control: 3. Stress management* (pp. 3–11). New York: Plenum Press.

McGuigan, F. J. (1991a). *Calm down: A guide for stress and tension control* (rev. ed.). Dubuque, IA: Kendall/Hunt.

McGuigan, F. J. (1991b). Control of normal and pathological cognitive functions through neuromuscular circuits: Applications of principles of progressive relaxation. In J. G. Carlson & A. R. Seifert (Eds.), *International perspectives on self-regulation and health* (pp. 121–131). New York: Plenum Press.

McGuigan, F. J., Culver, V. L., & Kendler, T. S. (1971). Covert behavior as a direct electromyographic measure of mediating responses. *Conditional Reflex, 6,* 145–152.

McGuigan, F. J., & Dollins, A. B. (1989). Patterns of covert speech behavior and phonetic coding. *Pavlovian Journal of Biological Science, 24,* 19–26.

McGuigan, F. J., & Pavek, G. V. (1972). On the psychophysiological identification of covert nonoral language processes. *Journal of Experimental Psychology, 92,* 237–245.

McGuigan, F. J., & Winstead, C. L., Jr. (1974). Discriminative relationship between covert oral behavior and the phonemic system in internal information processing. *Journal of Experimental Psychology, 103,* 885–890.

Means, M. K., Lichstein, K. L., Epperson, M. T.,

& Johnson, C. T. (2000). Relaxation therapy for insomnia: Nighttime and day time effects. *Behaviour Research and Therapy, 38*(7), 665–678.

Miller, M. P., Murphy, P. J., & Miller, T. P. (1978). Comparison of electromyographic feedback and progressive relaxation training in treating circumscribed anxiety stress reactions. *Journal of Consulting and Clinical Psychology, 46*(6), 1291–1298.

Miro, D. J. (1981). A comparative evaluation of relaxation training strategies utilizing EMG biofeedback. *Dissertation Abstracts International, 42*(3-B), 1183–1184.

Murphy, A. I., Lehrer, P. M., & Jurish, S. (1990). Cognitive coping skills training and relaxation training as treatments for tension headaches. *Behavior Therapy, 21,* 89–98.

Murphy, L. R. (1983). A comparison of relaxation methods for reducing stress in nursing personnel. *Human Factors, 25*(4), 431–440.

Nicassio, P., & Bootzin, R. (1974). A comparison of progressive relaxation and autogenic training as treatments for insomnia. *Journal of Abnormal Psychology, 83*(3), 253–260.

Nicassio, P. M., Boylan, M. B., & McCabe, T. G. (1982). Progressive relaxation, EMG biofeedback and biofeedback placebo in the treatment of sleep-onset insomnia. *British Journal of Medical Psychology, 55*(2), 159–166.

Oliver, A. M. (1997). A study of selected treatments of performance anxiety with a survey of performance anxiety among participants of the 1994 International Horn Symposium. *Dissertation Abstracts International: Section A. Humanities and Social Sciences, 58*(5-A), 1498.

Ost, L. G. (1988). Applied relaxation vs. progressive relaxation in the treatment of panic disorder. *Behaviour Research and Therapy, 26*(1), 13–22.

Paul, G. L., & Trimble, P. W. (1970). Recorded vs. "live" relaxation training and hypnotic suggestion: Comparative effectiveness for reducing physiological arousal and inhibiting stress response. *Behavior Therapy, 1,* 285–302.

"Progressive relaxation" eases masticatory pain syndrome. (1974). *Dental Survey, 50*(3), 28.

Puskarich, C. A., Whitman, S., Dell, J., Hughes, J. R., Rosen, A. J., & Hermann, B. P. (1992). Controlled examination of effects of progressive relaxation training on seizure reduction. *Epilepsia, 33*(4), 675–680.

Quayle, C. M. (1979). Relative effectiveness of audio-taped and live therapist presented relaxation in terms of physiological parameters. *Dissertation Abstracts International, 40B,* 3960.

Reinking, R. H., & Kohl, M. L. (1973). Effects of various forms of relaxation training on physiological and self-report measures of relaxation. *Journal of Consulting and Clinical Psychology, 43*(5), 595–600.

Renfroe, K. L. (1988). Effect of progressive relaxation on dyspnea and state anxiety in patients with chronic obstructive pulmonary disease. *Heart and Lung, 17*(4), 408–413.

Roatta, S., & Farina, D. (2010). Sympathetic actions on the skeletal muscle. *Exercise and Sport Sciences Reviews, 38*(1), 31–35.

Roatta, S., Windhorst, U., Ljubisavljevic, M., Johansson, H., & Passatore, M. (2002). Sympathetic modulation of muscle spindle afferent sensitivity to stretch in rabbit jaw closing muscles. *Journal of Physiology, 540,* 237–248.

Rothman, H. S. (1979). Electromyographic biofeedback relaxation training versus progressive muscle relaxation training in the treatment of test anxiety. *Dissertation Abstracts International, 40*(1-B), 463.

Rousseau, A., Hermann, B., & Whitman, S. (1985). Effects of progressive relaxation on epilepsy: Analysis of a series of cases. *Psychological Reports, 57*(3, Pt. 2), 1203–1212.

Schaer, B., & Isom, S. (1988). Effectiveness of progressive relaxation on test anxiety and visual perception. *Psychological Reports, 63*(2), 511–518.

Seers, K., Crichton, N., Tutton, L., Smith, L., & Saunders, T. (2008). Effectiveness of relaxation for postoperative pain and anxiety: Randomized controlled trial. *Journal of Advanced Nursing, 62*(6), 681–688.

Setterlind, S. (1983). Teaching relaxation in physical education lessons: I. Psychological results from empirical studies on school. *Scandinavian Journal of Sports Sciences, 5, 56–59.*

Shapiro, S., & Lehrer, P. M. (1980). Psychophysiological effects of autogenic training and progressive relaxation. *Biofeedback and Self-Regulation, 5,* 249–255.

Shaw, L., & Ehrlich, A. (1987). Relaxation training as a treatment for chronic pain caused by ulcerative colitis. *Pain, 29*(3), 287–293.

Sigman, M., & Amit, Z. (1982). Progressive relaxation exercises and human gastric acid output: A study using telemetric measurements. *Behaviour Research and Therapy, 20*(6), 605–612.

Sloman, R., Brown, P., Aldana, E., & Chee, E. (1994). The use of relaxation for the promotion of comfort and pain relief in persons with advanced cancer. *Contemporary Nurse, 3*(1), 6–12.

Smith, R. (2011). The "sixth sense": Towards a history of muscle sensation. *Gesnerus, 68*(1), 218–271.

Sprague, A. E. (2004). The evolution of bed rest as a clinical intervention. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 33*(5), 542–549.

Stuempfle, K. J., & Drury, D. G. (2007). The physiological consequences of bed rest. *Journal of Exercise Physiology Online, 10,* 32–41.

Surwit, R. S., & Feinglos, M. N. (1983). The effects of relaxations on glucose tolerance in non-insulin-dependent diabetes. *Diabetes Care, 6*(2), 176–179.

Tornoe, B., & Skov, L. (2012). Computer animated relaxation therapy in children between 7 and 13 years with tension-type headache: A pilot study. *Applied Psychophysiology and Biofeedback, 37*(1), 35–44.

Tsitsi, T., Charalambous, A., Papastavrou, E., & Raftopoulos, V. (2017). Effectiveness of a relaxation intervention (progressive muscle relaxation and guided imagery techniques) to reduce anxiety and improve mood of parents of hospitalized children with malignancies: A randomized controlled trial in Republic of Cyprus and Greece. *European Journal of Oncology Nursing, 26,* 9–18.

Turnbull, G. K., & Ritvo, P. G. (1992). Anal sphincter biofeedback relaxation treatment for women with intractable constipation symptoms. *Diseases of the Colon and Rectum, 35*(6), 530–536.

Turner, R. M., & Ascher, L. M. (1979). Controlled comparison of progressive relaxation, stimulus control, and paradoxical intention therapies for insomnia. *Journal of Consulting and Clinical Psychology, 47*(3), 500–508.

Vazquez, I., & Buceta, J. (1993). Relaxation therapy in the treatment of bronchial asthma: Effects on basal spirometric values. *Psychotherapy and Psychosomatics, 60*(2), 106–112.

Wallin, B. G. (2006). Regulation of sympathetic nerve traffic to skeletal muscle in resting humans. *Clinical Autonomic Research, 16*(4), 262–269.

Walsh, P., Dale, A., & Anderson, D. E. (1977). Comparison of biofeedback pulse wave velocity and progressive relaxation on essential hypertensives. *Perceptual and Motor Skills, 44*(3, Pt. 1), 839–843.

Wardell, D. W., Rintala, D. H., Duan, Z., & Tan, G. (2006). A pilot study of healing touch and progressive relaxation for chronic neuropathic pain in persons with spinal cord injury. *Journal of Holistic Nursing, 24*(4), 231–240.

Wesner, R. B., Noyes, R., & Davis, T. L. (1990). The occurrence of performance anxiety among musicians. *Journal of Affective Disorders, 18*(3), 177–185.

White, D. W., Shoemaker, J. K., & Raven, P. B. (2015). Methods and considerations for the analysis and standardization of assessing muscle sympathetic nerve activity in humans. *Autonomic Neuroscience Basic and Clinical, 193,* 12–21.

Whitman, S., Dell, J., Legion, V., Eibhlyn, A., & Statsinger, J. (1990). Progressive relaxation for seizure reduction. *Journal of Epilepsy, 3*(1), 17–22.

Wiener, N. (1948). *Cybernetics.* New York: Wiley.

Wilson, V. E., & Bird, E. I. (1981). Effects of relaxation and/or biofeedback training upon hip flexion in gymnasts. *Biofeedback and Self-Regulation, 6*(1), 25–34.

Wolpe, J., & Lazarus, A. A. (1966). *Behavior therapy techniques.* New York: Pergamon Press.

Woolfolk, R. L., Lehrer, P. M., McCann, B. S., & Rooney, A. J. (1982). Effects of progressive relaxation and meditation on cognitive and somatic manifestations of daily stress. *Behaviour Research and Therapy, 20,* 461–468.

Yilmaz, C. K., & Kapucu, S. (2017). The effect of progressive relaxation exercises on fatigue and sleep quality in individuals with COPD. *Holistic Nursing Practice, 31*(6), 369–377.

Yu, D. S., Lee, D. T., & Woo, J. (2007). Effects of relaxation therapy on psychologic distress and symptom status in older Chinese patients with heart failure. *Journal of Psychosomatic Research, 62*(4), 427–437.

CHAPTER 8

Eye Movement Desensitization and Reprocessing Therapy for Stress Management

Roger Solomon
Deany Laliotis
Francine Shapiro

Eye movement desensitization and reprocessing (EMDR) therapy is an approach to psychotherapy that emphasizes the importance of the information processing system in the treatment of psychopathology (Shapiro, 2007, 2014, 2018). The theoretical basis underlying EMDR therapy is the adaptive information processing (AIP) model. According to this theory, the causes of mental health problems (excluding those due to lack of information, physical injury, toxicity, or genetic factors) are the result of earlier disturbing or traumatic experiences that have been inadequately processed and maladaptively stored. These maladaptively stored memories contain the negative beliefs, emotions, physical sensations, and perceptions experienced during the original disturbing event; when triggered by similar circumstances in the present, they cause pronounced symptoms. For example, a disparaging remark by a colleague in the present can result in feelings of inadequacy that arise from triggering early childhood experiences with critical caregivers that resulted in a negative sense of self. The cornerstone of EMDR therapy is the identification and reprocessing of the memories that are informing the person's difficulties in the present, bringing him or her to an adaptive conclusion about the self that is more accurate.

The EMDR therapy model consists of eight phases and a clinical methodology that guides the clinician to understand the client's present problems as stemming from past experiences by identifying and processing (1) the specific memories of adverse experiences underlying their symptoms and (2) the current circumstances that trigger distress based on these earlier experiences, and then (3) developing the life skills needed to guide the responses to future situations.

EMDR therapy shares many elements with other major therapeutic orientations, but it is distinctly different from them as well. For instance, EMDR therapy recognizes the significant role of childhood events in the etiology of psychopathology but eschews the need to interpret these experiences to deliberately make cognitive connections between memories of past events and current emotional reactions, as is common in the psychodynamic therapies. Rather, EMDR directly accesses and processes the memories of earlier negative experiences that are the basis of current psychopathology. In addition to the spontaneous reduction in distress, there is also an emotional shift to a more adult understanding and insight as a by-product of the processing. Furthermore, in contrast to cognitive-behavioral therapy (CBT), EMDR therapy does not view beliefs and behaviors as the cause of psychopathology and the basis of change. Rather, EMDR therapy's procedures are informed by the premise that the negative beliefs and behaviors are the result of these maladaptively stored memories, and, therefore, the memories are the focus of treatment.

According to the World Health Organization (WHO, 2013), trauma-focused CBT and EMDR therapy are the only approaches recommended for children, adolescents, and adults with posttraumatic stress disorder (PTSD). However, there are important and significant procedural differences: "Unlike CBT with a trauma focus, EMDR does not involve (a) detailed descriptions of the event, (b) direct challenging of beliefs, (c) extended exposure, or (d) homework" (2013, p. 1). Consequently, EMDR therapy may be more gentle, may minimize the shame that may come with disclosure, and may lower the risk for retraumatization for clients with distressing narratives. Further, EMDR therapy may be emotionally less taxing because no homework is required. For example, Arabia, Manca, and Solomon (2011) found that cardiac patients with significant trauma reactions found the exposure homework too tiring, with the study concluding that EMDR therapy may be easier to tolerate.

HISTORICAL CONTEXT

EMDR therapy originated with Francine Shapiro in 1987, when she observed that repeated eye movements while focusing on a disturbing event resulted in a decrease in negative affect. The first controlled research study was published in 1989 (Shapiro, 1989), which demonstrated the efficacy of what was then called "eye movement desensitization" in treating traumatic memories. The name reflected the desensitization effect observed by the bilateral stimulation component (eye movement) that was administered while focusing on a disturbing experience. In 1991, the name was changed to "eye movement desensitization and reprocessing" (Shapiro, 1991) to reflect the broader conceptualization that, in addition to the desensitization effects, the targeted memory spontaneously linked to other related experiences, resulting in adaptive affective, somatic, and cognitive shifts that were stable over time. In EMDR therapy, therefore, the emphasis is on the comprehensive reprocessing of memories that underlie present symptoms, as these previous experiences are understood to be the basis of both pathology and mental health.

In 1989, when the initial study was first published, PTSD was considered largely intractable, and no empirically supported treatments were available. Consequently, the positive effects that were reported within one session resulted in years of controversy regarding both the efficacy of EMDR therapy and the role of the eye movements. However, in the 1990s and in decades afterward, research continued to demonstrate the effectiveness of EMDR therapy. Currently, EMDR therapy is widely regarded as an empirically validated treatment for trauma (Department of Veterans Affairs & Department of Defense, 2017; International Society for Traumatic Stress Studies, 2018; WHO, 2013) with more than 30 randomized controlled trials (RCTs) that demonstrate its effectiveness in treating a variety of clinical problems. Further, a meta-analysis has indicated that eye movements significantly contribute to treatment effects (Lee & Cuijpers, 2013).

THEORETICAL BACKGROUND

EMDR therapy is guided by the AIP model (Shapiro, 2001, 2007, 2018; Shapiro & Laliotis, 2011, 2015), which hypothesizes that current pathology generally results from adverse life experiences that are inadequately processed. The AIP model guides case conceptualization, specifies procedures and protocols of EMDR therapy, predicts successful clinical outcomes, and provides an explanation of personality development and the origin of psychopathology. Central to the model is the assumption that the human brain interprets the meaning of a present situation according to previously established memory networks of similar experiences (the similarity being among many discrete or overlapping dimensions, such as affect, perceptions, belief, type of situation, participants, etc.). For example, if one had never seen a computer, its purpose would be unknown. However, previous experiences with a computer would inform an understanding in the present. Likewise, if a person has a negative experience with a computer (e.g., cannot get the software to work properly), the experience may be encoded in a neural network of previous experiences informed by similar failures accompanied with feelings of inferiority. These experiences become physiologically encoded memories that are stored in associative neural memory networks. In turn, these memory networks provide the basis for the person's interpretation of subsequent experiences, informing perceptions, attitudes, feelings, and behaviors.

Everyday life experiences that are adaptively stored are readily integrated into the existing neural networks of the brain in such a way that useful information is retained and the rest discarded. Adverse life experiences, however, can overload the information processing system, overwhelming the person in such a way that these memories get stored in excitatory, distressing, state-specific form (Shapiro, 2001, 2018), encoded with the emotions, physical sensations, and perceptions experienced at the time of the event. These memories become stored in isolation, unable to link with other memory networks of adaptively stored life experiences, preventing adaptive learning from taking place. As a result, the episodic memory does not integrate into the semantic memory system (see Stickgold, 2002). Because perceptions of current situations are automatically interpreted through the connections and associations with existing memory networks, the AIP model predicts that a current stressful situation will trigger memory networks of maladaptively stored information. The perceptions, emotions, and physical sensations inherent in the unprocessed memory automatically arise. When this occurs, "the past becomes present," and a person can experience a distressing response to a current situation that is out of proportion to the current context. Examples include the PTSD symptoms of intrusive thoughts, nightmares, and flashbacks of a past trauma that are triggered by specific stimuli in the present. However, adverse life experiences that do not meet criteria for PTSD can also be maladaptively stored and provide the basis for a wide variety of clinical problems. The more ubiquitous and chronic experiences of humiliation, rejection, and abandonment often underlie negative self-beliefs, low self-esteem, self-efficacy issues, and attachment difficulties.

Substantial research has confirmed the AIP precept that adverse life experiences are the basis of many clinical and somatic disorders (e.g., Felitti et al., 1998). Further, research has demonstrated that common life experiences that are disturbing can generate even more symptoms of PTSD than major trauma (e.g., Mol et al., 2005). Therefore, EMDR therapy has been widely used to treat a broad range of clinical presentations and stress conditions (Shapiro, 2014). Table 8.1 lists EMDR therapy studies illustrating effectiveness for a wide variety of clinical presentations.

EMDR therapy involves the utilization of standardized procedures, which include sets of eye movements or other forms of bilateral dual attention stimuli (tactile or auditory), to access the client's memories as they are currently experienced and process them to an adaptive resolution. With the accessing of the maladaptively stored information and the concurrent bilateral stimulation, there is a reduction of negative thoughts, affects, and sensations that yields a new understanding, incorporating a present orientation to the past experience. The incident and other similar memories become successfully integrated in an adaptive memory network that contains other, similar conclusions about self

TABLE 8.1. Selected Studies of EMDR Therapy with a Variety of Clinical Populations

Randomized trials of trauma victims Abbasnejad, Mahani, & Zamyad (2007); Acarturk et al. (2016); Ahmad, Larsson, & Sundelin-Wahlsten (2007); Arabia, Manca, & Solomon (2011); Capezzani et al. (2013); Carletto & Pagani (2016); Carlson, Chemtob, Rusnak, Hedlund, & Muraoka (1998); Chemtob, Nakashima, Hamada, & Carlson (2002); de Bont, van Minnen, & de Jongh (2013); de Roos & de Jongh (2008); Diehle, Opmeer, Boer, Mannarino, & Lindauer (2014); Gil-Jardiné et al. (2018); Högberg et al. (2008); Ironson, Freund, Strauss, & Williams (2002); Jaberghaderi, Greenwald, Rubin, Dolatabadim, & Zand (2004); Jarero, Artigas, & Luber (2011); Jarero, Artigas, Uribe, & Garcia (2016); Kemp, Drummond, & McDermott (2010); Marcus, Marquis, & Sakai (1997, 2004); Nijdam, Gersons, Reitsma, de Jongh, & Olff (2012); Novo et al. (2014); Power et al. (2002); Rothbaum (1997); Rothbaum, Astin, & Marsteller (2005); Scheck, Schaeffer, & Gillette (1998); Shapiro & Laub (2015); Shapiro (1989); Solomon, Solomon, & Heide (2009); van den Berg & van den Gaag (2012); van den Berg et al. (2015); van der Kolk et al. (2007); Wilson, Becker, & Tinker (1995, 1997)	*Chemical dependency, sexual deviation/addiction, pathological gambling, and other behavioral (process) addictions* Abel & O'Brien (2010); Bae, Hahn, & Kim (2015); Brown, Gilman, Goodman, Adler-Tapia, & Freng (2015); Cox & Howard (2007); Hase, Schallmayer, & Sack (2008); Henry (1996); Littel, van den Hout, & Engelhard (2016); Perez-Dandieu et al. (2015); Ricci & Clayton (2008); Rougemont-Bucking & Zimmermann (2012); Shapiro, Vogelmann-Sine, & Sine (1994); Tsoutsa, Fotopoulos, Zakynthinos & Katsaonou (2013); Vogelmann-Sine, Sine, & Smyth (1999); Zweben & Yeary (2006)
Phobias, panic disorder, obsessive-compulsive disorder, and generalized anxiety disorder de Jongh (2012); de Jongh, Holmshaw, Carswell, & van Wijk (2010); de Jongh & ten Broeke (1998); de Jongh, van den Oord, & ten Broeke (2002); de Roos & de Jongh (2008); Doering, Ohlmeier, de Jongh, Hofmann, & Bisping (2013); Farretta (2013); Feske & Goldstein (1997); Gupta, Gupta, & Choudhary (2014); Marr (2012); Marsden (2016); Morrissey (2013); Nazari, Momeni, Jariani, & Tarrahi (2011); Newgent, Paladino, & Reynolds (2006); Rathschlag & Memmert (2014)	*Somatic problems/somatoform disorders, including migraines, chronic pain, phantom limb pain, chronic eczema, gastrointestinal problems, CFS, psychogenic seizures, eating disorders, and negative body image* Bloomgarden & Calogero (2008); Brown, McGoldrick, & Buchanan (1997); Chemali & Meadows (2004); de Roos et al. (2010); Farretta & Civilotti (2016); Gerhardt et al. (2016); Grant (1999); Grant & Threlfo (2002); Hughes (2014); Jarero, Artigas, Uribe, & Garcia (2016); Kelley & Selim (2007); Konuk, Epözdemir, Atçeken, Aydın, & Yurtsever (2011); Marcus (2008); Maroufi, Zamani, Izadikhah, Marofi, & O'Connor (2016); Mazzola et al. (2009); Royle (2008); Russell (2008); Schneider, Hofmann, Rost, & Shapiro (2008); van Rood & de Roos (2009)

and other, past and present. As a result, the client is better able to respond to the stressors in the present based on the current circumstances once the affective load from the past has been reduced or eliminated altogether.

EMDR THERAPY AND STRESS MANAGEMENT

Stress is part of daily life. Our response to stressors, however, is unique to each of us and is informed by previous life experiences, for better and worse. In AIP terms, a stressful situation stimulates similar memories that inform the appraisal of the situation and the coping strategies that are applied to deal with it. If previously stressful experiences have been fully processed and integrated within the wider memory network, the person will likely have a base of stability, comprehension, and manageability, enabling her or him to respond effectively to the stressor(s), both internally and externally. If, however, past experiences associated with the stressful event are inadequately processed and contain negative affects (e.g. powerlessness, defectiveness, or lack of safety), the person will be triggered by the current stressor(s), resulting in dysfunctional responses that often exacerbate the situation and the person's reaction to it. The disturbing life experiences that are maladaptively stored are activated by these present stressors, resulting in negative emotions and self-beliefs and diminished capacity to cope with the current demands of the stressful situation. In turn, the negative effects of diminished coping become maladaptively stored, decreasing one's resiliency and ability to cope, resulting in greater vulnerability and tendency to respond maladaptively in future stressful situations. Once the past experiences that are associated with the present stressor(s) are identified and processed, the present triggers may or may not still activate, depending on the correlation between the past and present situations. The present triggers should also be targeted, setting the stage to develop more adaptive responses to the same or similar situations in the future. A future template of adaptive action is also generated, in order to ensure that the client has a range of adaptive responses, as well as necessary skills to apply them to the same or similar stressful situations in the future.

DESCRIPTION OF EMDR THERAPY

EMDR therapy is an eight-phase, three-pronged treatment protocol of past, present, and future. The three prongs include (1) identification and treatment of the past memories underlying current symptoms; (2) current situations (present triggers) that elicit disturbance; and (3) needed skills and adaptive responses that generate adaptive templates for the future. This eight-phase approach is discussed in detail below with two case examples, Laura and Bill, that will be described throughout this section to illustrate how EMDR therapy can be effectively utilized with stress-related issues.

Presenting Problems

Laura

Laura is a 26-year-old single woman who sought treatment for work-related anxiety as part of a team in an office setting. She has worked successfully on a variety of projects over the time she's been in this job and regularly meets with different colleagues associated with these projects. Although she is well regarded among her colleagues, she would get particularly anxious when she had to make an oral presentation in front of them. Laura not only found herself anxious during meetings when she would have to make a presentation, but she also experienced increasing anticipatory anxiety and dread in the days preceding a meeting.

Bill

The case of Bill, described in a previous paper (Shapiro & Solomon, 2017), illustrates how EMDR therapy can be used to deal with the stress of PTSD and moral injury. Bill had two tours of duty in Iraq and Afghanistan. He presented for therapy with classic symptoms of PTSD 3 years after being discharged from the military. He had experienced numerous traumatic incidents, including being a passenger in a helicopter that was shot down and involvement in several firefights. The incident that caused him the most difficulty occurred when he was in charge of a convoy providing security for a VIP. A local man on a motorcycle tried to get next to the VIP's vehicle and refused to be waved off. Bill then gave the order to run down the motorcycle, and the man was killed. Bill and the man on the motorcycle made eye contact as he was being run over, and it was this personal moment that has haunted him since that time, more than other traumatic incidents. It was later discovered that there was a bomb on the motorcycle. Bill had nightmares of the man's face, along with other common PTSD symptoms. Current situations that triggered fear and anxiety included being in crowds, driving in heavy traffic, and sitting in a restaurant, where he would sit with his back against a wall in order to feel safe. He also reported difficulties at work, including a need to be "perfect" and an inability to deal with criticism.

Phase 1: Client History

The clinician obtains information on the client's background and current psychosocial functioning and evaluates the appropriateness and readiness for EMDR therapy. Once a mutual understanding of the presenting problem is reached, the past memories are identified that inform the client's difficulties, along with the present triggers and desired outcomes for the future. Further, an assessment is made of the client's overall emotional stability and available adaptive information for memory processing. The clinician also identifies the client's behavioral–emotional deficits, as well as the needed skills and psychosocial education important for future adaptive behavior. After processing past experiences and present triggers related to behavioral deficits, the client can learn new skills important for adaptive behavior that can, in turn, be enhanced and encoded through the future template.

Laura

The focus of treatment for Laura was her anxiety regarding presentations. Her history revealed that, despite making good grades in high school and college, she felt anxious about her academic abilities and dreaded being compared with others. Giving oral presentations had always been difficult, and she had memories from grammar school and high school of being very nervous and making mistakes during presentations. She described good relationships with her parents and older sister, but there was always pressure from them to perform well in school. She was often compared to her sister, who received better grades and excelled in sports. Laura got along well with coworkers, had several close friends with whom she regularly socialized, and had been dating a man for 22 months. She enjoyed yoga, hiking, and photography.

Laura was told about EMDR memory processing procedures and what to expect with EMDR therapy, and a mutually agreed-upon treatment plan was developed. Specific childhood memories were identified for processing: When she was 6 years old, her mother told her that her sister could draw better than she could and that she should try harder. Similar memories about comparisons with her sister's athletic abilities and scholastic achievement were identified. Memories of grammar school and high school were identified in which she was nervous about giving presentations or was worried that her classmates were bored. Present triggers at work included giving presentations and when she heard that there was going to be a meeting. The future template discussed involved feeling calm, centered, and confident in her abilities when presenting at a group meeting.

Bill

Bill grew up in a middle-class family. He described his father as strict and having high standards. His mother was described as gentler, supportive of him, but also expecting him to live up to his father's standards. Bill was an average student in school and joined the army after graduating from high school. He reported no major trauma in growing up. Treatment was to focus on his PTSD symptoms stemming from his war experiences.

Phase 2: Preparation

Clients deemed appropriate for EMDR therapy are prepared for memory processing. The goals in this phase include: (1) establishing a therapeutic alliance; (2) educating the client about symptoms (e.g., current problems are the result of past maladaptively stored memories); (3) explaining the EMDR process, its effects, and what to expect; and (4) teaching the client self-soothing methods that increase stability and enable a sense of personal self-mastery and control. A typical preparation procedure used during this phase is the safe/calm place exercise. The client is asked to visualize a scene that evokes a feeling of safety and/or calmness. Bilateral stimulation (e.g., eye movement, taps, or tones) is used to enhance the positive feelings (Shapiro, 2001, 2018). Resource development and installation (Korn & Leeds, 2002) is another stabilization method in which positive memories/images involving adaptive qualities and characteristics (e.g., memories that evoke self-efficacy) are identified and enhanced with bilateral stimulation. The history-taking and preparation phases can be provided in the first one or two sessions, with memory processing commencing in the next session. However, for complex trauma, more time may be necessary for history-taking and preparation phases to ensure comprehensive assessment, sufficient stabilization, and adequate readiness to begin memory processing.

Laura

Laura was taught a safe/calm place exercise, which she responded to well. She was able to recall a relaxing image of being in a forest associated with pleasant, peaceful feelings.

She was taught how to use this exercise to calm herself whenever she experienced distress in her day-to-day life.

Resource development and installation (RDI) procedures were also offered to strengthen her feelings of being competent and capable while managing her current challenges. The procedures were used to enhance positive imagery and feeling states, and then she practiced applying these resource states to better cope with the distress associated with presenting to her coworkers. Further, these resource experiences, along with a new ability to lower arousal with her safe place, helped her feel calm and capable while imagining her condescending colleague making a negative comment.

In the next session (Session 3), Laura reported she was able to use the safe place and resource exercises to calm herself, feeling more relaxed generally, but still reported being anxious in anticipation of group meetings and reporting to the group. It was decided to process the memories underlying her anxiety that were identified in the history-taking session.

Bill

Bill was given six sessions in which he was taught the safe/calm place exercise and provided a resourcing strategy, RDI, to bolster feelings of competence and safety in order to help him better cope with his hypervigilance and heightened sense of danger stemming from his war experiences.

Phase 3: Assessment of the Target Memory

The target memory to be treated is accessed through eliciting the client's current experience of the past event by identifying the components of the client's experience. These include the mental image that represents the worst part of the memory; the presently held negative, irrational belief about the self; the desired positive belief; the present emotions and physical sensation(s); and the baseline measurements. Baseline measurements include how true or valid the desired positive belief feels (Validity of Cognition, or VoC) on a 1-to-7 scale (with 1 indicating that the positive cognition feels totally false and 7 that it feels totally true) and an overall measure of distress on the Subjective Units of Disturbance (SUD) scale, on which 0 is no disturbance or calm and 10 is the highest disturbance one can imagine.

Laura

The first memory selected for EMDR processing was the memory of her mother's comparing Laura to her sister when she was 6 years old. Following are the baseline components of the memory for Laura when she brought it to mind: the image of her mother looking at her drawing with a disappointed expression on her face; the negative belief about herself, "I'm not capable"; the positive belief that she wanted to have about herself, "I am capable," with a VoC of 3. Her emotions were sadness and anxiety; her SUD was an 8; and the body sensation was a constriction in her chest.

Bill

The image for Bill was the man looking at him with fear and anger; the negative belief, "it's my fault"; and a positive belief, "I did what I had to do," with a VoC of 2. The emotions were fear and sadness, with an SUD of 8, and he experienced a painful constriction in his stomach.

Phase 4: Desensitization

The goal of this next phase is to initiate the reprocessing of the target memory and the associated negative experiences that are maladaptively stored. The client is asked to think of the memory (e.g., the image, negative cognition, and sensations) and follow the therapist's fingers back and forth across the visual field for 20–30 seconds, more or less depending on the needs of the client. Alternatively, if eye movements are uncomfortable or do not appear to be effective, bilateral stimulation using taps or tones can be applied. The bilateral stimulation seems to initiate an associative process, with each new set evoking new associations, such as new images, thoughts, feelings, and/or sensations about the targeted memory and other events that are in the memory network. The therapist facilitates the client's experience by administering the bilateral stimulation and observing the nonverbals. The therapist maintains a stance of detached compassion with minimal interference. Unless the client gets stuck in the process and requires an intervention, the clinician simply checks in with the client between sets of bilateral stimulation, instructing the client to "just notice" what comes up, and continues for multiple sets of bilateral stimulation until the client is no longer reporting a change in experience. The target memory and other associations transmute during the reprocessing, allowing them to be successfully integrated within adaptive memory networks. Clinical effects are measured utilizing the SUD scale, with the goal being 0 (no experience of actual distress in the present given what actually occurred). An adaptive resolution in EMDR therapy is always ecological; that is, it does not take away appropriate emotions, and, consequently, not all memories will process to "0." For example, a veteran whose close friend was killed went from an SUD of 8 to an SUD of 2 and could not go lower because "a good friend and person died."

Laura

The desensitization phase was initiated with bilateral stimulation while attending to the current sensory components of Laura's experience. Laura immediately accessed her shame and confusion about her mother's negative reactions. After two sets of bilateral stimulation, she recalled additional memories of her mother's relentless comparisons to her older sister. Feelings of shame and confusion gave way to anger and hurt at her mother for making such comparisons. With continuing sets, Laura's emotional distress diminished, and she began to remember times when her mother was more supportive, validating her abilities and encouraging her to keep up her good efforts. She was able to realize that her mother was trying to encourage her to do better by comparing her to her sister. Her childhood perception that she could never measure up to her sister yielded to an adult understanding—that she really was capable and talented, but in different ways than her sister was. Additionally, she could recognize that her mother was well intentioned, even though comparing her to her sister was ineffective and hurtful. When checking the work, the client is asked to bring to mind the original target memory. Laura reported feeling calm, and the SUD rating was 0. Her positive belief, "I am capable," in the context of the neutralized memory, felt completely true (VoC 7), with the body scan (described subsequently) indicating the congruency between the client's self-report and the felt sense of the memory.

Bill

A transcript of a portion of the desensitization is provided below. After the first set of eye movements, the therapist asked Bill what he noticed:

BILL: My mind feels calmer about it. It's not as intense. It's still there. I still feel it, but it's not as intense.

THERAPIST: Go with that. [*Set of eye movements*] What do you get now?

BILL: More relaxed—I picture his face. It's there, but it's not as intense—that's the only way I can explain it.

THERAPIST: Go with that. [*Set of eye movements*] What do you get now?

BILL: Much calmer, my mind is not racing around.

THERAPIST: Now when you bring it back to the incident, what do you notice?

BILL: More calmness. It's clear I had to focus on the safety of my men and the success of my mission. I see now that I read the situation for what is was . . . that it was dangerous and he wanted to kill us.

THERAPIST: Go with that. [*Set of eye movements*] What do you get now?

BILL: I notice I am not jumping around thinking about all the different scenarios in my head. It just feels like it happened and it was the only thing that could happen. I am OK, and my team is OK, and it's not as intense.

THERAPIST: How disturbing is it on a scale of zero to ten, with zero being calm and ten the worst it could be?

BILL: A one maybe, maybe a zero.

THERAPIST: Go with that. [*Set of eye movements*] What do you get now?

BILL: It's something that happened and I will always remember the face, but I'm not getting all tight now.

THERAPIST: Zero to ten?

BILL: I feel much calmer, zero.

Phase 5: Installation

The purpose of this phase is to link the neutralized memory and the positive cognition, strengthening the connection to the existing adaptive memory networks. This linkage facilitates the generalization effects to the associated memory networks. In this case, Laura was able to connect the positive cognition "I am capable" to the neutralized memory of her mother comparing her to her sister. With multiple sets of bilateral stimulation, she was able to achieve a VoC rating of 7 (out of 7).

Bill

The transcript continues:

THERAPIST: Do the words, "I did what I had to do" still fit, or are there other words that may be more suitable?

BILL: I feel more confident in those words. I was right. I don't have any doubts any more. I feel what I did was my only option.

THERAPIST: So, the words "I did what I had to do," fit now?

BILL: Yes, I don't have any doubt as I did before.

THERAPIST: How true do they feel now, one to seven, with "one" being totally false and "seven" being totally true?

BILL: Six to a seven.

THERAPIST: Think of that memory along with the words, "I did what I had to do," and hold them together. [*Set of eye movements*]

BILL: I definitely feel more relaxed about the whole thing. I feel I am justified and was justified in what I did. I can trust myself.

THERAPIST: When you bring up the memory, how true do those words "I did what I had to do; I can trust myself" feel? One to seven?

BILL: Seven.

Phase 6: Body Scan

This phase is to ensure a comprehensive reprocessing effect by holding the memory and positive cognition in mind and scanning the body to make sure the somatic experience is congruent with the desired state.

Laura

Laura reported that she mostly felt relaxed, but she had some residual tension in her chest, accompanied by feelings of anger and sadness. This was related to a recognition of the impact of these experiences on her self-esteem. Focusing on these sensations in subsequent sets allowed the negative feelings to diminish.

Bill

The transcript continues:

THERAPIST: Bring up the memory and the words, "I did what I had to do; I can trust myself" and scan your body, starting from the top of your head and going downward, and let me know if there is any tightness, tension, or unusual sensation.

BILL: [*Shakes his head, indicating "No."*]

THERAPIST: Just notice that. [*Set of eye movements*]

BILL: No tightness in my chest or body, no tingly sensations.

THERAPIST: Notice that. [*Set of eye movements*]

BILL: I feel relaxed, calm, no tightness anymore in my chest It has been a long time since I have felt this way.

Phase 7: Closure

In this phase, the clinician instructs the client to shift out of the memory work by initiating procedures to return clients to equilibrium, as needed, to ensure client stability. Further, clients are educated about what to expect in between sessions. For example, the processing may continue, and other memories, dreams, and related material may arise. They are instructed to keep a log of their psychological experiences and utilize calming strategies as needed.

Laura

The therapist assisted Laura in accessing her safe place to quiet the residual disturbance. In debriefing the session, the client reported feeling much calmer. Although she could understand what happened, she also knew she would continue to be sad about it, which seemed ecological, given her adult understanding of the negative impact of these experiences. She was also asked to keep a log and utilize her safe place and other calming skills when needed.

Bill

The transcript continues:

> THERAPIST: What's different for you?
>
> BILL: It's hard to explain, but it feels like it's in the past, like it happened before now. I feel relaxed and comfortable and solid. Like I am living in the now and not going over in my head all the different things about that situation, what could have happened. It's over.

Bill continued, saying that he was initially skeptical but felt a lot better and that the experience really did seem in the past. Bill was then told that, after the session, processing could continue, and other memories, images, thoughts, feelings, or sensations might arise. He was asked to keep a log of any situations he found disturbing, to help him remember what he might want to discuss at the next session. Further, he was reminded to use his safe place exercise to deal with any distress, as needed.

Phase 8: Reevaluation

This phase takes place at the next session. Clients are assessed regarding their current psychological state, whether the results of the previous session have maintained, what associations may have emerged since the last session, as well as changes in symptoms and behaviors. This information is used to guide the direction of treatment.

Laura

Laura reported that she was much calmer at work and was able to participate in group meetings with greater confidence. The treatment effects from the processing session were maintained. She could bring up the previously targeted memory of her mother comparing her to her sister with a feeling of calmness (SUD = 0) and the positive belief of "I'm capable," with a VoC of 7. However, in the subsequent week, more negative memories regarding her mother's handling of her came to mind. For the next four sessions, negative memories involving her mother and memories of distressing school incidents were processed. Two more sessions were utilized to process present triggers that included a time when she felt anxious during a presentation, another time when she was anxious upon learning she was required to give a presentation, and two incidents when her colleague was condescending toward her in front of others. The targeting of each present trigger was followed by a future template that allowed her to imagine successfully coping with the same situation or a similar one. As a result of these processing sessions, Laura was symptom-free and able to comfortably attend as well as present at meetings. The colleague's condescending attitude was annoying but no longer caused undue distress. Further, she reported a greater sense of interpersonal confidence and a renewed sense of positive motivation at work, given that she felt more capable and relaxed.

Bill

Bill reported that he felt more relaxed and his nightmares had stopped. He also described that at work he had been able to accept his employer's suggestions concerning a project without becoming defensive. When asked to access the memory worked on in the previous session, he reported that it was still a "0" SUD and stated, "He was trying to kill us and I did what I had to do." In subsequent sessions, a helicopter crash and other war incidents in which he experienced significant fear were processed.

At that point in his treatment, his reactions to going to places where he felt exposed, such as congested traffic, crowds, and restaurants, were processed, with future templates provided. The intensity of these situations lessened, and he could maintain a "relaxed vigilance" about the unseen potential for danger. A present trigger involving a recent time when he went shopping and felt anxious was processed. Then a positive future template (being more relaxed and alert when shopping) was installed.

Discussion

EMDR therapy can be used for both symptom reduction/relief as well as for more comprehensive treatment. Laura responded well to the safe place and RDI procedures. However, symptom reduction, which is a "state change," does not lead to "trait" change, which is permanent. Instead, it helps the client learn self-soothing strategies, as well as helping him or her to be better able to observe his or her own reactivity until it can be reduced or eliminated. EMDR therapy facilitates an accelerated reprocessing of maladaptively stored memories that underlie current symptoms and provides permanent relief, as the memories driving the previous distress are no longer disturbing and are now encoded with current information as a by-product of the processing. For comprehensive treatment effects, the processing of present triggers and generating a positive future template also need to be completed. For Laura, eight sessions were sufficient to obtain comprehensive treatment effects. For some clients, fewer sessions are needed (e.g., Wilson, Becker, & Tinker, 1997); for other, more complex clinical presentations, more sessions are likely. However, each treatment plan and duration of therapy will depend upon the client's symptomatology and the number of adverse life events that need to be addressed.

Bill had PTSD stemming from war events. His most disturbing experience, giving the order to run a man over, resulted in a moral injury. Moral injury is a construct that describes the harmful aftermath of exposure to events that "transgress deeply held moral beliefs and expectations" (Litz et al., 2009). Several sessions of history taking and stabilization (e.g., the safe/calm place and RDI provided during the preparation phase) were the first step. Then reprocessing was applied to address his war experiences, as well as his present triggers. Future templates were generated for dealing with triggering situations. There was the option of treating childhood memories having to do with his strict father, which may have contributed to his belief that he had to be perfect, influencing his PTSD. However, his progress and symptom relief were such that it was decided that further treatment was not indicated at that time. Hence, symptom reduction (e.g., those of his PTSD), rather than comprehensive treatment (family-of-origin issues), was deemed appropriate.

MECHANISMS OF ACTION

Several potential mechanisms of action appear to underlie the therapeutic results of EMDR therapy. It is hypothesized that bilateral stimulation may induce a shift in brain states that initiates an associational process and a prolonged orienting response (Stickgold, 2002). This brain state is perhaps similar to that occurring during rapid eye movement (REM) sleep and may enable the processing of episodic memories, resulting in: (1) the development of insight and understanding of the memories, (2) the reduction or elimination of associated negative affect, and (3) the integration of the memories into existing semantic networks. All three factors potentially contribute to the resolution of traumatic memories.

Further, it is posited that an important mechanism underlying EMDR therapy is memory reconsolidation (Solomon & Shapiro, 2008; Van den Hout & Engelhard, 2012; Shapiro, 2014). Accessing a memory provides the possibility of it being transformed and stored in altered form (Suzuki et al., 2004). On a theoretical level, the reconsolidation of the memory, which results in the transformation of the distressing memory, distinguishes EMDR therapy from trauma-focused cognitive-behavioral therapies (TF-CBT). The underlying mechanisms of CBTs are hypothesized to be habituation and extinction, which are posited to create a new memory during the therapeutic process, while leaving the original one intact. Research has shown that the long exposure utilized in TF-CBT leads to extinction, whereas the shorter exposures typically utilized in EMDR therapy lead to memory reconsolidation (Suzuki et al., 2004). Craske, Herman, and Vansteenwegen (2006) wrote, “recent work on extinction and reinstatement . . . suggests that extinction does not eliminate or replace previous associations, but rather results in new learning that competes with the old information” (p. 6). The clinical implications are that memory reconsolidation may be responsible for a variety of EMDR treatment effects (e.g., elimination of phantom limb pain, resolution of grief and mourning issues) not found with extinction-based therapies.

Research has shown that the eye-movement component of EMDR therapy has a significant effect in and of itself. A recent meta-analysis (Lee & Cuijpers, 2013) evaluated 26 RCTs that compared the eye-movement component of EMDR therapy with a control condition involving exposure alone in which participants concentrated on a disturbing memory. Both eye movement and exposure demonstrated significant pre/post reduction in standardized outcome measures regarding negative emotions and imagery vividness. Further, this study found that the eye movements used in EMDR therapy significantly contributed to the positive treatment effects and the processing of emotional memories, compared with the protocol without use of eye movements. The hypothesized underlying mechanisms of action for eye movement that have received research support (Schubert, Lee, & Drummond, 2016) are: (1) the eye movement interferes with working memory, (2) the eye movements elicit an orienting response that activates the parasympathetic nervous system and facilitates integration, and (3) the eye movements stimulate the same neurological processes that take place during REM (dream) sleep. Further, this research suggests that the eye movements may lower emotional arousal, increase the felt recognition and believability of adaptive (and true) information, and facilitate episodic retrieval, which is an important precursor to the processing of distressing memories. Perhaps these three mechanisms come into play at different intervals during the therapy process. This may account for the consistent clinical observations and research (Shapiro, 2018) that EMDR therapy results in a reduction of the vividness of disturbing images, a rapid decrease in emotional disturbance, the emergence of past associated memories (which perhaps underlie present problems) that also seem to spontaneously process and integrate, and the spontaneous emergence of insight, positive emotions, and adaptive beliefs.

EMPIRICAL EVIDENCE

There are approximately 30 RCTs validating the efficacy of EMDR therapy for trauma treatment. EMDR therapy has been designated an effective treatment internationally in the practice guidelines of organizations such as the American Psychiatric Association (2004), the Departments of Veterans Affairs and Defense (2017), the International Society for Traumatic Stress Studies (2018), and the WHO (2012). Research has demonstrated that, for single-episode trauma, after three to six sessions of EMDR therapy, the client no longer meets criteria for PTSD. Three studies (Marcus, Marquis & Sakai, 1997, 2004; Rothbaum, 1997; Wilson, Becker, & Tinker, 1995, 1997) have shown that after the equivalent of three 90-minute sessions, 84–100% of single-trauma victims no longer met criteria for PTSD. However, more sessions are needed when multiple traumatization exists. For example, one study found that 12 sessions with war veterans resulted in the elimination of PTSD diagnosis in 77% (Carlson, Chemtob, Rusnak, Hedlund, & Muraoka, 1998). Because clinical observations have found that EMDR therapy leads to a generalization effect, it may not be necessary to process each trauma event. Traumatic events can be clustered according to type of event (e.g., abuse, neglect), clinical theme (e.g., negative events involving a parent, humiliating experiences at school), time of event (e.g., events taking place at age 10), and so on. It has been found that after processing several prominent memories, other, similar memories also seem to spontaneously process and integrate.

SIDE EFFECTS AND CONTRAINDICATIONS

EMDR therapy can be very powerful, and, as with all therapies that involve exposure to traumatic events, caution must be taken to ensure that the client can stay present while processing the memory—that is, have "one foot in the past, and one foot in the present." Screening for dissociative disorders is recommended, as well as ensuring that the client has the ability to self-regulate in and out of processing. Safe/calm place (Shapiro, 1995) and RDI (Korn & Leeds, 2002) are strategies to help teach clients how to lower arousal and to strengthen integrative capacity. EMDR therapy can safely be used for complex trauma with appropriate preparation measures and some population-specific additions to the standard protocol (Van der Hart, Groenendijk, Gonzalez, Mosquera, & Solomon, 2013, 2014).

It is also recommended that physician consultation should be sought if there are significant medical problems (e.g., heart disease) or pregnancy to ensure that processing memories that are emotionally charged will not cause harm (Shapiro, 1995, 2001, 2018). Although there are no reports of heart attacks or miscarriages occurring during or as a consequence of EMDR therapy, these cautions are emphasized in the basic training to be prudent. Further, client comfort needs to be taken into account. For example, if a client reports eye pain, then other means of bilateral stimulation (e.g., taps, tones) should be used.

Processing often continues after an EMDR therapy session, even with complete resolution. Though a sign of progress in therapy, the realization of other emotionally charged memories can be challenging for some clients to deal with, especially when there are implications for them in the current context of their lives. For example, a client who idealized his or her parents through the eyes of a child may have to see them as good people who made mistakes, as in the case of Laura. Clients are educated about what to potentially expect from the processing (e.g., that the processing can continue and that other memories, thoughts, emotions, and so forth can arise) and how to deal with it. For example, clients are instructed to utilize the calming techniques and coping strategies previously taught and to keep a log or journal of what comes up (e.g., dreams, other memories) and triggering situations. Other situations, however, may require additional strategies to help the client fully adopt an adult understanding of their childhood experiences or, as in the case of Bill, how to move on in their lives given the tragedy they endured. Although what happened in the past can't be changed, how we relate to it in the present and going forward into the future can be.

SUMMARY AND CONCLUSION

EMDR therapy is an eight-phase, three-pronged protocol (past, present, and future) that provides an evidence-based treatment for trauma, applicable to a wide range of disorders. It is guided by the AIP model, which states that present problems are informed by past memories that are inadequately processed and maladaptively stored in the brain. Present situations that resemble these past, unprocessed memories trigger negative reactions that are disproportionate to the current situation. Thus adverse reactions to current distressing events are understood as being informed by these maladaptively stored memories that contain negative images, thoughts, feelings, and sensations. Standardized EMDR therapy procedures can be utilized to process these past memories underlying the present problems; to address present situations that trigger the maladaptive response; and to generate future templates of adaptive responses to similar situations.

EMDR therapy is a comprehensive psychotherapy approach applicable to a broad range of clinical presentations, from the most severely traumatized client suffering from PTSD to a client who is struggling with acute or chronic stress and is compromised in his or her ability to manage it effectively. Although there are inherent stressors in any set of circumstances, it is more often what we bring to these situations that further challenges our ability to cope. EMDR therapy addresses the variables that clients bring to their circumstances, eliminating the emotional load from those past experiences, restoring their resiliency, and optimizing their capacity to cope effectively given their abilities and the actual circumstances they find themselves in. It is in our capacity to be fully present to the here and now of our lives that gives all of us the opportunity to move forward into the future.

REFERENCES

Abbasnejad, M., Mahani, K. N., & Zamyad, A. (2007). Efficacy of "eye movement desensitization and reprocessing" in reducing anxiety and unpleasant feelings due to earthquake experience. *Psychological Research, 9,* 104–117.

Abel, N. J., & O'Brien, J. M. (2010). EMDR treatment of comorbid PTSD and alcohol dependence: A case example. *Journal of EMDR Practice and Research, 4,* 50–59.

Acarturk, C., Konuk, E., Cetinkaya, M., Senay, I., Sijbrandij, M., Gulen, B., et al. (2016). The efficacy of eye movement desensitization and reprocessing for post-traumatic stress disorder and depression among Syrian refugees: Results of a randomized controlled trial. *Psychological Medicine, 46*(12), 2583–2593.

Ahmad, A., Larsson, B., & Sundelin-Wahlsten, V. (2007). EMDR treatment for children with PTSD: Results of a randomized controlled trial. *Nordic Journal of Psychiatry, 61,* 349–354.

American Psychiatric Association. (2004). *Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder.* Arlington, VA: Author.

Arabia, E., Manca, M. L., & Solomon, R. M. (2011). EMDR for survivors of life-threatening cardiac events: Results of a pilot study. *Journal of EMDR Practice and Research, 5*(1), 2–13.

Bae, H., Han, C., & Kim, D. (2015). Desensitization of triggers and urge reprocessing for pathological gambling: A case series. *Journal of Gambling Studies, 31,* 331–342.

Bloomgarden, A., & Calogero, R. M. (2008). A randomized experimental test of the efficacy of EMDR treatment on negative body image in eating disorder inpatients. *Eating Disorders, 16,* 418–427.

Brown, K. W., McGoldrick, T., & Buchanan, R. (1997). Body dysmorphic disorder: Seven cases treated with eye movement desensitization and reprocessing. *Behavioural and Cognitive Psychotherapy, 25*(2), 203–207.

Brown, S. H., Gilman, S. G., Goodman, E. G., Adler-Tapia, R., & Freng, S. (2015). Integrated trauma treatment in drug court: Combining EMDR and seeking safety. *Journal of EMDR Practice and Research, 9,* 123–136.

Capezzani, L., Ostacoli, L., Cavallo, M., Carletto, S., Fernandez, I., Solomon, R., et al. (2013). EMDR and CBT for cancer patients: Comparative study of effects on PTSD, anxiety, and depression. *Journal of EMDR Practice and Research, 10*(2), 70–81.

Carletto, S., & Pagani, M. (2016). Neurobiological impact of EMDR in cancer. *Journal of EMDR Practice and Research, 10*(3), 153–161.

Carlson, J., Chemtob, C. M., Rusnak, K., Hedlund, N. L., & Muraoka, M. Y. (1998). Eye movement desensitization and reprocessing (EMDR): Treatment for combat-related post-traumatic stress disorder. *Journal of Traumatic Stress, 11,* 3–24.

Chemali, Z., & Meadows, M. (2004). The use of eye movement desensitization and reprocessing in the treatment of psychogenic seizures. *Epilepsy and Behavior, 5,* 784–787.

Chemtob, C. M., Nakashima, J., Hamada, R. S., & Carlson, J. G. (2002). Brief treatment for elementary school children with disaster-related posttraumatic stress disorder: A field study. *Journal of Clinical Psychology, 58,* 99–112.

Cox, R. P., & Howard, M. D. (2007). Utilization of EMDR in the treatment of sexual addiction: A case study. *Sexual Addiction and Compulsivity, 14,* 1.

Craske, M., Herman, D., & Vansteenwegen, D. (Eds.). (2006). *Fear and learning: From basic processes to clinical implications.* Washington, DC: APA Press.

de Bont, P. A., van Minnen, A., & de Jongh, A. (2013). Treating PTSD in patients with psychosis: A within-group controlled feasibility study examining the efficacy and safety of evidence-based PE and EMDR protocols. *Behavior Therapy, 44*(4), 717–730.

de Jongh, A. (2012). Treatment of a woman with emetophobia: A trauma-focused approach. *Mental Illness, 4,* 10–14.

de Jongh, A., Holmshaw, M., Carswell, W., & van Wijk, A. (2010). Usefulness of a trauma-focused treatment approach for travel phobia. *Clinical Psychology and Psychotherapy, 18,* 124–137.

de Jongh, A., & ten Broeke, E. (1998). Treatment of choking phobia by targeting traumatic memories with EMDR: A case study. *Clinical Psychology and Psychotherapy, 5,* 264–269.

de Jongh, A., van den Oord, H. J. M., & ten Broeke, E. (2002). Efficacy of eye movement desensitization and reprocessing (EMDR) in the treatment of specific phobias: Four single-case studies on dental phobia. *Journal of Clinical Psychology, 58,* 1489–1503.

de Roos, C., & de Jongh, A. (2008). EMDR treatment of children and adolescents with a choking phobia. *Journal of EMDR Practice and Research, 2,* 201–211.

de Roos, C., Veenstra, A. C., de Jongh, A., den Hollander-Gijsman, M. E., van der Wee, N. J. A., Zitman, F. G., et al. (2010). Treatment of chronic phantom limb pain using a trauma-focused psychological approach. *Pain Research and Management, 15,* 65–71.

Department of Veterans Affairs & Department of Defense. (2017). *VA/DoD clinical practice guideline for the management of posttraumatic stress disorder and acute stress disorder.* Washington, DC: Author.

Diehle, J., Opmeer, B. C., Boer, F., Mannarino, A. P., & Lindauer, R. J. (2014). Trauma-focused cognitive behavioral therapy or eye movement desensitization and reprocessing: What works in children with posttraumatic stress symptoms? A randomized controlled trial. *European Child and Adolescent Psychiatry, 226,* 227–236.

Doering, S., Ohlmeier, M. C., de Jongh, A., Hofmann, A., & Bisping, V. (2013). Efficacy of a trauma-focused treatment approach for dental phobia: A randomized clinical trial. *European Journal of Oral Sciences, 121,* 584–593.

Farretta, E. (2013). EMDR and cognitive behavioral therapy in the treatment of panic disorder: A comparison. *Journal of EMDR Practice and Research, 7,* 121–133.

Farretta, E., & Civilotti, C. (2016). EMDR therapy in psycho-oncology: A bridge between mind and body. *Journal of EMDR Practice and Research, 10,* 138–152.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., et al. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The adverse childhood experiences (ACE) study. *American Journal of Preventive Medicine, 14,* 245–258.

Feske, U., & Goldstein, A. (1997). Eye movement desensitization and reprocessing treatment for panic disorder: A controlled outcome and partial dismantling study. *Journal of Consulting and Clinical Psychology, 36,* 1026–1035.

Gerhardt, A., Leisner, S., Hartmann, M., Janke, S., Seidler, G. H., Eich, W., et al. (2016). Eye movement desensitization and reprocessing vs. treatment-as-usual for non-specific chronic back pain patients with psychological trauma: A randomized controlled pilot study. *Frontiers in Psychiatry, 7,* 201.

Gil-Jardiné, C., Gil-Jardiné, C., Evrard, G., Al Joboory, S., Tortes Saint Jammes, J., Masson, F., et al. (2018). Emergency room intervention to prevent post concussion-like symptoms and post-traumatic stress disorder: A pilot randomized controlled study of a brief eye movement desensitization and reprocessing intervention versus reassurance or usual care. *Journal of Psychiatric Research, 103,* 229–236.

Grant, M. (1999). *Pain control with EMDR.* New Hope, PA: EMDR Humanitarian Assistance Program.

Grant, M., & Threlfo, C. (2002). EMDR in the treatment of chronic pain. *Journal of Clinical Psychology, 58,* 1505–1520.

Gupta, D., Gupta, N., & Choudhary, T. (2014). Effectiveness of the EMDR therapy on specific phobia in young children. *Delhi Psychiatry Journal, 17,* 175–178.

Hase, M., Schallmayer, S., & Sack, M. (2008). EMDR reprocessing of the addiction memory: Pretreatment, posttreatment, and 1-month follow-up. *Journal of EMDR Practice and Research, 2,* 170–179.

Henry, S. L. (1996). Pathological gambling: Etiological considerations and treatment efficacy of eye movement desensitization/reprocessing. *Journal of Gambling Studies, 12,* 395–405.

Högberg, G., Pagani, M., Sundin, O., Soares, J., Aberg-Wistedt, A., Tärnell, B., et al. (2008). Treatment of post-traumatic stress disorder with eye movement desensitization and reprocessing: Outcome is stable in 35-month follow-up. *Psychiatry Research, 159,* 101–108.

Hughes, M. (2014). EMDR as a therapeutic treatment for complex regional pain syndrome: A case report. *Journal of EMDR Practice and Research, 8,* 66–73.

Ironson, G. I., Freund, B., Strauss, J. L., & Williams, J. (2002). A comparison of two treatments for traumatic stress: A pilot study of EMDR and prolonged exposure. *Journal of Clinical Psychology, 58,* 113–128.

International Society for Traumatic Stress Studies. (2018). New ISTSS prevention and treatment guidelines. Retrieved from *www.istss.org/treating-trauma/new-istss-prevention-and-treatment-guidelines.aspx.*

Jaberghaderi, N., Greenwald, R., Rubin, A., Dolatabadim, S., & Zand, S. O. (2004). A comparison of CBT and EMDR for sexually abused Iranian girls. *Clinical Psychology and Psychotherapy, 11,* 358–368.

Jarero, I., Artigas, L., & Luber, M. (2011). The EMDR protocol for recent critical incidents: Application in a disaster mental health continuum of care context. *Journal of EMDR Practice and Research, 5,* 82–94.

Jarero, I., Artigas, L., Uribe, S., & Garcia, L. E.

(2016). The EMDR integrative group treatment protocol for patients with cancer. *Journal of EMDR Practice and Research, 10,* 199–207.

Kelley, S. D. M., & Selim, B. (2007). Eye movement desensitization and reprocessing in the psychological treatment of trauma-based psychogenic non-epileptic seizures. *Clinical Psychology and Psychotherapy, 14,* 135.

Kemp, M., Drummond, P., & McDermott, B. (2010). A wait-list controlled pilot study of eye movement desensitization and reprocessing (EMDR) for children with post-traumatic stress disorder (PTSD) symptoms from motor vehicle accidents. *Clinical Child Psychology and Psychiatry, 15,* 5–25.

Konuk, E., Epözdemir, H., Atçeken, S. H., Aydın, Y. E., & Yurtsever, A. (2011). EMDR treatment of migraine. *Journal of EMDR Practice and Research, 5,* 166–176.

Korn, D. L., & Leeds, A. M. (2002). Preliminary evidence of efficacy for EMDR resource development and installation in the stabilization phase of treatment of complex posttraumatic stress disorder. *Journal of Clinical Psychology, 58,* 1465–1487.

Lee, C. W., & Cuijpers, P. (2013). A meta-analysis of the contribution of eye movements in processing emotional memories. *Journal of Behavior Therapy and Experimental Psychiatry, 44*(2), 231–239.

Littel, M., van den Hout, M. A., & Engelhard, I. M. (2016). Desensitizing addiction: Using eye movements to reduce the intensity of substance abuse related mental imagery and craving. *Frontiers in Psychiatry, 7,* 14.

Litz, B. T., Stein, N., Delaney, E., Lebowitz, L., Nash, W. P., Silva, C., et al. (2009). Moral injury and moral repair in war veterans: A preliminary model and intervention strategy. *Clinical Psychology Review, 29,* 695–706.

Marcus, S. V. (2008). Phase 1 of integrated EMDR: An abortive treatment for migraine headaches. *Journal of EMDR Practice and Research, 2,* 15–25.

Marcus, S. V., Marquis, P., & Sakai, C. (1997). Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy, 34,* 307–315.

Marcus, S. V., Marquis, P., & Sakai, C. (2004). Three- and 6-month follow-up of EMDR treatment of PTSD in an HMO setting. *International Journal of Stress Management, 11,* 195–208.

Maroufi, M., Zamani, S., Izadikhah, Z., Marofi, M., & O'Connor, P. (2016). Investigating the effect of eye movement desensitization and reprocessing (EMDR) on postoperative pain intensity in adolescents undergoing surgery: A randomized controlled trial. *Journal of Advanced Nursing, 72,* 2207–2217.

Marr, J. (2012). EMDR treatment of obsessive–compulsive disorder: Preliminary research. *Journal of EMDR Practice and Research, 6,* 2–15.

Marsden, Z. (2016). EMDR treatment of obsessive–compulsive disorder: Three cases. *Journal of EMDR Practice and Research, 10.* 91–103.

Mazzola, A., Calcagno, M. L., Goicochea, M. T., Pueyrredòn, H., Leston, J., & Salvat, F. (2009). EMDR in the treatment of chronic pain. *Journal of EMDR Practice and Research, 3,* 66–79.

Mol, S. S., Arntz, A., Metsemakers, J. F., Dinant, G. J., Vilters-van Montfort, P. A., & Knottnerus, J. A. (2005). Symptoms of post-traumatic stress disorder after non-traumatic events: Evidence from an open population study. *British Journal of Psychiatry, 186*, 494–499.

Morrissey, M. (2013). EMDR as an integrative therapeutic approach for the treatment of separation anxiety. *Journal of EMDR Practice and Research, 7,* 200–207.

Nazari, H., Momeni, N., Jariani, M., & Tarrahi, M. J. (2011). Comparison of eye movement desensitization and reprocessing with citalopram in treatment of obsessive–compulsive disorder. *International Journal of Psychiatry in Clinical Practice, 15,* 270–274.

Newgent, R. A., Paladino, D. A., & Reynolds, C. A. (2006). Single session treatment of non-traumatic fear of flying with eye movement desensitization reprocessing: Pre- and post-September 11. *Clinical Case Studies, 5,* 25–36.

Nijdam, M. J., Gersons, B. P. R., Reitsma, J. B., de Jongh, A., & Olff, M. (2012). Brief eclectic psychotherapy v. eye movement desensitisation and reprocessing therapy in the treatment of post-traumatic stress disorder: Randomised controlled trial. *British Journal of Psychiatry, 200,* 224–231.

Novo, P., Landin-Romero, R., Radua, J., Vicens, V., Fernandez, I., Garcia, F., et al. (2014). Eye movement desensitization and reprocessing therapy in subsyndromal bipolar patients with a history of traumatic events: A randomized, controlled pilot study. *Psychiatry Research, 219,* 122–128.

Perez-Dandieu, B., Lenoir, H., Othily, E., Tapia, G., Cassen, M., & Delile, J. M. (2015). The impact of eye movement desensitization and reprocessing and schema therapy on addiction severity among a sample of French women suffering from PTSD and SUD. *Drug and Alcohol Dependence, 146,* 68–69.

Power, K. G., McGoldrick, T., Brown, K., Buchanan, R., Sharp, D., Swanson, V., et al. (2002). A controlled comparison of eye movement desensitization and reprocessing versus exposure plus cognitive restructuring, versus waiting list in the treatment of post-traumatic stress disorder. *Journal of Clinical Psychology and Psychotherapy, 9,* 299–318.

Rathschlag, M., & Memmert, D. (2014). Reducing anxiety and enhancing physical performance by using an advanced version of EMDR: A pilot study. *Brain and Behavior, 4,* 348–355.

Ricci, R., & Clayton, C. (2008). Trauma resolution treatment as an adjunct to standard treatment for child molesters. *Journal of EMDR Practice and Research 2*(1), 41–50.

Rothbaum, B. O. (1997). A controlled study of eye movement desensitization and reprocessing for posttraumatic stress disordered sexual assault victims. *Bulletin of the Menninger Clinic, 61,* 317–334.

Rothbaum, B. O., Astin, M. C., & Marsteller, F. (2005). Prolonged exposure versus eye movement desensitization and reprocessing (EMDR) for PTSD rape victims. *Journal of Traumatic Stress, 18*(6), 607–616.

Rougemont-Bucking, A., & Zimmermann, E. N. (2012). EMDR-based treatment of psychotraumatic antecedents in illicit drug abusers: A report of two cases. *Swiss Archives of Neurology and Psychiatry, 163,* 107–115.

Royle, L. (2008). EMDR as a therapeutic treatment for chronic fatigue syndrome (CFS). *Journal of EMDR Practice and Research, 2,* 226–232.

Russell, M. C. (2008). Treating traumatic amputation-related phantom limb pain. *Clinical Case Studies, 7,* 136–153.

Scheck, M. M., Schaeffer, J. A., & Gillette, C. S. (1998). Brief psychological intervention with traumatized young women: The efficacy of eye movement desensitization and reprocessing. *Journal of Traumatic Stress, 11,* 25–44.

Schneider, J., Hofmann, A., Rost, C., & Shapiro, F. (2008). EMDR in the treatment of chronic phantom limb pain. *Pain Medicine, 9,* 76–82.

Schubert, S. J., Lee, C. W., & Drummond, P. D. (2016). Eye movements matter, but why?: Psychophysiological correlates of EMDR therapy to treat trauma in Timor-Leste. *Journal of EMDR Practice and Research, 10*(2), 70–81.

Shapiro, E., & Laub, B. (2015). Early EMDR intervention following a community critical incident: A randomized clinical trial. *Journal of EMDR Practice and Research, 9*(1), 17–27.

Shapiro, F. (1989). Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories. *Journal of Traumatic Stress, 2,* 199–223.

Shapiro, F. (1991). Eye movement desensitization and reprocessing procedure: From EMD to EMD/R: A new treatment model for anxiety and related traumata. *Behavior Therapist, 14,* 133–135.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols and procedures.* New York: Guilford Press.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols and procedures* (2nd ed.). New York: Guilford Press.

Shapiro, F. (2007). EMDR, adaptive information processing, and case conceptualization. *Journal of EMDR Practice and Research, 1,* 68–87.

Shapiro, F. (2014). The role of eye movement desensitization and reprocessing (EMDR) therapy in medicine: Addressing the psychological and physical symptoms stemming from adverse life experiences. *Permanente Journal, 18,* 71–77.

Shapiro, F. (2018). *Eye movement desensitization and reprocessing (EMDR) therapy: Basic principles, protocols and procedures* (3rd ed.). New York: Guilford Press.

Shapiro, F., & Laliotis, D. (2011). EMDR and the adaptive information processing model: Integrative treatment and case conceptualization. *Clinical Social Work Journal, 39,* 191–200.

Shapiro, F., & Laliotis, D. (2015). EMDR therapy for trauma-related disorders. In M. Cloitre & U. Schnyder (Eds.), *Evidence based treatments for trauma-related psychological disorders: A practical guide for clinicians.* New York: Springer.

Shapiro, F., & Solomon, R. (2017). Eye movement desensitization and reprocessing (EMDR) therapy. In S. N. Gold, J. M. Cook, & C. J. Dalenberg (Eds.), *Handbook of trauma psychology: Vol. 2. Trauma practice.* Washington, DC: American Psychological Association.

Shapiro, F., Vogelmann-Sine, S., & Sine, L. (1994). Eye movement desensitization and reprocessing: Treating trauma and substance abuse. *Journal of Psychoactive Drugs, 26,* 379–391.

Solomon, E. P., Solomon, R. M., & Heide, K. M. (2009). EMDR: An evidence-based treatment for victims of trauma. *Victims and Offenders, 4,* 391–397.

Solomon, R., & Shapiro, F. (2008). EMDR and the adaptive information processing model: Potential mechanisms of change. *Journal of EMDR Practice and Research, 4,* 315–325.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58,* 61–75.

Suzuki, A., Josselyn, S. A., Frankland, P. W., Masushige, S., Silva, A. J., & Kida, S. (2004). Memory reconsolidation and extinction have distinct temporal and biochemical signatures. *Journal of Neuroscience, 24,* 4787–4795.

Tsoutsa, A., Fotopoulos, D., Zakynthinos, S., & Katsaounou, P. (2013). Treatment of tobacco addiction using the Feeling-State Addiction Protocol (FSAP) of the eye movement desensitization and reprocessing (EMDR) treatment. *Tobacco Induced Diseases, 12*(Suppl. 1), A25.

van den Berg, D. P. G., de Bont, P. A. J. M., van der Vleugel, B. M., de Roos, C., de Jongh, A., van Minnen, A., et al. (2015). Prolonged exposure vs. eye movement desensitization and reprocessing vs. waiting list for posttraumatic stress disorder in patients with a psychotic disorder: A randomized clinical trial. *Journal of the American Medical Association Psychiatry, 72*(3), 259–267.

Van den Berg, D. P. G., & van den Gaag, M. (2012). Treating trauma in psychosis with EMDR: A pilot study. *Journal of Behavior Therapy and Experimental Psychiatry, 43,* 664–671.

Van den Hout, M. A., & Engelhard, I. M. (2012). How does EMDR work? *Journal of Experimental Psychopathology, 3,* 724–738.

Van der Hart, O., Groenendijk, M., Gonzalez, A., Mosquera, D., & Solomon, R. (2013). Dissociation of the personality and EMDR therapy in complex trauma-related disorders: Applications in phase 1 treatment. *Journal of EMDR Practice and Research, 7,* 81–94.

Van der Hart, O., Groenendijk, M., Gonzalez, A., Mosquera, D., & Solomon, R. (2014). Dissociation of the personality and EMDR therapy in complex trauma-related disorders: Applications in phase 2 and 3 treatment. *Journal of EMDR Practice and Research, 8,* 33–38.

van der Kolk, B. A., Spinazzola, J., Blaustein, M. E., Hopper, J. W., Hopper, E. K., Korn, D. L., et al. (2007). A randomized clinical trial of eye movement desensitization and reprocessing (EMDR), fluoxetine, and pill placebo in the treatment of posttraumatic stress disorder: Treatment effects and long-term maintenance. *Journal of Clinical Psychiatry, 68*(1), 37–46.

van Rood, Y. R., & de Roos, C. (2009). EMDR in the treatment of medically unexplained symptoms: A systematic review. *Journal of EMDR Practice and Research, 3*(4), 248–263.

Vogelmann-Sine, S., Sine, L., & Smyth, N. (1999). EMDR to reduce stress and trauma-related symptoms during recovery from chemical dependency. *International Journal of Stress Management, 6*(4), 285–290.

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1995). Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *Journal of Consulting and Clinical Psychology, 63,* 928–937.

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1997). Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for PTSD and psychological trauma. *Journal of Consulting and Clinical Psychology, 65,* 1047–1056.

World Health Organization. (2013). *Guidelines for the management of conditions that are specifically related to stress.* Geneva, Switzerland: Author.

Zweben, J., & Yeary, J. (2006). EMDR in the treatment of addiction. *Journal of Chemical Dependency Treatment, 8,* 115–127.

CHAPTER 9

Neurofeedback with Biofeedback for Stress Management

Michael Thompson
Lynda Thompson

SCIENTIFIC BASIS AND HISTORY OF NEUROFEEDBACK

Neurofeedback (NFB) is a learning technique that allows one to change brain-wave patterns in a healthy direction. Understanding that there is a link between brain activation patterns and a person's mental state and behavior goes back almost a century to Hans Berger's publication in 1929 concerning the human electroencephalogram (EEG). About 50 years later, research done by M. Barry Sterman (Sterman & Thompson, 2014) showed that EEG patterns can be trained in both animals and humans. These two ideas underlie using NFB for stress management because there are particular brain-wave signatures that are associated with anxiety and with ruminating, mental states that are associated with stress, and other patterns that appear when a person is feeling calm. With NFB training, an individual becomes able to self-regulate emotions and attention and thus better manage his or her response to stress.

A German psychiatrist, Hans Berger, studied electrical activity in the human brain in the 1920s and called his recordings *Das Elektroenkephalogramm*, or EEG. He established that different patterns were seen depending on a person's mental state. He called the high amplitude, sinusoidal waves that appeared in the eyes-closed, resting state *first-order waves*, or "alpha." The smaller amplitude, faster, uneven waves that appeared when a person was mentally active were termed *second-order waves*, or "beta" (Berger, 1929). These Greek letter names are still used, with alpha waves running at about 8–12 cycles per second (Hz) and beta waves being above 13 Hz. Slower frequencies were later named delta (1–4 Hz) and theta (4–8 Hz), whereas gamma frequencies are above 30 Hz.

Two lines of research led to the development of brain-wave training, which came to be called EEG biofeedback or NFB. One investigated consciousness and self-awareness of mental states (Kamiya, 1968, 1979) and the other applied operant conditioning techniques to change brain-wave patterns (Wyrwicka & Sterman, 1968; Sterman, 2000a), so both influences came out of research labs. In the mid-1950s, Joe Kamiya, at that time a young psychologist at the University of Chicago, demonstrated that participants could identify when they were producing a mental state that was related to particular brain-wave frequencies called *alpha*. This work, begun in the mid-1950s, eventually led to considerable research about this rhythmic, high-amplitude, sinusoidal activity that stands out when you look at an EEG recording. Most of the studies on alpha have concerned relaxed states, because alpha is an inverse indicator of activation; that is, when alpha is present in an area of the brain, that area is resting or in standby mode. This is easily seen when a person closes his or her eyes: There is a marked increase in alpha in the occipital region in which visual processing is done. Studies concerning the treatment of anxiety using NFB have usually indicated successful outcomes when alpha was increased over central locations (Hardt & Kamiya, 1978; Rice, Blanchard, & Purcell, 1993). Some participants, however, do not benefit from increasing alpha but, instead, improve when alpha is decreased (Plotkin & Rice, 1981; Rice et al., 1993; Thomas & Sattlberger, 2001). Those cases are usually people who have high amplitudes of alpha to begin with and in whom raising it further can increase anxiety. It is therefore important when doing NFB to do a baseline assessment and individualize the training.

Research establishing that it was possible to train study participants to produce specific brain-wave patterns was published a decade after Kamiya's early experiments showing that people could develop awareness of when they were producing alpha. It came from an entirely different line of investigation conducted on animals. Brain-wave training using operant conditioning (rewarding the behavior being trained) was done first in cats and monkeys, then in humans (Wyrwicka & Sterman, 1968; Sterman, 2000a). M. Barry Sterman, a sleep researcher at the University of California at Los Angeles and the Sepulveda Veterans Administration Hospital, had noticed that cats, waiting for a signal before making a response that produced food, showed brain-wave patterns that resembled sleep spindles. He set up experiments that rewarded the cats for producing bursts of that activity, which he named *sensorimotor rhythm,* usually shortened to SMR. SMR is characterized by bursts of rhythmic, spindle-like EEG activity at frequencies between approximately 12 and 15 Hz (the low end of the beta range) measured across the somatosensory and motor cortex of the brain, which is located across the top of the head at sites called C3, Cz, and C4 in the International 10–20 Electrode Placement System, which is shown in Figure 9.1.

Once it had been shown that operant conditioning techniques that involve rewarding desired brain-wave behavior could be used to train cats to produce higher amplitudes of SMR, the learning technique known as NFB, or EEG biofeedback, was ripe for further investigation. This kind of learning is based on Thorndike's law of effect: When a behavior is rewarded, it is more likely to recur. After Sterman published his research on training the SMR rhythm in cats, he began an unrelated study funded by the National Aeronautics and Space Administration (NASA) concerning a component of rocket fuel, hydrazine, that could produce seizures. During his research involving hydrazine-induced seizures in laboratory cats, Sterman discovered that the cats that had previously been trained to increase sensorimotor rhythm were resistant to developing seizures. Sterman's research then shifted to include working with humans who had seizure disorders. Through his research and replication of his results at other labs, it was established that EEG operant conditioning of particular brain-wave frequencies in patients who had epilepsy that was not well controlled by medications was associated with a reduction in the frequency, duration, and intensity of seizures in the majority of participants. Indeed, there was about

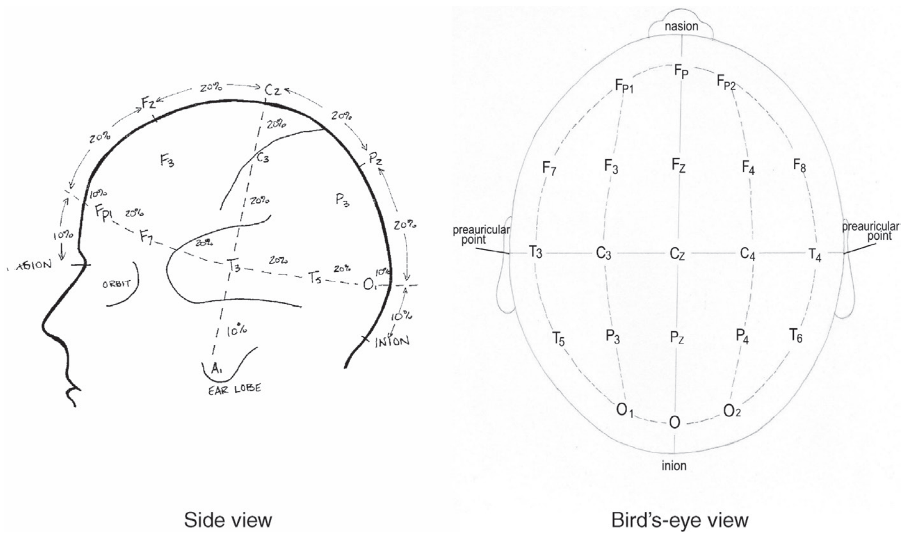


FIGURE 9.1. International 10–20 Electrode Placement System. Neurologists use updated designations for some sites; namely, T3 becomes T7, and T5 becomes P7 on the left. On the right, T4 becomes T8, and T6 becomes P8. From Thompson and Thompson (2015), *The Neurofeedback Book* (2nd ed.). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

a 75% positive response rate across the various studies involving participants who had NFB training in addition to taking their usual medications. For more detail on research involving NFB for epilepsy, see the two meta-analyses (Sterman, 2000b; Tan et al., 2009) and a chapter detailing the use of SMR training for patients with epilepsy (Sterman & Thompson, 2014).

By the beginning of the 21st century, NFB was well established for the treatment of attention-deficit/hyperactivity disorder (ADHD) and seizure disorders, and there were preliminary studies applying NFB to other disorders, such as depression, anxiety, and postconcussion syndrome. Applications were reviewed in a special edition of *Clinical Electroencephalography* (Sterman, 2000b), in which the editor noted that if any medication showed the beneficial effects seen for NFB, every physician would know about it. For efficacy levels of NFB in various disorders, see *Evidence-Based Practice in Biofeedback and Neurofeedback* (Tan, Shaffer, Lyle, & Teo, 2016), and, for a listing of articles, see the bibliography available on the website of the International Society for Neurofeedback and Research (*www.isnr.org*), which is organized by conditions being treated. A readable and detailed account of the history of NFB is found in the popular book *A Symphony in the Brain* (Robbins, 2000).

In the 1970s, Joel Lubar, a professor at the University of Tennessee who was already investigating EEG in humans, applied these techniques to the assessment and training of clients who exhibited symptoms of hyperactivity (Lubar & Shouse, 1976; Lubar, 1991; Lubar, Congedo, & Askew, 2003; Lubar & Lubar, 1999). EEG changes, improved behavior, IQ increases, and improved academic performance have been reported in studies using NFB in clients with ADHD (Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Linden, Habib, & Radojevic, 1996; Thompson & Thompson, 1998). Controlled research concerning children with ADHD that used functional magnetic resonance images (fMRI) as the pre–post measure has documented changes in specific brain areas (frontal lobes, basal ganglia, substantia nigra, and anterior cingulate) after NFB training (Beauregard & Levesque, 2006). More recent research showed increased volume of both gray and white matter, using structural MRI for gray matter measurements and diffusion tensor imaging (fractional anistrophy) to measure white matter. Increased volumes were documented after just 20 sessions of NFB training done with university students (Ghaziri et al., 2013). Evidence is strong enough, based on several randomized controlled trials (RCTs), that some reviewers feel that NFB for ADHD has the highest level of efficacy, being both efficacious and superior to other interventions because results last and there are no negative side effects (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). Though the precise mechanisms that produce improvements have not been identified, it is clear that NFB, properly applied, produces changes in neural circuits. These changes in the brain correlate with positive changes in behavior. The basic textbook by Thompson and Thompson (2015a) describes assessment and treatment of numerous symptoms, syndromes, and disorders, as well as the use of NFB for improving performance in athletes and executives.

Using NFB for stress management is considered experimental because RCTs are still needed. The same is true for using NFB for depression and traumatic brain injury, which have a current level 3 efficacy rating: "probably efficacious" (Tan et al., 2016). NFB does have great face validity; clients can see, by viewing feedback from a computer system that is monitoring their brain waves, how their EEG patterns correlate with their own perceptions of mental state—for example, relaxed versus tense, focused versus daydreaming.

SINGLE-CHANNEL EEG ASSESSMENT

Single-channel EEG assessment should be done to establish parameters for training before starting NFB, and we briefly describe that assessment here in order that the remainder of the chapter can be more easily understood. An additional 19-channel assessment may be helpful in complex cases. The overall goal for training involves the client's mastering self-regulation of his or her mental states, as reflected in his or her EEG pattern; so establishing an individual's patterns at the outset is essential.

One goal for assessment is to ascertain what is different from expected patterns in a client's EEG so that you may decide on parameters for an EEG training program. There are numerous variables to consider, such as comparing the power of the EEG in different frequency bands to an expected pattern. There are no quantitative norms available for single-channel EEG except for theta/beta power ratios (the squared ratio of 4–8 Hz/13–21 Hz activity). These are used when dealing with people who have symptoms of ADHD (Monastra et al., 1999; Monastra, Lubar, & Linden, 2001). Age norms are available for 19-channel EEG data collection; indeed, a number of different normative databases are available for purchase.

The NFB practitioner can gain a great deal of information from a single-channel assessment. This can be done with an "active" electrode placed at Cz, the vertex, as shown in Figure 9.1, and a "reference" electrode on one earlobe. The other earlobe is used for the "ground" electrode. Figure 9.2 shows an example of an assessment screen using Biograph software and the Infiniti instrument available from Thought Technology (Thought Technology Ltd., 8205 Boulevard Montréal-Toronto Suite #223, Montreal-West, QC H4X 1N1, Canada; *www.thoughttechnology.com*).

The screen is part of the Thompson Clinical Success Suite from the Biofeedback Foundation of Europe (*www.BFE.org*). The EEG with a bandpass filter from 2 to 40 Hz is at the top. The raw EEG is graphed as amplitude, measured in microvolts, by time. A mathematical fast Fourier transform (FFT) allows one to see the same information as a spectrum at the bottom of the screen. The spectrum shows the average power for each Hertz (1.0–1.99 Hz, 2.0–2.99 Hz, etc.) from the previous 1 second of EEG activity. It is graphed as power on the *Y*-axis by frequency on the *X*-axis. The red column in the spectrum shows the frequency with the highest power for that 1 second. It is therefore usually moving from one frequency to another as one watches the EEG while data are collected. The client is asked to relax the shoulders, breathe diaphragmatically, and keep the eyes steady by looking at a single spot at the bottom of the screen. This reduces eye movement and muscle contractions, both of which are sources of artifact in the EEG. While the EEG is recording for 3 minutes, the trainer will tap the space bar every time the client blinks, swallows, or makes some other movement of the head or face. That tap is recorded as a vertical line, which is helpful when later removing artifacts from the EEG. (For more information on artifacting, see Hammond & Gunkelman, 2001, and Thompson & Thompson, 2015a, Chapter 9.) After removing artifacts, the program calculates statistics for 1-Hz bins, and these are graphed, using the Excel program from Microsoft, as shown in Figure 9.3. The graphic displays can be very helpful in determining which frequency ranges to train for each client. Jay Gunkelman in lectures has suggested, "You act like a bulldozer, filling in dips and eliminating blips." The EEG pattern in Figure 9.3, for example, shows excess slow-wave activity (a "blip" up.) This boy's theta/beta power ratio was well above the Monastra–Lubar norms for children age 6 because ratios of 3 are normal and ratios above 5 are > 1.5 standard deviations above the mean for children without ADHD (Monastra et al., 1999). This single-channel EEG profile indicates that the child could benefit from NFB training that would reduce the excess theta and lower alpha frequencies (2–9 Hz) and increase SMR frequencies.

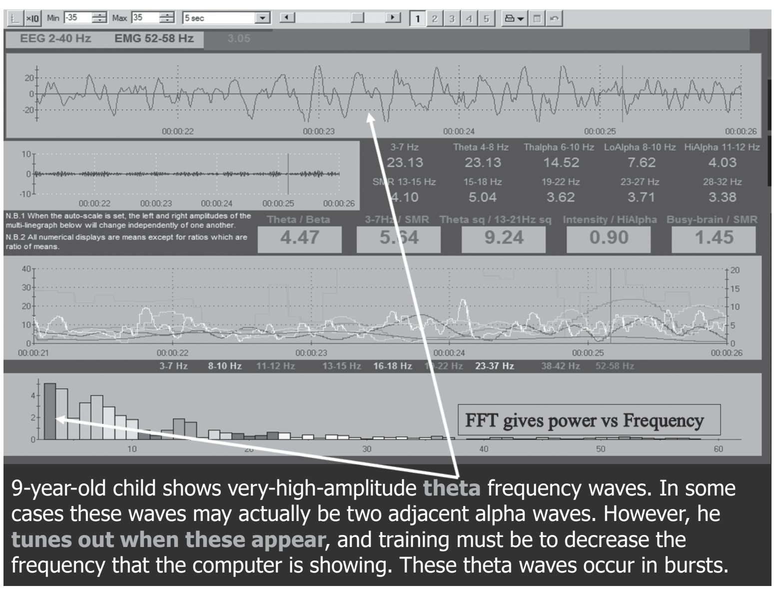


FIGURE 9.2. Screen with raw EEG, relevant ratios, and EEG power spectrum. Spectrum at the bottom shows the power in frequencies from 3 to 61 Hz.

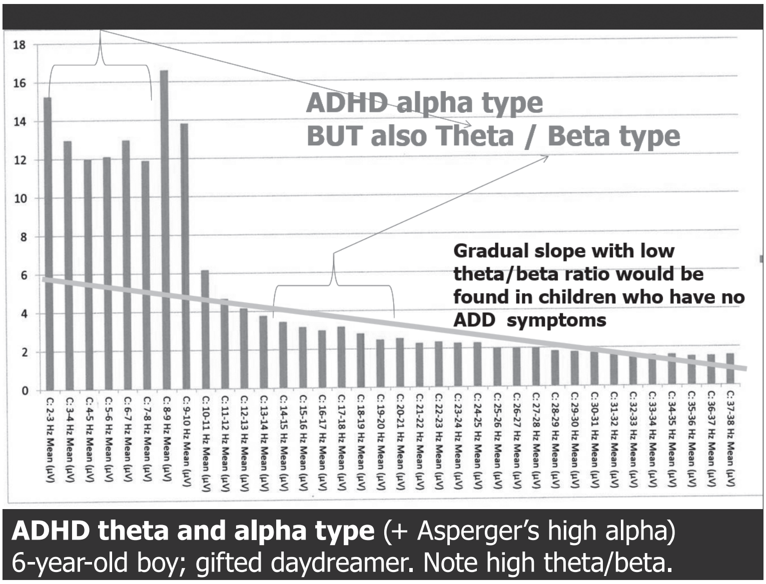


FIGURE 9.3. Single-channel (Cz) spectral graph, done in Excel, based on 3 minutes of artifacted data. *Y*-axis is magnitude/power. *X*-axis is frequency bins from 2 Hz to 38 Hz.

THEORETICAL FOUNDATIONS: USING NFB TO MANAGE STRESS

Underlying Principles

The same event can be stressful for one person and enjoyable for someone else; for example, many people are nervous about public speaking, but others enjoy it. A circumstance is stressful to the degree that it evokes a feeling of inability to cope—that is, a *personally perceived potential adaptive incompetency* (Thompson, 1978). It often involves a sense of crisis. The most severe stressors can be purely internal, such as repetitive negative thoughts or "flashbacks." For external stressors, performance (*Y*-axis) plotted against degree of stress (*X*-axis) produces an inverted *U*-shaped curve. As stress rises, performance initially increases with a small amount of stress, and then performance drops drastically as the stress gets higher. Hans Selye's (1976) concept of eustress involves maintaining an optimal level of stress represented by the top of the inverted *U*. This principle, also known as the Yerkes–Dodson law, first published in 1908, states that performance increases with arousal up to a certain point, but then decreases when arousal gets too high (Yerkes & Dodson, 1908). Stress cannot be avoided in life, but people can become aware of the appropriate level of stress for each circumstance and can try to remain within that range. Being resilient and returning to a healthy baseline after a stressful event is the goal in most cases.

The effects of stress on mental state are reflected in EEG patterns, as well as in other physiological variables, such as muscular tension (electromyogram), heart rate, respiration, the electrodermal response, and skin temperature. Stress also affects physical health (Aich, Potter, & Griebel, 2009), especially if it is prolonged. Stress can be acute or chronic. It can be the result of external events, such as heavy workload, natural disasters, loss, attacks (physical or verbal), and other stressors that are outside of one's control. It can be generated internally (money worries, feelings of embarrassment or guilt, obsessive thoughts). It can also have physical causation, as in illness or chronic pain. Regardless of the source, there are typically two options for managing a stress: take action to change the stressful situation or change the response. In some instances, one can and should do both. Changing one's response or attitude toward a stressor includes such things as cognitive reframing, that is, seeing the stressor in a new light, so that attitude/cognitive set changes. One can also consciously change the physiological response using such techniques as diaphragmatic breathing. Cognitive approaches—changing attitude or beliefs about the stressor—form the basis of cognitive-behavioral therapy (CBT) and are also basic to the steps developed in acceptance and commitment therapy (ACT). Step 3 in the ACT approach is a technique akin to mindfulness training: The person is connecting fully with whatever is happening right here, right now. The individual is totally engaged in whatever she or he is doing or experiencing at the moment, with a focus that is external; thus attention to negative thoughts and ruminations decreases. The corresponding pattern in the EEG when ruminations decrease is that the amplitude of any spindling beta above 20 Hz will decrease (Thompson & Thompson, 2006).

Both taking action and changing the response or attitude require appropriate, though different, mental states, as reflected in EEG patterns, and also an appropriate physiological state in order to be maximally effective. Training that includes NFB can give a person the mental edge he or she needs, with a flexible brain that can get in the appropriate zone to respond to various kinds of stress. Note that this is not a static state. It changes with task demands as the stressful situation unfolds. Thus the emphasis is on a flexible brain that can activate or inhibit various mental states, as needed. This is summed up in the motto of our ADD Center, Mississauga and Toronto, Canada: "You cannot change the wind, but you can adjust the sails."

Another principle upon which intervention for stress should be based comes from Roman times: *mens sana in corpore sano* ("a sound mind in a sound body"). This phrase exemplifies the idea of mind–body unity. With respect to stress management, this means that both mind and body must be flexible, that is, able to withstand stress, recover from stress, and return to a baseline characterized by a relaxed mind in tune with a relaxed body. Thus NFB is not a stand-alone intervention; rather, a comprehensive stress management approach involves training the brain (NFB), the mind (using cognitive strategies), and the body (using biofeedback modalities) for maximal effectiveness and resilience. Paying attention to diet, sleep, and exercise also plays a role in stress management.

Poor sleep is often a symptom of stress, and improving sleep is one of the goals of an NFB-plus-biofeedback intervention. Specifically, it usually involves increasing SMR through NFB training and practicing effortless diaphragmatic breathing using heart rate variability (HRV) training. Increasing SMR involves rewarding an increase in amplitude of 13-, 14-, and 15-Hz activity across the sensorimotor strip, most often using sites C3, C4, and Cz (Figure 9.1). Daytime training to increase SMR amplitude has been shown to increase sleep spindle activity at night, smoothing the transitions between stages of sleep and resulting in the person awakening feeling more refreshed. SMR training can be used as a treatment for insomnia. Improvement in sleep underlies a reduction in the symptoms observed in clients with both ADHD and epilepsy when NFB training is done (Sterman 2000a, 2000b).

Systems theory applies to NFB interventions. When altering any component in a system, the other elements in the system will shift their functions in order to accomplish the goal, or task, of that system. In keeping with this principle, we often find on reassessment, after 40 sessions of training, that functions that were not directly targeted have improved. A good example is improvement in social appropriateness in clients with ADHD and in those with Asperger's syndrome, in cases in which the main intervention was to encourage brain waves that reflect calm attentiveness to the outside world. Applying this to biofeedback for stress management, it is observed that, when the client changes one variable, such as breathing, other variables usually adjust in the expected direction. Slow diaphragmatic breathing will be accompanied by warmer hands and decreased arousal measured by skin conductance (SC). Individual differences do occur; for example, some clients with yoga or meditation training can consciously control their breathing at a perfect rate but without improvement in HRV, skin temperature, muscle tension, or EEG variables. They still look and feel "stressed out," as reflected in those other measures. The goal of intervention is to assist clients, using NFB combined with biofeedback, to change their mental and physiological states to calm and relaxed ones while efficiently and effectively accomplishing a task (Thompson & Thompson, 1998; Thompson, Thompson, & Reid, 2010; Thompson & Thompson, 2011). A 56-year-old lawyer who came for treatment of anxiety and ADHD reported, after completing 40 sessions of NFB and training in diaphragmatic breathing, that his working day was now about 2 hours shorter. Being calm and focused increased his efficiency.

The final principle that underlies work with NFB is that operant conditioning of measurable neuronal variables (such as amplitude variations in various brain-wave frequencies or changes in coherence that reflect shared activation between areas in the cortex) can directly alter the biochemical and physiological underpinnings of a client's response to stress. This can result in a long-term change in one's response to stressors in daily living. A summary of the body's stress response system is outlined below.

Overview of the Neurophysiology of the Stress Response

The stress response is an adaptive biological mechanism that evolved because it was important for survival of the human species. Ongoing stress, however, increases inflammation, lowers the immune response, and leaves one more vulnerable to illness (see Kusnecov, Norton, & Nissenbaum, Chapter 4, this volume).

The stress response is controlled by interactions from the brainstem (especially the locus coeruleus, where epinephrine is produced) through to the frontal lobes, as seen in Figure 9.4. The amygdala, a structure that becomes active with fear or threat, links to the hypothalamus–pituitary–adrenal (HPA) axis. The frontal lobes and hippocampus have a major effect on affect through connections to the amygdala and hypothalamus. This amygdala–HPA (A-HPA) axis influences the sympathetic nervous system and thus the fight-or-flight response. Smith-Pellettier (2002) notes that, when overstressed, this A-HPA system can become dysregulated—either over- or underactive. Hypoactivity may result in symptoms of anxiety, fibromyalgia, and chronic pain. Lower levels of the analgesic actions of hypothalamic corticotropin-releasing hormone (CRH) could contribute to pain. Hyperactivity of this A-HPA axis, with resultant increased CRH, has been implicated in depression. There is increased response sensitivity to noxious stimuli and decreased immune system response. The immune response is dampened by glucocorticoids (GC) if stress continues. Not only will chronic stress increase GC and suppress the immune response, but it may even lead to a reduction in the size of the thymus and a reduction in circulating lymphocytes. These negative changes are among the effects of *allostatic load,* a term for "the wear and tear on the body" that accumulates as an individual is exposed to repeated or chronic stress (McEwen & Stellar, 1993). For a discussion of allostatic load, see Lehrer, Chapter 2, this volume. For more information on the

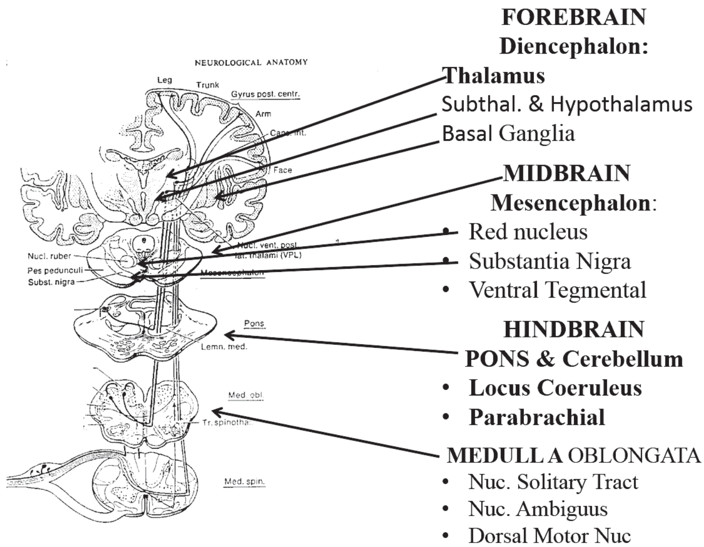


FIGURE 9.4. Location of selected nuclei. Figure from Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

stress response, dysregulation, and inflammatory and immune processes, see Kusnecov et al., Chapter 4, and Lehrer, Chapter 2, this volume.

The symptoms of overactivity in the A-HPA axis are familiar to anyone in the mental health field. These clients exhibit a narrowing of their perspective that, in turn, produces a focus on their own preoccupations, ruminative thinking, and poor cognitive performance. Due to stress, their sleep is impaired, which further compounds their difficulties. There are male–female differences in this process, with the A-HPA axis and CRH activity being generally higher in females. This might make females more susceptible to the negative effects of insomnia and stress. Aging is another factor, and the elderly experience frequent awakening, decreased growth hormone secretion, a decrease in deep or slow-wave sleep, and a concomitant decrease in immune regeneration and a reduction in the pain threshold. This and chronic fatigue can be a result of allostatic overload. The central nucleus of the amygdala is important in its potential to stimulate the locus coeruleus production of norepinephrine, which can activate the sympathetic responses of the autonomic system. Research indicates that the central nucleus of the amygdala (CNA) is inhibited by inputs from the superior temporal lobe and the fusiform gyrus (Porges, 2007). There is also tonic inhibition of this CNA from the medial aspect of the prefrontal cortex (Thayer, Hansen, Saus-Rose, & Johnson, 2009). Dysfunction in these areas can show up in 19-channel EEG assessments that include low-resolution electromagnetic tomography (LORETA) and LORETA coherence (connectivity) analysis. These areas may then become targets for LORETA *z*-score NFB treatment that targets normalization of significant amplitude and connectivity differences, indicated by deviations from database norms (*z*-scores are standard scores with a mean of 0 and a standard deviation of 1).

LORETA is a mathematical solution that can take information from 19 channels of EEG and calculate the source of the electrical activity deeper in the cortex. LORETA and its applications are discussed later in this chapter.

The principal components of these responses to stress related to the A-HPA axis are all unconscious/automatic. However, the cortex has a major input to this axis. Perhaps the most central and clearest controlling influence comes from the anterior cingulate. The anterior cingulate is an area of the cortex that has major linkages to the frontal lobes (particularly the medial and orbital surfaces of the frontal lobes) and is linked to all areas of the A-HPA axis and to other key control areas, such as the thalamus.

Figures 9.4, 9.5, and 9.6 are given to orient the reader with respect to important nuclei in the brainstem and to the effect of stress on the A-HPA axis.

Note that the negative (*-ve*) in Figure 9.5, shown by a dotted line indicating cortisol circulating, refers to inhibition by GCs from the adrenal cortex that will stop the normal stress response. With chronic stress, the principal effect is that this normal inhibition is no longer effective. In chronic stress, cortisol can weaken the activity of the immune system. Cortisol prevents proliferation of T cells, and cortisol may also inhibit memory retrieval of already stored information. Patterson and colleagues (2013) note that chronic stress causes reduced sensitivity of immune cells to cortisol. Such a loss of sensitivity would impair regulatory responses to dampen inflammation, affecting T cell activation and more rapid progression to advanced human immunodeficiency virus (HIV) disease (Patterson et al., 2013). Stress generally impairs long-term memory retrieval (Gagnon &

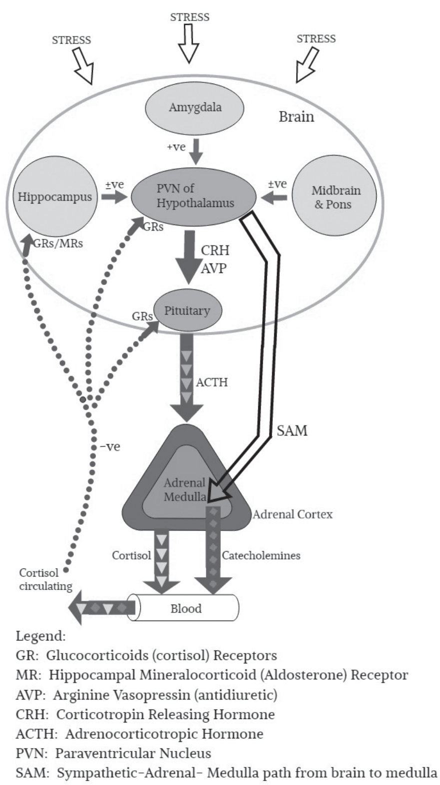


FIGURE 9.5. A-HPA axis with glucocorticoid receptor (GR) sites. Figure by Maya Berenkey, adapted from Randall (2010). From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

Wagner, 2016), whereas it can enhance memory encoding (Wiemers, Sauvage, Schoofs, & Hamacher-Dang, 2013), memory retention (Cahill, Gorski, & Le, 2003), and decision making (Shields, Sazma, & Yonelinas, 2016).

The adrenal medulla has cells that secrete epinephrine (adrenaline), norepinephrine (noradrenaline), and a small amount of dopamine in response to stimulation by sympathetic preganglionic neurons, and this process contributes to responses to stress. The adrenal medulla is partially under the control of the sensory and motor cortex, but the major control is the paraventricular nucleus of the hypothalamus (Aich et al., 2009). For further detail, see Carmichael and Stoddard (1992).

It is important to note, as illustrated in Figures 9.4 and 9.5, that the hypothalamus—in particular, the paraventricular nucleus of the hypothalamus—is a keystone in the normal stress response. The assessments that we use identify central nervous system (CNS) areas and brain networks and other systems, such as the cardiac system, that are not functioning within a normal range. NFB plus biofeedback interventions target dysfunctional areas and work to restore healthy patterns. The Brodmann areas (BAs) in the brain, which reflect different cell structures (cytoarchitecture), and connectivity between BAs become the principal targets for NFB interventions when doing LORETA *z*-score NFB (LNFB). Connectivity is shared activity, possibly from a common generator, and is measured in the NeuroGuide (*www.appliedneuroscience.com/neuroguide*) program by *coherence* and *phase*. Coherence is the mathematical measure of degree of similarity of the EEG recorded at two sites; that is, the degree to which activity at each frequency is phase locked. Coherence ranges from 0 to 1. If the phases—rising and falling—of the two signals are more similar over time (remain in the same time relationship to each other), then it suggests functional connectivity—that those two areas of the brain are working together. Like amplitudes, these coherence measurements can be compared with a normative database, and deviations from those database norms can then be expressed as a *z*-score (Catani & de Schotten, 2012). In addition to training the brain, HRV training (see Lehrer, Chapter 10, this volume) is added to recruit parasympathetic, calming influences. This dual effect on the same structures, outlined in Figure 9.6, is the reason we recommend combining HRV training with NFB interventions.

Posttraumatic Stress Disorder: A Special Form of Chronic Stress

Posttaumatic stress disorder (PTSD) has become an increasingly common diagnosis, affecting approximately 8% of the population in the United States. The trauma survivor who develops PTSD conceptualizes and experiences the present world through the lens of the past. The characteristic symptoms of PTSD include reexperiencing of traumatic memories through intrusive thoughts or nightmares, avoidance of trauma reminders, distress and physiological reactivity in response to reminders of trauma, hyperarousal, and, in some persons, emotional numbing and dysphoria. Veterans with PTSD show persistent problems in concentration and everyday memory. Scott and his colleagues (2015) note that a substantial literature has accumulated over the past 25 years showing performance deficits on neurocognitive measures of attention, working memory, episodic memory, speed of information processing, and executive functioning in individuals with PTSD. They note that the amygdala, hippocampus, cingulate cortex, and prefrontal cortex are critically involved in emotion processing and emotional memory formation, including the acquisition of fear and the establishment of emotional context and valence for memories (Scott et al., 2015). They also cite numerous publications that demonstrate that, in response to cognitive demands, individuals with PTSD do not show the hyperactivation

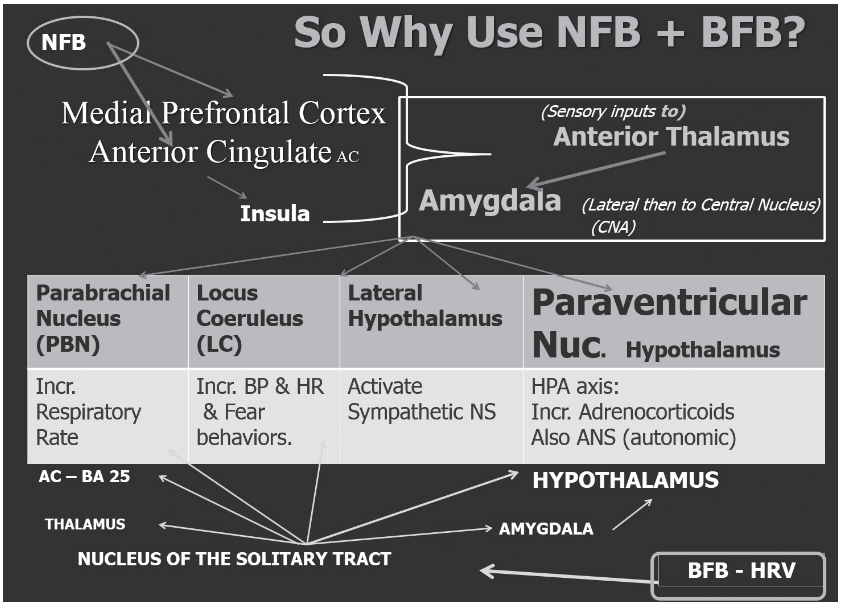


FIGURE 9.6. HRV and NFB can influence the same areas. In this figure, both NFB and HRV connect to the major endpoint that is the paraventricular nucleus of the hypothalamus (PVN). The PVN connects to both afferents and efferents of the sympathetic and parasympathetic portions of the ANS. It also connects to the HPA axis that is central to the stress response. Chronic stress overactivates this HPA axis, causing a state in which the production of norepinephrine from the locus coeruleus does not shut down in the manner that it would with a normal stress response. From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

of areas such as the cingulate gyrus that is found in other anxiety disorders but, rather, evidence hypoactivation of regions involved in attention, working memory, encoding, and executive processing. Hypoactivation was found in the dorsal anterior cingulate cortex (ACC) and dorsomedial prefrontal cortex (PFC), as well as in ventral portions of the ACC and the ventromedial PFC, but there was hyperactivation in the right insula (Rosso et al., 2014). In PTSD and in disorders with anxiety as a major problem, the rostral anterior cingulate is a key area (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006).

Heightened noradrenergic sensitivity, bias to threat, or hyperarousal may divert prefrontally mediated attentional resources to extraneous stimuli, which may disrupt goal-based attention and negatively affect encoding and retention of verbal information, as well as other cognitive processes involving PFC networks, such as sustaining focused attention (Posner, Sheese, Odludas, & Tang, 2006; Green et al., 2008). Executive functioning and speed of information processing rely on the integrity of PFC networks and efficient connectivity between frontal regions and other brain networks, and these functions can be affected in PTSD, as can the ability to inhibit inappropriate or automatic responses. A 19-channel EEG assessment can identify which of these areas are outside database norms for the individual patient, and LNFB can target areas with large deviations from the database norms, as well as the connectivity between them. HRV training will also affect these systems due to the heart–brain links outlined above and as described in Figure 9.6.

Much of the literature on PTSD has involved studies of war veterans, and this results in a large group with a comorbidity of traumatic brain injury (TBI). The persistent post-concussion symptoms found in up to 30% of people who suffer concussions overlap with many of the symptoms listed for PTSD (Howe, 2009). Not surprisingly, there will be dysfunction in the same areas of the brain that are mentioned above for PTSD. Major depression will also show many of the cognitive and affect difficulties that are noted above for PTSD, and depression is three times more likely in people who have suffered a concussion (Tator, 2013). A further comorbidity may be drug abuse. Detrimental effects on memory, attention, processing speed, visuospatial abilities, set shifting, and abstraction and conceptualization, even after months to years of abstinence, is possible with many substances. Preexisting ADHD can also be associated with the cited cognitive problems found in PTSD. It is generally accepted that there appears to be PTSD-related atrophy of the anterior cingulate, hippocampus, and amygdala (Scott et al., 2015). This atrophy could contribute to symptoms of inattention, hindered working memory, and frontal or executive function deficits.

Due to dysfunction related to pathways involving the reticular activating system, the brain orchestrates high arousal and heightened alert states. The medial prefrontal cortex (mPFC) and anterior cingulate are involved in decision making and emotional regulation (Devinsky, Morrell, & Vogt, 1995). These functions are essential to efficiently attend to the environment, quickly decipher the meaning or implications of the available stimuli, and, based on prior experience, inhibit or engage in an appropriate emotional and cognitive response. This response will also engage the neuroendocrine system. However, when the anterior regions of the brain are dysfunctional, as occurs among those with PTSD, then the stress response is poorly regulated, and the patient will demonstrate overarousal and incongruent emotional responses. Perhaps worse, the salience, affect, executive, and default mode networks may be overactivated (Hagedorn, 2015). On the other hand, people with PTSD evidence hypoactivation of regions involved in attention, working memory, encoding, and executive processing, including dorsal prefrontal, inferior frontal, superior parietal, and orbitofrontal regions. This suggests dysfunction in neural networks composed of PFC, cingulate cortex, and limbic regions, which have the potential to affect emotion processing, cognitive functioning, and their interaction (Scott et al., 2015). At our center, we have observed that patients with PTSD can demonstrate low-amplitude, high-frequency beta (> 20 Hz) at Cz, whereas patients with severe anxiety without PTSD tend to demonstrate high-amplitude, high-frequency beta at Cz with a source in BAs 23 and 24, the ACC, as illustrated in our case example at the end of the chapter (see Figure 9.15 later in the chapter). These activation findings correspond to reports by Mayberg and colleagues (Mayberg, 2003; Mayberg et al., 2000; Mayberg et al., 2005; Kennedy et al., 2011). These hypoactivations are said to be significantly more common in PTSD than in social anxiety and phobia disorders.

The right insula is involved in self-awareness and is part of the default mode network (Sridharan, Levitin, & Menon, 2008). The default mode network (DMN) is activated when one reflects on oneself and how one relates to the world, both physically and psychologically. It is also part of the salience network that determines where to direct attention. It is hyperactive in people with PTSD: Its activity might be hypothesized to be self-protective to block imaginal reexposure to traumatic sensory and visual memories stored in the brain and thus block associated emotions (Rosso et al., 2014). In addition to the insula, the anterior cingulate and amygdala are part of the salience network (SN), and these structures are all involved in maintaining an appropriate balance between introspection and attention to external stimuli. Those with PTSD have been found to have hindered SN connectivity with either hyper- or hypoarousal. These areas can be specifically targeted with LNFB. Bessel van der Kolk and colleagues (2016) say that the traumatized person needs to engage his or her cingulate, insula, and mPFC in the context of focusing on internal experiences that involve integration of cognitive, sensory, and emotional functions. Their conclusion, based on controlled research concerning NFB for chronic PTSD, was that "twenty-four sessions of NF produced significant improvements in PTSD symptomatology in multiply traumatized individuals with PTSD who had not responded to at least six months of trauma-focused psychotherapy, compared to a waitlist control group" (van der Kolk et al., 2016, p. 12).

Bluhm and his colleagues (2009) have found reduced function in the DMN and SN among those with histories of early childhood trauma. The involved DMN structures include the posterior cingulate, precuneus, and mPFC. In addition, as it involves emotional processing and stimulus identification, the fusiform gyrus (BA 37) is part of the DMN and is also implicated in PTSD. The insula (BAs 13 and 43) has also been repeatedly implicated in PTSD imaging studies (Lazar et al., 2005), and, with its integral connection to autonomic nervous system (ANS) balance, it is easy to see how PTSD is jointly an autonomic and CNS dysregulation condition. Other imaging research further supports the relationship between PTSD and the posterior cingulate, precuneus, anterior cingulate, and right amygdala (Lanius et al., 2010). Compared to normal controls, imaging results of two nodes of the DMN, the posterior cingulate cortex (PCC) and mPFC, suggest that those with PTSD have increased connectivity between the posterior cingulate and the frontal gyrus (BA 10) plus the mPFC and left parahippocampal gyrus (BA 35; Daniels et al., 2010). PTSD may show right parietal faster frequency alpha (Rabe, Beauducel, Zollner, Maercker, & Karl, 2006).

Due to the multiple networks affected, a central–parietal (Cz–Pz) single-channel sequential montage can be utilized for training. It has the advantage of being easy to set up, and activity from those sites reflects several networks; namely, the affect, sensory, executive, salience, and default networks. Hagedorn (2015) notes that combining this simple, single EEG channel training with HRV biofeedback has certain advantages, good clinical logic, and research support from a randomized, controlled trial using fMRI as an outcome measure.

To summarize, regions of interest in PTSD include: insula (BAs 13 and 43), posterior cingulate (BAs 23 and 31), precuneus (BA 7), mPFC (BAs 8–12, 24, and 32), ACC (BAs 24, 25, 32, and 33), fusiform gyrus (BAs 36 and 37), and perirhinal cortex (BA 35 [hippocampus]). Although single-channel NFB that affects the cingulate gyrus and therefore, quite probably, the cingulum has been effective, many of the areas involved in PTSD are central and ventral; thus it is harder to influence them directly with single-channel training. Therefore, combining LNFB with HRV training is posited to be an efficient and effective treatment for those with chronic stress and also for people suffering from PTSD or from a combination of PTSD and postconcussion symptoms. HRV training, which activates the parasympathetic nervous system's calming responses, can have an effect on affect, attention, and stress management. HRV has the added advantage of being easy to practice at home and at work using paced breathing apps or just doing slow (about 6 breaths per minute), effortless, diaphragmatic breathing. Using HRV biofeedback in the office setting and paced breathing when outside the training milieu, a person can become able to increase parasympathetic tone and decrease sympathetic drive, with lasting results (Hagedorn, 2015). Furthermore, Hagedorn has mentioned that the self-regulation dynamic of biofeedback may very well itself be an essential and salient healing component for those exposed to moral injuries where one's ability to maintain self-protection and personal regulation was removed. For a further discussion of brain areas involved in the stress response, see Kusnecov et al., Chapter 4, this volume.

The Polyvagal Theory and Its Relevance to Treating Stress

Stephen Porges (2007) notes that the vagus nerve contains specialized subsystems that control competing adaptive responses. Porges's polyvagal theory describes the different roles of the unmyelinated vagus (dorsal vagal complex) and newer myelinated vagus (ventral vagal complex) and the sympathetic nervous system (see Figure 9.7). The unmyelinated vagus responds to threats through immobilization, feigning death, passive avoidance, and shutdown. The unmyelinated vagus is activated in traumatic circumstances in which a person believes that he or she may die and gives up. A different mechanism involves the sympathetic nervous system, which, in concert with the endocrine system, responds to threats with mobilization, fight-or-flight, and active avoidance. The sympathetic nervous system inhibits the unmyelinated vagus and mobilizes the individual for action. A third system depends on the highest evolutionary level, the myelinated vagus system. The myelinated vagus rapidly adjusts cardiac output and promotes social engagement.

Daily stressors can result in a decrease in the myelinated vagal response and a corresponding increase in sympathetic drive. Relaxing muscle tension, emptying the mind of negative ruminations, and practicing diaphragmatic breathing at one's resonance frequency (about six breaths per minute for most adults) can decrease this sympathetic drive and restore the calm state mediated by the myelinated vagal system. See Lehrer, Chapter 10, this volume, for a thorough description of this method. Thus, in a normal individual in a safe environment, the defensive activation of the primitive unmyelinated vagal and sympathetic systems is actively inhibited, allowing the phylogenetically newer, myelinated, medullary vagal system to dominate (Porges's polyvagal theory). This results in a decrease in heart rate and blood pressure and an activation of striatal muscle systems in the face, head, middle ear (stapedius muscles), larynx, and pharynx so the individual can function and appropriately respond to safe social interactions (Porges, 2007). Porges's theory supplies a rationale for doing HRV training.

ANS Component	Activated/Inhibited by	Responses
Unmyelinated vagus (dorsal vagal complex)	Survival threats activate	**Feigning death, immobilization, passive avoidance, shutdown (PTSD)**
Sympathetic-adrenal system	Survival threats activate	Mobilization, fight-or-flight, active avoidance
Myelinated vagus (ventral vagal complex)	Social engagement, facial muscle relaxation activate; daily stressors inhibit	Bonding, calming, inhibition of sympathetic-adrenal activation, respiratory sinus arrhythmia

FIGURE 9.7. ANS components central to the polyvagal theory. Figure used with permission from workshop given by Fred Shaffer based on work by Dick Gevirtz (2011).

Heart Rate Variability Training

NFB and HRV training are the core components of training that reliably produces long-lasting results. Both approaches train oscillatory systems, and they are complementary because they influence many of the same structures in the nervous system due to heart–brain links, as discussed above.

The way that HRV biofeedback training can be combined with NFB is illustrated in Figure 9.9 later in the chapter. Statistical analysis of HRV is done using the Cardio-Pro program software, available from Thought Technology, to give the internationally accepted statistics. Breathing diaphragmatically at one's resonance frequency will usually be observed to result in the highest amplitude of HRV. Training HRV is important for the majority of clients who suffer from stress because it is known to reduce anxiety and modulate the stress response (Mikosch et al., 2010; Reiner, 2008). It can also improve cognitive functioning in stressful conditions (Prinsloo et al., 2011). For a detailed explication of the mechanisms that influence the changes in heart rate, including the role of baroreceptors and vagal connections involving the nucleus of the solitary tract in the medulla of the brainstem (*nucleus tractus solitarius*), the reader is referred to Thompson and Thompson (2015a). The clinically important point is that HRV training involves learning effortless diaphragmatic breathing to pace heart rate changes and produce a calm state. This is an enormously helpful self-regulation tool for stress management, and it therefore nearly always accompanies NFB training at our center.

The EEG in Stress and Choosing Targets for NFB

The EEG of clients who present with anxiety or panic usually show bursts of high-amplitude, high-frequency beta (> 20 Hz). When the source of this excess, often spindling, beta, characterized by high amplitude, narrow frequency range, and synchronous beta (see Figures 9.11, 9.13, 9.14, later in the chapter)—which we have called "busy brain"—is identified using LORETA, the anterior cingulate and, in particular, BAs 24, 33, and 25 are usually identified as the source of this unusual EEG activity. Spindling beta in the inferior right PFC area is also found, especially in people prone to experiencing panic. Sleep disturbance may be associated with right parietal (P4) area beta and hypervigilance. (For a review of early LORETA literature, see Pascual-Marqui, Esslen, Kochi, & Lehmann, 2002.) Whereas normal beta is low amplitude, desynchronous, and includes many frequencies, spindling beta is higher amplitude, synchronous, and in a very narrow frequency band. The particular frequency range of the spindling beta will differ: for example, it might be 24–25 Hz in one person and 31–32 Hz in another. Assessment using LORETA allows the practitioner to precisely identify the BAs that are the source of the activity. In addition, use of a normative database, such as NeuroGuide, will indicate how many standard deviations from the mean the activity is. Using NeuroGuide's LNFB program, one can match the client's symptoms to the edited EEG to identify relevant BAs. Matching involves taking known functions from the literature for BAs and matching them to the BAs that are outside database norms (Thompson & Thompson, 2015b). The NeuroGuide program does this automatically, but the practitioner always checks the result. Data for amplitude, coherence, and phase values are available, and this allows the practitioner to choose specific targets for the LNFB.

Figure 9.8 illustrates some of the central midline BAs that can be involved in a patient who complains of anxiety and inattention. LNFB can address multiple sites and various frequencies at the same time. Many BAs cannot be easily trained using single-channel

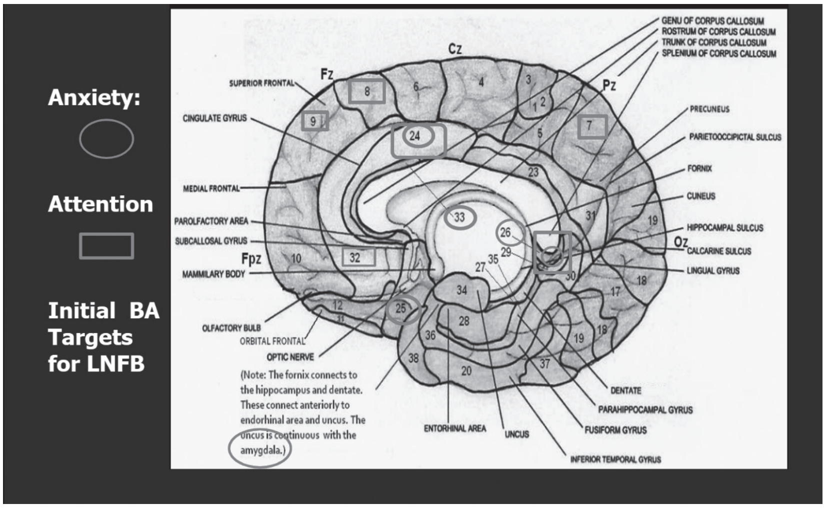


FIGURE 9.8. LNFB targets for two principal symptoms of chronic stress: anxiety and attention. From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

NFB but can be targets of LNFB at whatever frequencies are chosen for each BA to be trained. In Figure 9.8, both BAs and the 10–20 sites on the surface of the scalp are shown. Note that using 10–20 individual locations, such as Cz or FCz (halfway between Fz and Cz), as shown in Figure 9.1, is completely different from specifying numerous Brodmann areas as targets when LNFB is used. With LNFB, a cap with 19 electrodes is placed on the patient's head, and every site is checked to be sure impedance is less than 5 Kohm and preferably within 1 Kohm of each other. Then we choose, using the NeuroGuide program, the specific electrode sites we wish to target in any particular session. The program helps with this choice by listing the sites outside the database norms that best correlate with that patient's major symptoms. The task for the client is to find and maintain a mental state that keeps the chosen parameters within a designated standard deviation (*SD*) range, such as a starting level between 1.8 and 2.1 *SD*. The program can target amplitude, coherence, and phase values that exceed whatever *SD* the practitioner decides to use. The cingulate gyrus and the cingulum (white matter connections within the cingulate gyrus) are often among the targets due to that area's importance in the default, salience, affect, and executive networks.

Single-Channel NFB and the Cingulum

Despite the apparent precision for targeting sites with LNFB, single-channel NFB can still be extremely effective, as demonstrated by clinical outcomes over the last 25 years (Thompson & Thompson, 1998, 2010). FCz placement (Figure 9.1, between Fz & Cz) when using single-channel NFB is hypothesized to influence BAs 24, 32, and 33 and, thus, the cingulum, a fasciculus (white matter connections) within the cingulate gyrus. The excellent results using single-channel NFB at this site are possibly due to the number of neural networks that can be influenced, because the cingulum is a central midline structure that is like a common highway for the affect and executive networks, as well as the default mode network. It runs through the cingulate gyrus and has connections to frontal, central, parietal, occipital, and temporal lobes. At the termination of the cingulum in the temporal pole is the uncinate fasciculus that connects the entorhinal and uncinate temporal cortex, through the amygdala and hippocampal region, to the inferior and medial frontal cortical areas. The cingulum and the uncinate fasciculi would both appear to be central to limbic and thus affect network functions. Among other connections of the anterior cingulate are those to premotor areas, spinal cord, red nucleus, and locus coeruleus.

The cingulum is said to have more connections with the thalamus than any other part of the brain. Through its connections to autonomic brainstem motor nuclei, it exerts control over the sympathetic and parasympathetic portions of the ANS. It connects to the amygdala and, in particular, to the paraventricular nucleus of the hypothalamus. The paraventricular nucleus of the hypothalamus not only has direct connections to the HPA tract, but it is also, as previously noted, the only nucleus to have afferent and efferent connections to both sides of the ANS, sympathetic and parasympathetic. It also has a major influence over endocrine responses through its connections to all parts of the limbic system, including the amygdala, hypothalamus, and periaqueductal gray matter. The anterior cingulate and the cingulum can, therefore, be thought of as the neurological hub of our emotional control system (Thompson & Thompson, 2015b). It is central in discrimination tasks concerning the motivational content of internal and external stimuli. It is engaged in response selection, cognitively demanding information processing, and motor actions (Devinsky, 1995). Training that influences this important structure is nearly always indicated for stress management.

ASSESSMENT

Assessments should include all of the measures that are relevant to assisting your client in meeting his or her objectives. This means that you begin with clinical history taking and discussing goals before doing measurements of both EEG and physiological parameters. Listening to the client's story first is crucial, following the adage that "they must know that you care before they care what you know." The approach used for stress management combines NFB (training the brain) with peripheral biofeedback. Using appropriate psychophysiological measures, you can design and periodically evaluate a training program for stress management. It is very similar to the training program used to help executives and athletes optimize their performance. The goal is to be able to achieve a mental state that is relaxed yet alert, calm, focused, and able to solve problems. Returning to this state of relaxed attentiveness after a period of stress is a sign of resilience. The key to efficiently and effectively handling stress is the ability to self-regulate one's mental and physiological state throughout each day. NFB plus biofeedback helps provide the tools to do that.

With respect to the EEG, we look at the raw EEG and also at quantitative EEG; that is, mathematical calculations concerning the amount of activity across different bandwidths. The bandwidths are described in terms of frequency ranges, with frequencies measured according to cycles (complete waves) per second. The unit of measurement is Hertz (Hz). EEG frequencies are much slower than those used in electromyography (EMG); often a bandpass filter of 2–45 Hz is used, with a higher range monitored to gauge whether there is EMG artifact affecting the signal. Using a range of 2–62 Hz is helpful for the latter purpose. For the beginner in NFB, a table showing EEG bandwidths at Cz and correlation to mental states may be helpful and is available in Thompson and Thompson (2015a).

How do you discern that a person is experiencing stress using the EEG? This topic has not yet received adequate research, and in this chapter we are sharing clinical observations, not established findings concerning this question. Our observation is that anxiety and emotional intensity usually correspond to an increase in amplitude for 19- to 22-Hz activity as compared with the 15- to 18-Hz activity measured with EEG recorded from a single electrode on top of the head (Cz location). This is easily seen when the raw EEG, which is graphed as a line showing amplitude over time, is transformed into a spectral array that plots the amount of electrical activity, either magnitude (average amplitude) in microvolts (μV, millionths of a volt) or power in picowatts (pW), across frequencies (Figure 9.3). Ruminating is associated with higher beta frequencies. The specific frequency range that is elevated can be quite narrow and will usually be above 22 Hz, sometimes in a range as high as 32–35 Hz. The case history at the end of this chapter is a good example of someone with a lot of excessive "high beta" (beta at frequencies >22 Hz). When we observe high-amplitude beta that is synchronous and has a sinusoidal waveform and a narrow frequency range, we refer to it as "spindling beta." Its waveform is quite distinct from the usual beta activity that is low amplitude and desynchronized, because it looks like a series of sine waves; indeed, one could mistake it for alpha activity if one did not notice that the frequency, the number of cycles per second, is more than twice as fast. This higher frequency beta is often seen in conjunction with a decrease in SMR activity. The EEG of someone under stress usually corresponds to a decrease in alpha (both 9–10 Hz and 11–12 Hz) and in 12–15 Hz SMR that is observed only on the sensorimotor strip, so it is measured at C3, Cz, or C4. However, in some rare cases, there may be very high-amplitude, high-frequency activity in the SMR range. We posit that this activity does not represent SMR generated from the thalamus but, rather, cortically generated beta related to anxiety. There may also be an increase in the amplitude of the EEG somewhere between 23 and 35 Hz (compared with activity immediately above and below that bandwidth). Bursts of increased high beta in the low 20s seem to be a state marker; the person reports feeling intense and often quite anxious. The bursts of activity in frequencies in the mid-20s to low 30s may indicate that the person has what we term a "busy brain," which, in some, may correspond to worrying and ruminations (Thompson & Thompson, 2006). These patterns may be associated with clients' reporting that they have trouble falling asleep because they cannot turn off their thinking. There may also be hypervigilance that often is associated with high-amplitude, high-frequency beta also being present in the parietal (P4) region of the brain.

EEG Assessment

Overview

The EEG acts like a "flag" that reflects brain functioning. Just as a flag's activity reflects the wind's velocity and direction, inferences about the brain's activity can be made by reading the EEG. One goal for assessment is to ascertain what is different from expected ("normal") patterns in a client's EEG so that the clinician can decide on an EEG normalization training program. This was discussed in relation to single-channel assessment previously and shown in Figures 9.2 and 9.3. Further clarification can be achieved using a 19-channel EEG. When doubt remains after a 19-lead assessment, comparing characteristics of the EEG in various frequency bands with expected age norms using databases can be useful. However, database norms for EEG doing active tasks, such as reading and math, are very limited. They are available in the SKIL program (Sterman, 1999). Databases are available for eyes-open and eyes-closed EEG from a number of sources, such as those created by Frank Duffy (Duffy, Iyer, & Surwillo, 1989; Duffy, Hughes, Miranda, Bernad, & Cook, 1994); E. Roy John (John, Easton, & Isenhart, 1997); or Robert Thatcher (*www.appliedneuroscience.com*). More recently, a Korean database that has both male and female norms and one from Evoke Neuroscience in New York are being developed.

If there is any concern about a primary medical condition, such as a seizure disorder, head injury, and so on, an appropriate medical workup should be done before undertaking EEG biofeedback. The quantitative EEG (QEEG) looks at data concerning *normal* brain waves. Assessing and recognizing abnormal waveforms or patterns is the task of a specially trained neurologist.

Method

Placement of electrodes follows a standardized arrangement called the International 10–20 System (Jasper, 1958). Newer references may use updated designations for some sites; namely, T3 becomes T7, and T5 becomes P7 on the left. On the right, T4 becomes T8, and T6 becomes P8. New terminology is based on the modified combinatorial nomenclature of the American Clinical Neurophysiology Society (Fisch, 1999). There is also an expanded 10–20 system that has names for sites between these basic ones (Thompson & Thompson, 2015b). As the EEG can show different patterns depending on the conditions under which it is recorded, the gold standard for doing an assessment is to assess four conditions: eyes closed, eyes open, reading, and math. A challenging mental math task constitutes a stress for most people, so this task can be used to look at stress-induced EEG changes.

ELECTRODE PLACEMENT

The initial assessment can use 1, 2, or 19 channels of EEG. (*Full-cap assessment* is a term sometimes used for 19-channel assessments because a cap with the 19 electrodes plus a ground electrode is used for data collection.) The decision will depend on the complexity of the case, the equipment used, and the experience of the clinician. The most common placement for an initial single-channel recording is Cz, the vertex. This site is less influenced by artifact because it is far from the eyes (eye-blink and eye-movement artifacts) and from the jaws (a common source of muscle artifact). Cz is also furthest from the ear reference, so there is less common mode rejection and higher amplitude activity is seen than from sites closer to the ear lobe reference. It provides information about activation (or lack of it) in the central region and across the sensorimotor strip. It also often picks up frontal beta spindling and other high-frequency beta activity that may correspond to the effects of stress on the client and that often can be seen, using LORETA, to have its source in the cingulate gyrus. If your client has ADHD, which is most often characterized by excess theta in frontal and central regions, this can also be readily seen at the Cz location (Figures 9.2 and 9.3). However, this technique misses such things as differences between the hemispheres.

For clinicians who do not have 19-channel equipment, a second channel or a second assessment can usefully be carried out at FCz (halfway between Fz and Cz), and/or at F3 and F4. This will sometimes pick up either high-amplitude “thalpha” (6–10 Hz) or high-amplitude, higher frequency beta (above 19 Hz) that had not been seen at Cz and may be linked to inattention. It can pick up excessively high-amplitude (12–13 Hz, or 20–21 Hz) and high-frequency spindling beta that can all be associated with anxiety. This can be very helpful with clients who present due to difficulties coping with stress. A two-channel assessment, one channel at F3 and a second channel at F4, with either a Cz or a linked-ears common reference, can be helpful in assessing dysphoria or depression and show high alpha (or theta) in the left frontal lobe compared with the right (Davidson, 1998). This may underlie a susceptibility to stress. High-amplitude beta spindling at both sites may have its source in BA 25. Less activation in the left frontal lobe, as compared with the right frontal lobe, has been identified by Richard Davidson as a pattern associated with a focus on negative thoughts and avoidance behavior, because his research identified left frontal activation in association with positive thoughts and approach behavior, whereas right frontal activation related to a focus on negative thoughts and avoidance behavior (Davidson, 1995). The F3–F4 two-channel assessment may also indicate overactivation of the right frontal lobe, a pattern observed in some clients who present with anxiety and panic (Weidmann et al., 1999).

ARTIFACTS

If one does not carefully remove artifacts, the data may be distorted; for example, beta activity may appear high due to muscle tension, giving a false impression of a low theta/beta ratio or of high-amplitude, high-frequency beta, which might be misinterpreted as reflecting stress with anxiety or ruminating. In the range up to about 4 or 5 Hz (thus affecting the theta range), activity may be high due to electrode movement, eye movement, or eye blinks affecting not just delta but also lower theta frequencies. After artifacts have been removed, a graph like the one shown in Figure 9.3 can be generated that shows the amount of electrical activity at each frequency. Frequencies from 2 to 40 are of particular interest, as this is the range in which EEG training is done. No normative database is available for amplitudes at each frequency, in part because amplitudes measured on the scalp are affected by things such as skull thickness and not just the electrical potentials produced by the pyramidal cells in the cortex. Amplitudes will be higher in children due to thinner skulls, and there are also age-related changes in dominant frequency: Infants show the highest amplitude (measured eyes closed in the occipital region) in the delta range, and children have a dominant frequency in the theta range, whereas 10 Hz in the alpha range is the usual dominant frequency in adults (Fisch, 1999).

MEDICATION EFFECTS

Medications may also distort EEG findings. Even supposedly "nonsedating" antihistamines (commonly used by clients for allergy relief) or antibiotics may cause increased amplitude of theta waves. In our case example of Diane, given later in the chapter, her 4–8/16–20 ratio moved from an average amplitude of 2.1 microvolts to 3.4 microvolts when she took a small dose of over-the-counter antihistamine medication. That change produced a high theta/beta ratio, suggesting ADHD, but it was actually a temporary change due to an antihistamine. Another person might have high-frequency beta, suggesting another type of attention deficit (the type that is overly focused on worries), but the increased beta might be related to taking a benzodiazepine for anxiety or an antidepressant for a mood disorder. Benzodiazepines and selective serotonin reuptake inhibitors

(SSRIs) may increase beta activity—particularly beta over 20 Hz. They may also decrease alpha. Tricyclic antidepressants may increase asynchronous slow waves and may even result in some spike and wave activity. Lithium can increase asynchronous slow waves. If you are working with teenagers and young adults, you may find some self-medicating activity is taking place to try to relieve stress. Marijuana will increase alpha, and this may be observed in the EEG even 1–2 days after use. People with alcohol problems can show increased beta above 20 Hz and decreased theta and alpha as a baseline. The alpha increases when they have a drink. Medication effects on the EEG is a complex topic, but the message is that many clients who come wanting to relieve stress may also have tried prescription and nonprescription drugs. Although these drug influences may seem disconcerting, they are usually easily seen, and the practitioner may, to a large extent, decrease their influence on the 19-channel EEG assessment by using different views of the EEG activity (Hammond & Gunkelman, 2001; Thompson & Thompson, 2010). The different views are based on how each electrode site is referenced, with changes in reference, referred to as different montages, giving different information. Nineteen-channel EEG data are collected with linked ears as the reference for each site, and that provides one view of the data. With the click of a button, assessment programs, such as NeuroGuide, allow one to switch to a Laplacian montage. Laplacian montage compares the amplitude at each electrode site to those electrodes that surround it, thus, usually, canceling out medication effects that are common to all sites. You do need to know what the client is taking and recognize that it may have an effect on your EEG assessment, so the rule is to always ask what medications a client is taking (Thompson & Thompson, 2015a).

CHECK THE RAW EEG

If you do not look carefully at the entire raw EEG, you may miss very important EEG changes. For example, a quantitative analysis of EEG that contained a brief absence seizure, seen in the EEG as high-amplitude spike and after-following slow-wave activity that occurs at 3 cycles per second, would give the false impression of very high delta activity (the slow waves) and some increase in high-frequency beta activity due to the spikes (Thompson & Thompson, 2015a). There is simply no substitute for a careful analysis of every second of the raw, dynamic EEG. Being able to see a mathematically derived spectrum, plotting frequencies by power, is helpful in discerning a client's patterns, but only after you have carefully looked at, and artifacted, the raw EEG.

OVERVIEW OF INTERPRETATION OF FINDINGS USING DIFFERENT TYPES OF ASSESSMENT

Single-Channel Findings. These have been described earlier in the chapter.

Two-Channel Findings. Two-channel results may clarify differences between two different areas of the brain. Special caution must be exerted to ensure that impedance at both electrode sites are within 1 Kohm of each other and below 5 Kohms at each site before findings at different sites are compared. Again, because many areas of the brain are ignored, findings are still limited. It is easier and better to compare two sites using two channels with a common reference using a linked-ears reference electrode. Two-channel assessment at F3 and F4 can be helpful in depression, in which F3 may have less activation and show more theta or alpha activity. Alternatively, do a 19-channel assessment in which the data collection is always linked-ears, and the bigger picture can be seen.

Full-Cap (19-Channel) Findings. These findings are obviously more complex and complete. Different sites (Brodmann areas) may be compared, and thus coherence values can be calculated. As previously explained, coherence relates to shared activity at two sites and suggests that there is a common source for the activation. Databases can give *z*-scores that calculate whether the amount of shared activity is what is expected or whether it is more (hypercoherence) or less (hypocoherence). A head injury, for example, usually disrupts connections in the cortex and results in hypocoherence. As previously noted, it takes many years of experience to accurately interpret the findings, and one must purchase the database(s), which can be expensive. Alternatively, one can collect the data and outsource the interpretation. Findings from 19-channel assessments can be used to guide single-channel training, and we always do a single-channel assessment to evaluate the spectrum from 2 to 62 Hz at Cz because that central location gives us a lot of information. When we do the 19-channel assessments, we use two different instruments and databases, though this is not the usual practice, and using one system is the usual standard of practice. Using a second system gives added reliability to the findings and, more importantly, the second assessment gives other measures, such as evoked responses (usually called event-related potentials, or ERPs) that give information about brain speed. We use Mitsar equipment (*https://mitsar-eeg.com*) for one set of data collection and analyze the data using NeuroGuide. The second assessment uses the eVox system with the analysis provided by Evoke Neuroscience (*https://evokeneuroscience.com*). The latter report provides five kinds of information: (1) self-report from questionnaires, (2) data from a continuous performance test (CPT; response time, variability, omission and commission errors), (3) HRV data, (4) ERP data, and (5) EEG data, including brain maps for eyes-open and eyes-closed and the top three deviations (eo and ec) that are > 2 *SD* from database norms, showing LORETA source localization and explaining the functions of that Brodmann area. Currently, the eVox system that provides the comprehensive assessment with 20 minutes of data collection is sold only to physicians. There are many good amplifiers and databases on the market from which a practitioner can choose.

LORETA Assessment. This method of EEG analysis is a mathematical program that uses the surface EEG to infer which structures deeper in the cortex are the source of the electrical activity being measured. LORETA's ability to pinpoint the origins of EEG activity has been validated against MRI findings. LORETA was developed by Roberto Pascual-Marqui of the Key Institute in Zurich. It can be downloaded free of charge over the Internet. Programs such as NeuroGuide and the one developed by Evoke Neuroscience combine LORETA's mathematics with database norms to calculate *z*-scores (*SD* from the mean) for each frequency, showing the magnitude of the deviation and the source of the activity; for example, someone with anxiety might have a deviation of 3.1 *SD* for the amplitude of 25-Hz activity with source localization identified as BA 24, the ACC. Both LORETA and other imaging techniques, such as fMRI, indicate that hyperactivation of the cingulate cortex is the most common finding in people with high anxiety; however, hypoactivation can be the more common finding in PTSD, as discussed earlier in this chapter. This underactivation is significantly more common in people with PTSD than in those with social anxiety or phobias. In PTSD, the EEG, whether single channel or 19 channel with LORETA analysis, usually shows one of three distinct patterns: high arousal reflected in fast-peak alpha frequency equal to or greater than 11 Hz (thalamic origin), high-frequency (fast) spindling beta (cortical origin), or low-amplitude (low voltage) EEG with increased fast (beta) activity.

Quality Data Collection. The following tips apply to data acquisition, whether 1 or 19 channels. They also apply whether one is doing an assessment or running a training session. Training outcomes are going to be influenced by the quality of the feedback, which depends, first and foremost, on the quality of the EEG information. So it is good to make it a habit to be careful about site preparation: Abrade gently with an EEG preparation material, such as NuPrep, to remove things such as surface skin cells or hair spray that could act as insulators, and then use a conductive gel or paste, such as 10–20 Conductive Gel, for each of the electrodes. As previously noted, aim to meet research standards of impedance readings that are all below 5 Kohms and, if possible, within 1 Kohm of each other. Checking impedance is very important for good quality work. Applying electrodes without skin preparation is insufficient with most equipment used for neurofeedback. (There are special saline electrodes for research that use dense arrays of > 100 electrodes that do not require the same kind of preparation, but that is quite different equipment than is generally used for NFB.) Dry electrodes have been developed, but many do not allow for adequate impedance checks.

Autonomic Nervous System and Muscle Tension Assessment

Goal of the Stress Assessment

In addition to the EEG changes, stress also induces changes in other physiological variables. It is very simple to show how even a small stress can result in a decrease in peripheral skin temperature, an increase in SC, muscle tension, and HR, plus a respiratory pattern that is shallow, rapid, and irregular. Heart rate variations are not in synchrony with respiration when one feels stressed. With electronic measurements, the client can see that, with appropriate diaphragmatic breathing and a relaxed mental state, he or she can rapidly shift these variables to produce a healthier pattern.

The goal of the EEG and stress assessment (ANS and electromyogram assessment) is to discover how a particular client responds to and recovers from mental stress. These findings may then be used to set up both NFB and biofeedback interventions to help that client self-regulate, that is, control his or her own mental and physiological responses even under stressful circumstances. In addition, practicing this control may produce an automatic, unconscious, beneficial change in future responses to stress. The objective is to help produce an optimal state of mental and physiological functioning at will. In this state, the client is both relaxed and alert. This will broaden associative capabilities and perspective, decrease fatigue, allow calm reflection on alternative approaches to tasks, and, when combined with high levels of alertness, improve reaction time and increase response accuracy. The individual will be flexible in terms of mental state and resilient in terms of his or her physiology. To demonstrate for the client his or her own physiological response to stress, we developed a quick stress test that collects 12 minutes of data. It is one of the Thompson Scripts designed for use with the Biograph Infiniti from Thought Technology and is available from the Biofeedback Federation of Europe (*bfe.org*).

Description of a Quick Stress Test

This is not a research assessment protocol, and you can modify it to suit your needs. It is a practical, clinical approach to clarifying what general biofeedback modalities, if any, are going to be important, in addition to NFB, for training a particular client. We want to observe changes in respiration, including rate, depth, and regularity. We like to be able to see the same types of changes in HR: regularity, rate, and extent of variability in HR. To do this requires a computer feedback screen that shows respiration and the HRV that occur with inspiration and expiration as line graphs. It is desirable, with the client, to be able to correlate these changes visually with changes in peripheral skin temperature, electrodermal response (EDR, a measure of arousal dependent upon sweat), and muscle tension (EMG).

In the stress assessment, we want the clients to begin in as relaxed a manner as possible to establish a baseline. We explain that we are going to ask them to carry out two tasks that are meant to be stressful, that is, emotionally uncomfortable. After each task, they will sit for 2 minutes and try to relax and think of something pleasurable (recovery period). The first stressor is the Stroop color test. Color words are printed in various colors—the word *red* printed in green, for example. The client says the color a word is printed in, which is stressful because the reflex response is to read the word and the task gets harder as the words are shown for shorter lengths of time. Next, the client rests (recovery) for 2 minutes. This is followed by a second stressor, mental math, such as serial 7s (counting backward by 7s from 900). The client then rests again for 2 minutes. Then the client is asked to open his or her eyes (if they were closed during recovery) and is guided to breathe diaphragmatically at about six breaths per minute (a little slower for large men and a little faster for small women), according to a protocol described by Lehrer (see Chapter 10, this volume). Also included are instructions to relax the muscles and feel warmth in the hands. The aim of training is for the client to achieve a degree of synchrony between HRV and breathing, known as respiratory sinus arrhythmia (RSA). This provides an introduction to HRV training. This HRV training is incorporated into regular EEG training as shown in Figure 9.9. The summary graph of the whole time period (Figure 9.10) is reviewed with the client to show him or her how his or her body's physiology responded to each phase of the test. One expects a stress response that is normal. The important variable to note is how quickly and completely the client recovers from the stressors. (See Lehrer, Chapter 10, this volume, on HRV biofeedback for more detail.)

The trainer must be careful to help the client distinguish between overbreathing and relaxed "effortless" breathing. Overbreathing (hyperventilation) is a potentially serious side effect that may be avoided by emphasizing that breathing should be effortless and comfortable and that the next breath should only be taken when the "need" is felt by the client. Just let the air in and the diaphragm expands. There is no need for the shoulders to rise with effortless diaphragmatic breathing.

Figure 9.10 illustrates a classic stress response and then a relaxation response. This client's response to stress was normal: heart rate increased and respiration became fast, irregular, and shallow. SC rose, and peripheral skin temperature fell. This client was able to demonstrate quick control of these variables by being reassured and asked to breathe along with the trainer at six breaths per minute in the last 3 minutes of the test. She was able to see the synchrony between her HRV and her breathing on the feedback screen. Two objectives were reached: First, the client observed that even minimal stress can alter a number of physiological variables, and, second, she recognized her capability for normalizing much of this physiology quite rapidly, particularly with effortless diaphragmatic breathing.

It was also clear from the EMG data from her shoulder sensor (trapezius muscle) that she was moving her shoulders as she inhaled. Therefore, her training would include teaching her to just use diaphragmatic breathing and relax her shoulders as she inhaled. The authors combine HRV training with NFB for all clients, as is shown in Figure 9.9.

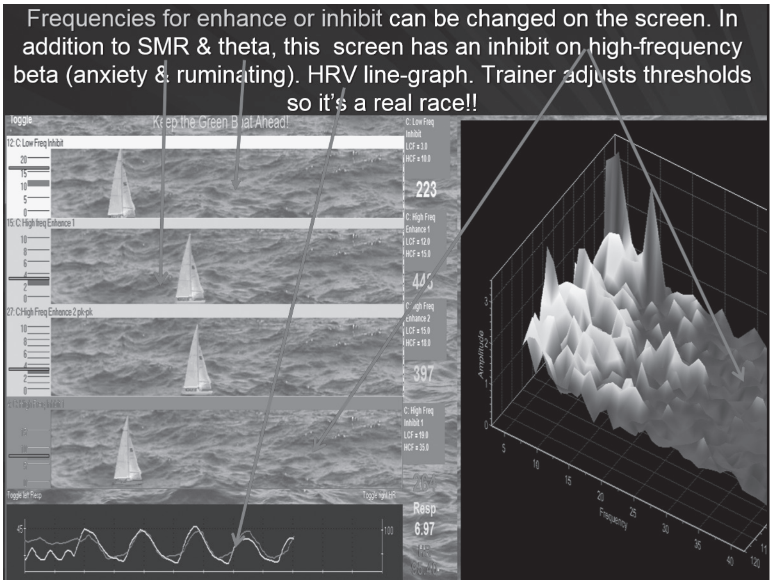


FIGURE 9.9. Example of screen for NFB + HRV training. The figure shows sailboat race for the NFB variables. Client wants the two middle boats, representing SMR and 15–18 Hz beta, to win the race. The bar graphs on the left show amplitude of EEG activity for different frequency ranges. The top boat (theta) and bottom boat (high-frequency beta) score points for remaining below threshold for 2 seconds, whereas the middle two boats score points and move forward for remaining above threshold for 2 seconds. The line graph at the bottom shows heart rate and breathing, which should change synchronously. The spectrum on the right is on a separate monitor for the trainer to track amplitudes of activity from 2–40 Hz over time. From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

CONTRAINDICATIONS TO INTERVENTION

NFB Side Effects

In general, it appears that NFB is a safe, noninvasive intervention that rarely produces negative side effects. Over the past 25 years, we can relate one incident of a side effect in an adult that might have been related to NFB training. In this case, a senior executive who had started training for optimal performance felt “spaced out” and was unable to chair a meeting with his usual intensity. He had abruptly left his training session when he recalled the meeting time. On reviewing the incident later, we felt he might have trained too hard to lower his very high 21 Hz activity and inadvertently raised alpha, especially 9 Hz activity, and put himself into a dissociated state.

People doing “neurotherapy” that might involve stimulation and adding other potentially helpful techniques, such as audiovisual stimulation or brain–blood flow biofeedback (hemoencephalography [HEG]), might see more side effects, such as headaches. The majority of ADD Center clients are primarily learning to reduce slow-wave activity, especially theta, and reduce “busy brain” (beta above 20 Hz), while increasing calm attentiveness that is associated with increased SMR and/or beta somewhere in the 13–19 Hz range. The frequency range is individualized. Those training parameters, when customized and based on the client’s initial assessment data, do not produce negative side effects.

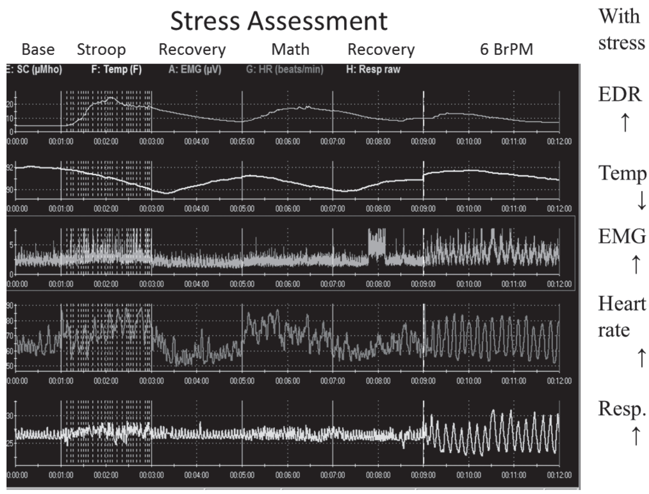


FIGURE 9.10. Stress Assessment Profile. Note that with the two stressors, the Stroop color test and the arithmetic task, skin conduction (EDR) increased, peripheral skin temperature decreased, muscle tension (EMG) increased, heart rate increased, respiration became irregular, fast, and shallow. Observe in this example that in the final segment, when breathing at six breaths per minute, this client used her shoulder (and presumably thoracic) muscles (EMG) when breathing. This was noted, and she soon learned to relax her muscles and breathe diaphragmatically. This screen is from Thompson and Thompson, "Setting–up-for-clinical-success suite," available through the Biofeedback Federation of Europe (*BFE.org*). It is used with the Thought Technology Biograph Infiniti eight-channel instrumentation. With the Infiniti equipment, different configurations of the channels can be used, but in this text, we are using two channels of EMG (2,500 samples/second) and two channels of EEG, plus channels for electrodermal, temperature, heart rate, and respiration, each recording at 250 samples per second. From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

It is true that NFB training is frequency-specific and site-specific, so, theoretically, if one were to train the wrong frequencies at a particular site, then one could get negative results or side effects. In Sterman's early experiments, when cats were trained to decrease SMR activity rather than increase it, they became "twitchy," flicking their tails and moving their ears. Two early research studies employing an A–B–A design, one with hyperkinetic children and one with adults who had epilepsy, showed improvement during the treatment phase, worsening when the contingencies were reversed, and improvement when the correct treatment was again given (Lubar & Bahler, 1976; Lubar, 1997). Training the wrong frequencies should not, of course, happen if the client has been properly assessed and the training appropriately individualized.

A kind of side effect, which could be either positive or negative depending on circumstances, is that family relationships and interpersonal relationships can change as a person's behavior changes in response to NFB.

With respect to contraindications, it has been the experience of many clinicians that children in dysfunctional families are not good candidates for NFB training. NFB is a kind of learning, and a person must have emotional energy for learning. This may not be available if a child is preoccupied with a difficult situation at home. In those cases, money might be better spent on family therapy, and NFB can then be done after the family dysfunction has been addressed.

Biofeedback Side Effects

Biofeedback has more potential side effects than NFB, especially when it is used in conjunction with relaxation training. As mentioned above, there is, for example, the danger of overbreathing (hyperventilation) with resultant hypocapnea (decreased partial pressure of carbon dioxide in the bloodstream). When some clients are asked to breathe diaphragmatically, they may, in their eagerness to follow the trainer's instructions, breathe too deeply. This overbreathing may also occur with anxiety. The result is that hemoglobin does not release oxygen (Bohr effect), and there is vascular constriction and acidosis with resultant symptoms. These can include lightheadedness, anxiety, emotional lability, and confusion. Medical conditions such as migraine headaches or seizure can be triggered. Detailed information is contained in other chapters of this book (see Lehrer, Chapters 2 and 10, Meuret and Ritz, Chapter 11, and Van Dixhoorn, Chapter 12).

Although overbreathing is a very rare side effect, it is important to watch your client carefully and ensure that he or she is breathing comfortably. Some practitioners use a capnometer to measure expired carbon dioxide. However, most practitioners find that they do not have a problem if they warn their clients about the early signs of overbreathing and observe the client to ensure he or she does not overbreathe/hyperventilate.

Relaxation training can result in a client experiencing discomfort as he or she relaxes if the person is not used to that experience. A less often discussed side effect of biofeedback training for relaxation is a loss of alertness. In our work with clients who complain of the effects of stress, we emphasize how the client can optimize his or her performance despite the frequent stressors found in everyday life. Traditional relaxation training is an excellent way to help a client fall asleep. This is not the goal, however, in the athletic, academic, or job situation. The client needs to be able to relax physically while still maintaining a highly alert mental state.

METHOD OF INTERVENTION

Principles upon Which Intervention Is Based

Individual differences dictate that we must be flexible in terms of how we deal with similar appearing clinical entities. How a specific individual responds to various biofeedback modalities will help determine the best approach for that client. Assessment measurements are tools that help us decide which feedback modalities to use and what EEG parameters to train. Therefore, we do not advocate using protocols that utilize one set of parameters for all clients. An example of one potential danger of a standard protocol for raising Cz SMR (13–15 Hz) would be a client who had very high-amplitude spindling beta at 14 Hz. We have observed this with a child who had ADHD symptoms and with adults who had depression. In either of these cases, raising 13–15 Hz would make them worse, not better! Instead, we use a model for determining what to train and where to train based on three things: assessment findings, knowledge of functional neuroanatomy, and the client's goals. The intervention is later adjusted according to the client's response to training. The premise is that our knowledge base is continually updated and that each client is unique in her or his responses, both to our measurements and to our feedback techniques. The model also allows for change in the way we practice over the years, as we learn more about neurophysiology and as our biofeedback instruments improve.

This same caution concerning protocols applies to doing LORETA NFB in which 19 channels of EEG are utilized. Although programs helpfully allow the professional to choose to address a network or even a disorder, we suggest that the practitioner look very carefully at what that program is identifying as the BAs that show the client's deviations from database norms. Our procedure is to see how the program "matches" symptoms to our edited EEG. Then we compare this with our assessment findings, including what the LORETA analysis identified as the source in the cortex for deviations from the database found at surface locations; that is, at the 19 sites from which EEG is measured. We can adjust the training parameters that are automatically selected by the program as needed. For example, in the NeuroGuide program, if we just enter "anxiety" or choose the affect network program, the matching can leave out BA 25 unless we enter "depression" as a symptom. However, BA 25 can be of central importance in some patients with anxiety or even rage rather than depression. The practitioner needs to know something about Brodmann areas and their functions in order to check that the matching done by the program makes sense for each client (Thompson & Thompson, 2015b).

The client's goal is more than just to reduce stress. Reduction of stress implies a change in stressors, but this is often outside of the client's control. If it was within the individual's power to alter the external stressors but he or she could not do so for psychological reasons, then one of the appropriate initial interventions should be psychotherapy. Psychotherapy can be combined with NFB, biofeedback, and cognitive strategies. In this chapter, our assumption is that, from his or her own point of view, the client is under realistic stress and has not found a way to productively deal with the effects of this stressor. A client like Diane, described in the case study later in the chapter, needs to find a means of dealing with stress in a manner that allows her to move ahead even though she cannot eliminate the stressors from her life.

Stress can change a client's entire physiology, including alteration of brain-wave patterns. The result can be akin to a kind of paralysis of efficient mental functioning. The mind may get stuck and ruminate on a repetitive thought or bounce from thought to thought and never stay with one idea long enough to produce constructive problem solving. In some people, there may be a combination of both patterns. Another change with stress, especially when the person is prone to ruminating, is insomnia: trouble falling asleep, trouble staying asleep, and not feeling refreshed in the morning. Fortunately, NFB training that increases SMR is an effective treatment for insomnia, because sleep spindle density is increased at night when SMR production is increased during daytime training. Increased sleep spindle density is associated with smoother transitions in sleep and fewer awakenings (Sterman, 2000b; Sterman & Thompson, 2014).

Whatever the specifics of their original reasons for coming, most clients want to optimize conscious, task-oriented performance. The goal to optimize performance can be expanded as follows: to help a client achieve self-regulation of his or her mental state and his or her mental flexibility and to help him or her to produce a self-defined optimal mental state. This is usually a state of relaxed, alert, aware, calm, focused, problem-solving concentration. Different aspects will be emphasized for particular clients and at various times, so every client's training is individualized. The individual training plans are variations on a theme, because each of these states, such as being relaxed, alert, and so forth, can be regarded as an enabling objective. To achieve each enabling objective, one or more biofeedback and NFB procedures can be used that are designed to assist the client in learning self-regulation. The following is a brief overview of these enabling objectives and some of the NFB and biofeedback methods that can be used to improve them. Only further research will be able to discern whether the emphasis here on flexibility of attention states and the linkage to increasing high-frequency alpha with relaxed, broad open awareness can obtain similar results to Les Fehmi's attention and neurotherapy synchrony training (McKnight & Fehmi, 2001). The latter techniques emphasize the importance of the client being able to voluntarily increase and decrease phase synchrony in the alpha range and improve attentional flexibility. Similarly, conscious changing of mental states is a learning process that is emphasized in all our work. Self-regulation is the goal.

Note in the following discussion that NFB is absolutely entwined and enmeshed with regular biofeedback when working with adults. For this reason, EEG is not described in isolation in most of the following, though it is the core modality being trained.

Combining EEG and General Biofeedback Modalities

As previously described, mental states are correlated to particular EEG bandwidths. Trainers should help their clients discover this for themselves. Our case example later in this chapter shows how Diane was able to confirm correlations between EEG patterns and mental states for herself using EEG biofeedback screens, purposefully altering her mental state, and noting the changes in the EEG. The following seven-step overview of objectives follows the questionnaire format (Tools for Optimal Performance States [TOPS]) that is described in Thompson and Thompson (2015a). These steps utilize single-channel training with the electrode site usually at FCz (halfway between Fz and Cz). Activity with its source in Brodmann area 24 of the ACC is well represented at this site. The principles apply just as well to 19-channel LORETA *z*-score neurofeedback. The goal is for the client to achieve a *state of physiological readiness,* followed by a *state of mental readiness,* and, finally, a *state of active mental work*. This approach is not about relaxation but, rather, about true management of stress for effective everyday functioning.

Objectives for Training and Their Corresponding Measures

To Achieve a Relaxed Mental and Physical State

To help the client achieve a relaxed state, we most often have him or her increase EEG activity between 11 and 12 Hz (high alpha) and/or 13–15 Hz (SMR), and combine that with diaphragmatic breathing at a rate of about six breaths per minute, relaxing muscles, and raising peripheral skin temperature. The 11- to 12-Hz EEG bandwidth may be associated, in most adults, with an open external awareness state, when measured at Cz or FCz. In this mental state, the individual can experience a peaceful awareness of the whole environment. The competent archer or competitive shooter may go into this mental state immediately prior to releasing the arrow or shooting. The martial artist is in this readiness state when totally alert and ready to spar.

The 13–15 Hz activity measured at Cz or FCz is usually associated with SMR and a mentally calm readiness state without muscle tension or movement.

When working with an already anxious client, an exception to this general rule of raising high alpha (11–12Hz) is in those infrequent clients for whom alpha 8–12 Hz (which includes the high alpha) is already very high amplitude in the eyes-open state when that person is feeling anxious. In these instances, as previously noted, increasing alpha may not have the expected relaxing effect. The correct procedure in these cases is to downtrain the overly high-amplitude alpha. This is a good example of how results from a careful assessment dictate intervention.

In addition, the expected good outcomes from uptraining SMR (13–15 Hz) may, like the high-alpha example above, be confounded in those very rare circumstances in which you have a client whose impulsive–hyperactive behavior correlates with sudden bursts of high-amplitude spindling beta at 14 or 15 Hz, as previously mentioned. Also, in some cases of major depression, spindling 14 Hz beta at FCz is observed.

Anxiety and/or emotional intensity may also be reflected in an increase in 19–22 Hz activity relative to 15–18 Hz beta. A relaxed frame of mind implies that the individual is not negatively ruminating and worrying about aspects of his or her life. Ruminating may be found in conjunction with an increase in the amplitude of the EEG somewhere between 23 and 35 Hz, which contrasts with the usual pattern of steadily decreasing amplitudes as frequencies go higher in the eyes-open resting state. Clients are usually able to correlate their ruminating with these bursts of spindling beta, regardless of the frequency. There may be no evidence of excess activity at C3 or C4 when it is elevated at Cz, thus allowing for training of true SMR (13–15 Hz) at C3 and C4. (Note that SMR is produced in the thalamus and projects to the sensorimotor strip—C3, Cz, C4 sites—but Cz may also reflect beta activity from the anterior cingulate; thus C3 and C4 are purer representations of SMR.)

Adjust frequency ranges for individual clients. Depending on the EEG findings, one may, for example, train 14–16 Hz for SMR, rather than 13–15 Hz. As previously mentioned, standard protocols should be strongly discouraged. Rather, the competent trainer should base training strategies on a careful assessment of the symptoms and goals of the client in the light of the EEG and psychophysiological stress test findings and her or his knowledge of functional neuroanatomy and neurophysiology.

To Achieve an Alert Mental State

To accomplish this objective, we encourage maintaining appropriate levels of SC: not as extreme as is seen with anxiety (too high), nor as low as is seen with a drop in mental alertness (too low), nor flat as may be seen with chronic stress. SC and EDR are well covered by Gevirtz, Chapter 6, this volume). The EEG signature of alertness is an appropriate level of SMR, because one must be still and alert to produce bursts of that rhythmic activity; remember that the cats in Sterman's research were alert and waiting for a signal when he first noticed the activity that looked like sleep spindles but that was being produced by a very awake cat.

To Be Aware of the Environment

As noted earlier, in athletics, awareness corresponds to the ability to respond rapidly to changing conditions. For example, a goalie in hockey must be aware of every player's position around the net. In this mental state, the EEG can show a clear increase in 11–12 Hz. The athlete moves rapidly in and out of this state depending on the task. For example, an archer, shooter, or golfer may be in *narrow focus* (beta, 16–18 Hz activity) when judging the wind and the distance. He or she then moves to a state of calm, *open awareness* (11–12 Hz) just prior to releasing the arrow, pulling the trigger, or putting.

However, nothing is ever as simple as it first appears. It is, quite possibly, an inverted U-shaped Yerkes–Dodson–type curve, in which one can raise this alpha activity too much and decrease athletic performance (Kerick, Douglass, & Hatfield, 2004; Kerick & Allender, 2006; Landers et al., 1991).

Flexibility is important in terms of moving appropriately between different states, and small changes between different ranges within alpha appear to be important in performance. The alpha rhythm appears to be associated with a number of functional states. Some authors have said that low alpha (approximately 8–10 Hz) is associated with general or global attention. We find this is more often global in the sense of internal rather than external attention. People with good (vs. poor) memories desynchronize (show desynchronized beta activity) and 8–12 Hz alpha drops in amplitude during the encoding and retrieval of memory, while a brief rise in 6 Hz synchronous theta is observed (Klimesch, 1999). The 6 Hz activity is sometimes referred to as *hippocampal theta* and is related to memory functions. A high alpha band (approximately 11–12 or even 13 Hz) may be associated with task-specific attention (Klimesch, Pfurtscheller, & Schimke, 1992). In this case, the words *task specific* may refer to what we term *broad total awareness,* which is a state of heightened focus and readiness, without tension, that is seen in the high-level athlete and may turn into a singular focus on the target (being one with the target).

To Be Appropriately Reflective

This mental state may correspond to a temporary increase in the amplitude of high alpha, around 11–12 or 13 Hz and SMR 13–15 Hz, followed by desynchronization (less synchronous alpha) and more beta in the 15–18 Hz range, the so-called thinking waves. A task such as scanning text, organizing the important facts and registering them in one's memory, requires that a student move from broad to narrow external focus on the material, then back to internal reflection. Moving flexibly and continually between states is characteristic of effective learning.

To Remain Calm Even under Stress

This mental state is also associated with being both *aware* and *reflective*. As noted previously, it may be attained by increasing 11–12 Hz high alpha and 13–15 Hz SMR and by decreasing 19–22 Hz and 23–35 Hz if they are found to be high. Frequencies between 11 and 15 Hz correspond to the production of high alpha both for open external awareness and for reflection, plus SMR, which relates to a sense of *calm*. As discussed earlier, increases in the 19–35 Hz range are often found to correspond to moments when a person is anxious and worrying or ruminating (usually negatively). This kind of busy brain with ruminating mental state is the opposite of being calm. It is not usually conducive to efficient action or to the breadth of associations that would be necessary for creative thinking. In a calm mental state, a person may also be physically relaxed. This state may, therefore, be associated with diaphragmatic breathing, good RSA, warm hands, and relaxed musculature. One can also be calm mentally while carrying out a physically strenuous task. Then breathing and muscle tension would vary with the task.

To Be Capable of Sustaining a Narrow Focus

Being capable of sustaining a *narrow focus* for the required length of time for a specific task is a key to efficient academics and to optimal athletic performance. The student who is continually distracted by either internal or external stimuli will not achieve at the level of her or his intellectual potential. The athlete who loses focus in the middle of a golf swing will not win that round. Most of us produce bursts of waves either in the theta or the alpha range (somewhere between 3–10 Hz) when we are *internally* distracted. However, some adults produce high-amplitude, high-frequency beta activity when they are distracted by internal ruminations, something we refer to as "busy brain." The latter high-amplitude, high-beta state appears to be particularly evident in persons who feel under stress.

To Be Able To Turn On and Remain in a State of Problem-Solving Concentration

Clients need to be able to turn on and remain in a state of *problem-solving concentration* until a task is complete or a problem is solved. This sustaining of focused concentration is a key to success both in school and in business. This state is usually associated with 16–18 Hz activity. It is also said to be associated with focused attention (Steriade & McCarley, 2005) and activity in the 39–41 Hz range known as Sheer rhythm, or a "binding" rhythm (Sheer, 1977).

These objectives and the TOPS questionnaire still apply when you are doing 19-channel LNFB in addition to, or instead of, single-channel NFB. However, remember that with LNFB you are identifying and addressing BAs and connectivity between BAs that is both above (+ve *SD*s) and below (–ve *SD*s) the 0 *SD* database average. In this manner, you can enhance low amplitudes, such as may be occurring in central midline theta (5.5–6.5 Hz), low or high alpha, and in beta 13–15 and/or 15–18 Hz all at the same time and in most of the relevant locations.

Notes Concerning Frequency Ranges

1. To sustain external focus, one usually wants to control or reduce the production of theta (4–8 Hz). However, cognitive tasks that require encoding, memory retrieval (episodic memories), and semantic processing produce brief bursts of central midline theta, usually around 6 Hz. Theta may, in general, be associated with tuning out from the external environment, but it is also apparently necessary for encoding and for *memory recall*. This activity at 6 Hz is referred to as *hippocampal theta* (Klimesch, 1999) because it is actually due to bursts of high beta in the hippocampal region occurring about six times per second. By asking someone to recall information, you can observe the frequency of theta and the waveform produced for that individual. It will be different from the theta produced when that person is in a rather dreamy, "tuned-out" state. Often, a lower frequency theta of 3, 4, and 5 Hz is evident in the tuned-out state. This theta that appears to correspond to a tuned-out state is more likely produced by a thalamo-cortical circuit.

2. Alpha-theta training in very accomplished music students (people who probably quite readily produced a lot of beta) was found to enhance aspects of their musical performance related to the emotional interpretation of the music (Gruzelier & Egner, 2003). In addition, semantic processing with eyes open has been observed to be associated with a decrease in synchronous high alpha (Klimesch, 1999). The best performers have higher amplitudes of high-frequency alpha at rest and greater desynchronization of both low- and high-frequency alpha on task; that is, alpha attenuates and beta is produced when the person focuses on a task. Clearly, one does not just shift into beta (15–18 Hz) and stay there to solve problems. There is a constant shifting among frequencies as one actively receives and processes information. The analogy of coordinated brain activity being like

a symphony orchestra is apt (Robbins, 2000). The thalamus is the conductor, setting all the rhythms.

3. When working, studying, or being involved in athletic events, the efficient individual is constantly shifting between the beta of narrow problem-solving focus, high-amplitude alpha for inner reflection and autopilot states, and brief bursts of theta for encoding and memory retrieval. Sterman (personal communication, 1993) observed a brief burst of alpha activity after the successful completion of a task that required sustained concentration: Top-gun pilots produced an alpha burst when the wheels touched down during landing. These bursts are sometimes referred to as *event-related synchronization* or *post-reinforcement synchronization*. This seems to be a way in which the brain rewards itself with a brief rest after accomplishing a task.

4. Self-regulation combined with mental flexibility is the final goal.

Generalization

Most clients can learn reasonable control of general biofeedback modalities, such as HRV, in 10–20 sessions, although the NFB results, to ensure lasting changes, typically take about 40 sessions. The two kinds of training are done concurrently. The real challenge is helping the clients generalize what they have learned in your office to their everyday lives. This requires that, from day 1, the trainer assist the client to find ways to integrate what she or he is learning into the routine of daily living. The techniques for doing this include attaching a new habit, such as relaxing with diaphragmatic breathing, to old habits or routines. The latter are simply regular daily activities, such as getting out of bed, brushing one's teeth, eating meals, driving, or answering the phone. The new learning that combines both NFB and biofeedback must become an integral part of daily living. Most clients are able to pair relaxed diaphragmatic breathing with control of the other biofeedback and NFB variables. Thus, through classical conditioning, clients who begin to breathe at six breaths per minute in different situations automatically enter a relaxed, open external awareness state with faster reflexes, just as they practiced in the clinic. When driving, for example, they can use a red light that lasts about 30 seconds to practice three effortless diaphragmatic breaths; when they breathe appropriately, they relax their muscles and focus better, relatively free of internal distracters and ready for contingencies that they may meet on the road.

Goal Setting with the Client

Clients deserve to have a full understanding of the what, why, and how of the steps they will be taking to make positive changes in their everyday living. After all, the therapist is not changing them or even telling them how to make the variables on feedback screens change. The clients are working to change themselves with feedback concerning the electronic measurements with the therapist as a coach. The more knowledge they have about the process, the more they will feel in control. Because people subjectively feel less stressed when they have control in a situation, this approach is a logical part of setting up for success in stress management. Each client should understand the correspondence of EEG bandwidths and mental states, in addition to knowing about the fundamental physiology of the ANS—sympathetic and parasympathetic, accelerator and brakes. This allows for the setting of goals and is at the heart of the therapeutic alliance a practitioner establishes with a client. The NFB practitioner must help the client formulate goals that are measurable and realistic with respect to what can be achieved using NFB + biofeedback + metacognitive strategies. Coaching in metacognition means going beyond regular cognition. It refers to awareness of how learning and remembering things occurs. It is sometimes defined as "thinking about thinking" or "what you know about what you know" (Thompson & Thompson, 2015a). With respect to goals, they must be specific rather than vague. Instead of saying "I want to reduce my stress and not appear so anxious," a woman who had anxiety triggered by a critical daughter-in-law came up with the following: "Not to have my heart racing and not to talk a mile a minute but, rather, to breathe calmly and be able to listen when my son and his wife visit."

Normalization of the EEG

Normalization includes amplitude training (increasing or decreasing the power in specified frequency bands) and coherence training (the amount of shared activity between two areas). This can be done with single- and two-channel training for which there are no norms other than the theta–beta power ratios used to identify problems with attention. Those ratios are used when working with people who have ADHD (Monastra et al., 1999). When doing LORETA *z*-score NFB, one trains to achieve amplitudes and coherence measures that are within a normal range according to *z*-scores based on database norms. (A *z*-score has a mean of 0 and a standard deviation of 1, and the normal range is usually considered to be + or –1, the range in which two-thirds of the scores fall.)

Figure 9.11 illustrates the EEG at a single location (FCz) in a complex case in which single-channel training at that site was very effective.

The data shown in Figure 9.12 were from a 50-year-old woman who presented before LORETA *z*-score NFB was available. We did the full 19-channel assessment to better design single-channel training that, in this case, was done at FCz. She had a long history of anxiety, occasionally panic, and generally her emotions were quite labile. She had very high high-frequency beta ratios; for example, the 3-minute sample shown in the figures showed 23–35 Hz/13–15 Hz = 2.52. (We find that > 1.55 can correlate with ruminating or a very busy brain.) The ratio 19–22 Hz/11–12 Hz was 3.05. (We find that > 1.0 often correlates with anxiety or intensity.) In light of our previous discussion of the effects of drugs on the EEG, it is of interest that she reported that stimulants, benzodiazepines, and SSRIs, all of which raise the amplitude of beta frequencies, had all precipitated terrible panic attacks. Spindling beta, characterized by bursts of high-amplitude sinusoidal waves in a narrow frequency range, may represent an irritable cortex. She was medication-free at the time of this recording.

We find that mental states correspond to specific frequency bandwidths, but they may vary slightly from one individual to the next. The trainer, therefore, has the client experiment with making relevant brain-wave frequency bands rise and fall by changing mental state. Then the client knows what those frequencies represent in terms of his or her own mental state. The particular frequencies trained in one case would not be appropriate for all clients. As previously emphasized, we do not advocate a set protocol. The clinician tailors the intervention to each of the client's patterns.

LNFB for Postconcussion Syndrome

Some clients present not just with problems handling stress and anxiety but with more complex difficulties that are due to diffuse axonal injury (DAI) from one or more concussions (Thompson & Hagedorn, 2012). The 19-channel assessment with LORETA

FIGURE 9.11. Spectral array at the bottom graphs power (*Y*-axis) by frequency (*X*-axis) 3 Hz–61 Hz. This client presented with anxiety and panic. Beta spindles are seen in the above figure at around 64 seconds (frequency of 25 Hz seen in the spectral array at the bottom) and at 65.5 seconds (frequency of 20 Hz).

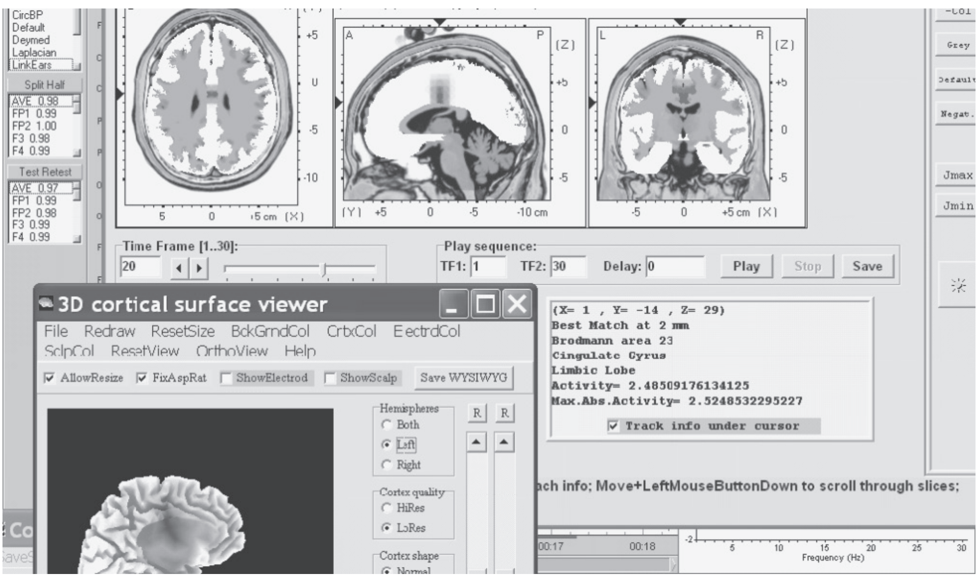


FIGURE 9.12. For this client with anxiety, high-amplitude beta activity at 20 Hz was observed at Cz and FCz, both with single-channel and 19-channel recordings. LORETA analysis suggested the best fit for a source was in the anterior cingulate, Brodmann areas 23 and 24. The activity was 2.5 *SD* greater than the mean for her age using the NeuroGuide normative database.

images often demonstrates multiple sites of dysfunction, often in deep midline areas that cannot be directly influenced by surface EEG feedback. In these instances, we may use LNFB combined with HRV training. The majority also have symptoms related to affect networks, including depression, anxiety, irritability ("short fuse"), and less resiliency in the face of stress (Thompson, Thompson, Thompson, & Hagedorn, 2011). In some, the injury appears to have resulted in abnormal EEG findings similar to those found in people with Asperger's syndrome, with less activation in the right temporal–parietal junction, the posterior cingulate, and the left orbital frontal cortex. These patients show changes in their ability to correctly interpret social interactions (Thompson et al., 2010).

These symptoms may be present even many years after they have suffered from one or more concussions or whiplash injuries, a condition referred to as *postconcussion syndrome*. Most people recover completely from a concussion, but 15–30% have ongoing symptoms. The symptoms usually are found to correlate with dysfunction and deviations from the database norms in areas of the brain that are known to be related to the symptoms, and they indicate networks that are not functioning properly in that individual. These often include combinations of the affect, executive, salience, attention, memory, and default networks. No two clients will be exactly alike, but virtually all these clients with postconcussion syndrome complain about cognitive, memory, and affect impairments, with markedly increased irritability, anger, and decreased resilience when under stress. It can be so serious that other family members state that the patient is no longer the person they knew. When there are postconcussion symptoms, one almost invariably finds results from QEEG, ERP, CPT, HRV, balance testing, and neurocognitive tests that differ from normal. We are only focusing on QEEG and HRV in this chapter. For such complex cases, LORETA *z*-score neurofeedback is appropriate because there are so many areas to train, and many are deep in the cortex.

The details of this type of training are described in Thompson and Thompson (2015a), so a detailed description is not repeated here. The 19-channel assessment of the EEG gives surface brain maps. However, surface sites on brain maps that show deviations from database norms may be quite distant from the principle BA that is the source of the deviation. That source is identified through LORETA mathematics, as developed by Roberto Pascual-Marqui. For example, BA 25 is a deep, ventral, midline structure located in the subgenual area ("below the knee" of the corpus callosum), and it is often localized as the source of observed deviations that show up on brain maps at F3 and/or F4 and Fz. For this reason, and because LNFB can target multiple areas, frequencies, and amplitudes at BA sites, in addition to connectivity including coherence and phase between these BAs, we tend now to use LNFB in very complex cases and single- and two-channel NFB for people with less complex presentations. The advantage of LNFB over traditional neurofeedback is increased specificity of the training and the possibility of training activity at locations not otherwise easily influenced by conventional surface EEG training. In addition, there is the possibility of training many targeted areas (BAs) at the same time. One can also target networks and even different conditions; for example, there is a program for addressing PTSD that is being used with veterans. A more complete overview of LNFB can be found in Thompson and Thompson (2015).

CASE EXAMPLE: PUTTING THEORY INTO PRACTICE

Some clients who present with anxiety turn out to have other associated disorders and needs. In these cases, the goal of training will include the reduction of a defined symptom or problem, such as a learning disability, seizure disorder, movement disorder, stroke, speech disorder, depression, anxiety, Asperger's syndrome traits, and so on. Although a client has requested NFB services in order to decrease the effects of stress and optimize academic and workplace performance, one or more other conditions either may be the source of the stress or may interfere with the client's performance. ADHD or a mild learning disability are two common coexisting conditions. In the case of Diane, a different syndrome was present, albeit in a mild form. She displayed many features of Asperger's syndrome (Thompson et al., 2010). In some ways, this accelerated her learning, because she was very compliant with concrete suggestions and would follow the trainer's advice concretely and perfectly. She also had symptoms of attention-deficit disorder (ADD), which included a tendency to drift off or even fall asleep when studying, a specific learning disability related to spatial reasoning, a degree of dysphoria (not meeting criteria for clinical depression), and a clinically significant panic disorder. On the plus side, she had a verbal IQ in the gifted range and was a very honest and pleasant person.

Diane's case is used in part because it is an excellent example of findings that in recent years would have led to combining LNFB with HRV training. However, she presented long before LNFB was available. Fortunately, she responded extremely well to training done with single-channel NFB plus biofeedback. Our theory is that, by training over FCz (halfway between Fz and Cz), we were influencing whole networks that are linked by the cingulum and uncinate fasciculus, including executive, attention, salience, affect, and default networks.

First Interview/Session: History and EEG Assessment

Diane was unable to pass her doctoral-level practical examinations in chiropractic medicine due to anxiety, despite already holding two doctoral degrees. She had been diagnosed with ADD, and she had social anxiety and had always found relating in both personal and professional situations very stressful. She conquered the stress of written examinations by withdrawing from other activities and overlearning the subject matter. She could not effectively use this overlearning formula for practical and oral examinations, however, due to the interpersonal stress. Panic attacks, anxiety, and tension had led to her failing the practical examination twice. She had attempted to deal with her anxiety and ADD symptoms through medications prescribed by her physician but suffered side effects on stimulant medication, including increased anxiety. She had ongoing psychotherapy with a psychologist. She had also tried relaxation techniques, dietary approaches, and chiropractic interventions. She faithfully followed the prescriptions of her practitioners, but her panic attacks, ruminating, and general anxiety had not subsided. She constantly felt stressed and feared that, after years of study, she would never be able to practice.

After thorough history taking and measurement of attentional variables using questionnaires and CPTs (she scored in the bottom 2% for females her age on the Attention Quotient of the Integrated Visual and Auditory [IVA] computerized test of attention), a single-channel EEG assessment was done. The active electrode was at FCz referenced to A2 (the right ear) with ground on the left ear (A1). The findings indicated differences from the EEG pattern that would normally be expected in terms of excess slow-wave activity and, even more pronounced, excess high beta (beta frequencies > 20 Hz).

When initially graphed, Diane demonstrated low amplitude 12–15 and 15–18 Hz and high amplitude (beta spindling) at 21, 23, and 31 Hz. She correlated 20 Hz with anxiety and 31 Hz with negative ruminations.

Figure 9.13 shows 3 seconds of EEG activity that was typical for Diane when she was feeing stressed. In a typical woman of the same age, we would see a different picture; namely, an eyes-open recording of EEG without high-amplitude beta spindling. A normal spectral pattern would usually show a low voltage and relatively flat histogram with a gradual reduction in power as frequencies get higher.

Diane's EEG demonstrated a contrasting picture. It shows an unusual form of beta measured at FCz called *spindling beta*. The spectrum shows extremely high 31 Hz activity in conjunction with very low 13–15 Hz (SMR) activity. High-amplitude 19–21 Hz activity can also be observed. Throughout the remainder of the record, this pattern of a rise around 31 Hz would repeatedly correspond to low amplitude at 11–12 and/or 13–15 Hz. Diane was able to identify that this pattern corresponded to her feeling tense, anxious, and worried.

Diane also showed spindling beta at Cz. These spindles were at 23 Hz. The brain map in Figure 9.14 shows this high amplitude 23 Hz at Cz on the surface EEG. LORETA source derivation is shown in Figure 9.15. It indicates that the origin of this 23 Hz activity is the anterior cingulate, BA 24 (immediately anterior to area 23). The anterior cingulate often appears to be the origin of activity that is found to correspond to anxiety and the syndromes related to anxiety.

Second Interview/Session: Stress Assessment

In addition to Diane's EEG differences indicating vulnerability to stress and high anxiety, there were also alterations in ANS variables characteristic of stress: her heart rate increased and respiration became fast, irregular, and shallow.

Diane's initial stress assessment demonstrated some fewer common responses due to the chronic nature of her stress. Instead of peripheral skin temperature decreasing with

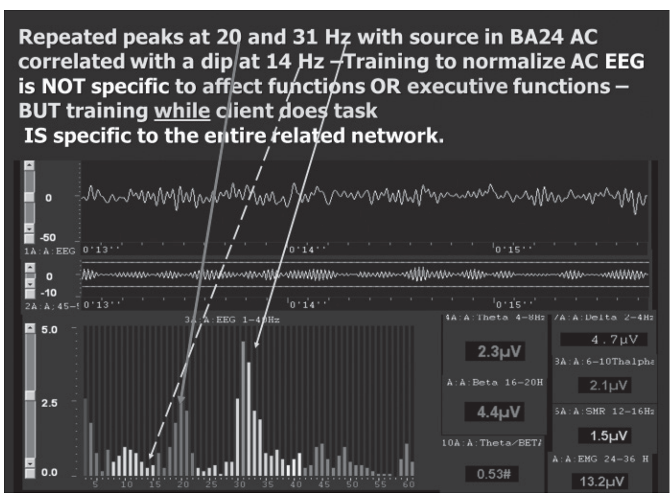


FIGURE 9.13. When initially graphed, Diane demonstrated low amplitude at 12–15 and 15–18 Hz and high amplitude (beta spindling) at 21 and 31 Hz. She correlated 20-Hz increases with anxiety and 31 Hz with negative ruminations. From Thompson and Thompson (2015a). Copyright © 2015 Michael Thompson and Lynda Thompson. Reprinted with permission from the Association for Applied Psychophysiology and Biofeedback.

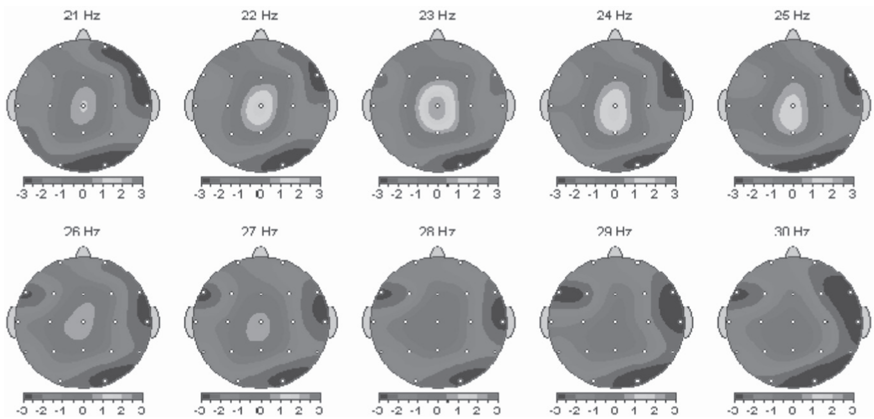


FIGURE 9.14. This eyes-closed Laplacian-montage, relative-power brain map shows that the amplitude of beta at 23 Hz is > 3 *SD* above the NeuroGuide database mean at Cz. This corresponds to the beta spindling seen at this frequency in the raw EEG.

the math stress, math was her weak area, and it elicited a chronic stress reaction. The math stress initially elicited a rise in finger temperature rather than a fall. Skin temperature was in the mid-80s and skin conductance was below 3 mhos. Being very conscientious, she followed instructions for breathing at six breaths per minute but was anxious and worried about how well she was doing. At the beginning of the session, her anxiety was high, and her skin temperature was lower and her EDR higher than at the end of the session, when she said she was feeling quite a bit calmer. Her forehead EMG was also higher—22 μV—at the beginning of the session, and lower—13 μV—at the end of the session. We concluded, while discussing the profile with her, that breathing, EMG, and skin temperature could all be used as her indicators of stress. We further agreed that SC

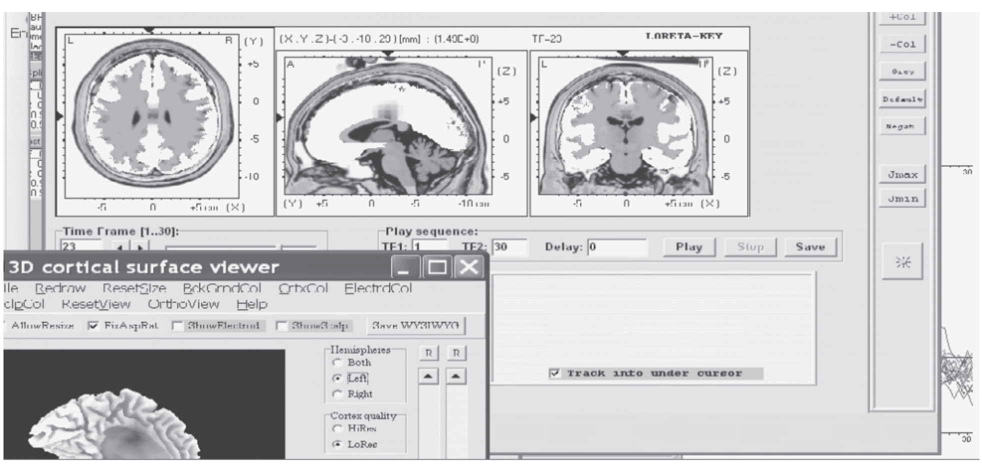


FIGURE 9.15. LORETA analysis shows that this high-amplitude 23 Hz beta activity may be originating in the anterior cingulate. In this figure, it is 1.6 *SD* greater than the database mean in BA 24.

might be a useful measure of alertness and that it was valuable to maintain alertness, along with being calm and relaxed.

Sessions 3–6: Initial Training Sessions Using NFB + Biofeedback

During the first few training sessions, the goal was for Diane to control variables in the following order: (1) breathing diaphragmatically at six breaths per minute; (2) decreasing her forehead EMG while continuing the breathing at six breaths per minute; (3) maintaining diaphragmatic breathing and low EMG while, in her words, "empty[ing] my mind." This meant she stopped ruminating. When that occurred, she noted that both 23–34 Hz and 30–32 Hz activity were reduced.

One training screen that helped her to follow changes in a number of EEG variables, in addition to HRV and other biofeedback variables, is shown in Figure 9.16. This shows a screen from the Infiniti program (hardware and software produced by Thought Technology) that allows the client to observe HRV beginning to synchronize with diaphragmatic breathing. At this juncture, Diane had not quite achieved this, but she eventually did so when she began to breathe at a rate of six breaths per minute. When this synchrony occurred, the HRV amplitude increased. This screen also allowed her to observe skin temperature, EDR, and SC while she increased her 13–15 Hz (SMR, "calm waves"), and decreased her high beta; namely, 19–22 Hz and 23–35 Hz. Diane's skin temperature

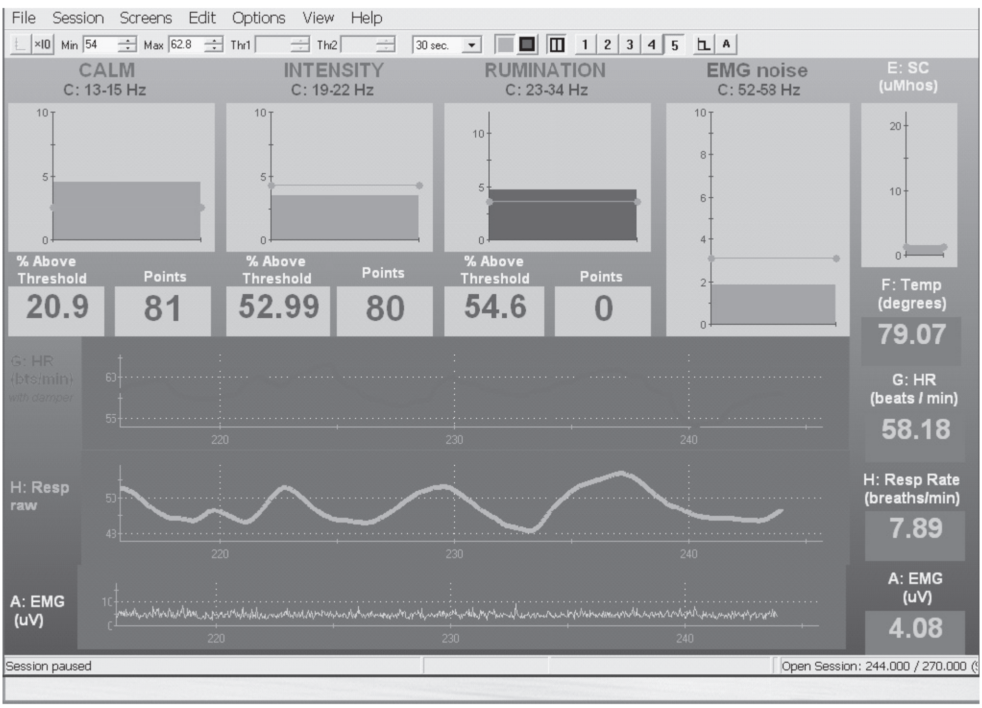


FIGURE 9.16. All the bar graphs are in color in the biograph program, but here they appear in light gray for all but the ruminations 23–35 Hz, signifying that the patient is achieving her goals for areas called Calm, Intensity, EMG (muscle tension) noise, and SC (skin conduction). The Rumination bar graph, however, is in a darker shade, suggesting that she is ruminating and worrying.

was normally higher than shown in the figure, and it was not necessary to have it or the EDR on most feedback screens after the first few sessions. Forehead EMG was sometimes included on feedback screens: here it is high, but it did eventually drop to < 2 microvolts.

Even when surface EMG was low, Diane still might experience the sensation of tension. Perhaps this may be thought of as corresponding to the findings of Gevirtz, Shannon, Hong, and Hubbard (1997) that muscle spindle fusiform fiber tension can exist without surface EMG showing tension. The muscle spindle tension appears, in large part, to relate to sympathetic drive. Sympathetic drive may be effectively decreased through training of respiration and HRV. The muscle spindle fusiform fiber tension may also be related to gamma motor efferent overactivity from the red nucleus (Figure 9.4). Increasing SMR decreases firing of the red nucleus, as documented by Sterman (2000a). Feedback that emphasizes both EEG and ANS biofeedback theoretically should be effective for stress management, and combining them was highly effective for Diane.

By her sixth session, Diane was trying to combine effortless diaphragmatic breathing and maintaining an external focus with a technique of looking at old paintings and her favorite dog pictures (she loved animals), while at the same time opening her awareness to the gestalt of these pictures. She experienced her hands getting warm, her forehead and shoulder muscles relaxing, and a sense of calm. She said that, when she truly sensed this peaceful, open, external awareness, then everything seemed to fall into place. In addition to control of breathing, raising peripheral skin temperature to 93 degrees and SC to 5–7 mhos, there were shifts in her EEG pattern: 19–21 Hz, 24–32 Hz, and 4–7 Hz all dropped markedly while 11–15 Hz and 17 Hz rose. She could only get into this state for a few minutes at a time near the end of the session, but she was elated at how this corresponded to feeling peaceful and calm, yet alert.

Sessions 7–9

At this juncture, some cognitive-behavioral work was added to augment the feedback, which was continued. Diane was given two strategies to help her generalize what she was learning in her sessions to her everyday life. The first method is called "Attach a Habit to a Habit." She was asked to practice her diaphragmatic breathing with hand warming and relaxation of her forehead, neck, and shoulders, combined with feeling calm and "emptying her mind," before (and later during) each activity that she listed as being a "habit" or daily "routine," such as while brushing her teeth and while driving.

The second strategy was called SMIRB: "Stop My Irritating Ruminations Book." It is a method for compartmentalizing negative thoughts. The book was a pocket-sized daily calendar. The front pages were for her positive goals in each key area of her life. The back pages were set aside to be her SMIRB. She wrote one "worry" heading at the top of the left-hand page and listed below it the repetitive thought or thoughts that kept coming into her head. She then put the second worry area on the next left-hand page, working her way from the back toward the middle of the book. She was surprised to discover that she had only a small number of worry areas and only a few repetitive thoughts under each area. The problem for her was that they were continually repeated in her head and were preventing her from performing important activities. She agreed that she would worry about these things at one, and only one, preset time each day. She also agreed that, if she woke up in the middle of the night, she would ask herself, before opening her eyes, if the thing she was ruminating about was in her SMIRB. If it was, she would remind herself that this was sleep time, not worry time, and would not open her eyes but just think of a repetitive pleasant activity, do her breathing, and relax until she fell back asleep. If, however, the thought was not in her SMIRB, then she would open her eyes, turn on her light, write it into her SMIRB, and then leave that thought and go back to sleep. If she ever did think of a solution to one of these worries, she would enter it on the right-hand page opposite the negative thought.

Diane carried out these two strategies faithfully. The speed with which she began to take control of her mental states, as reflected in her EEG changes, physiological shifts, and her self-report of controlling her negative thinking was probably associated with her practicing every day. By her 10th training session, she was feeling some control over a number of variables. The screen shown in Figure 9.17 was one of Diane's favorites. She could now, by the end of a session, sometimes manage to keep 24–32 Hz and 19–23 Hz very low and 11–13 Hz up for a short while (1–2 minutes). The "%-over-threshold" computations were originally set at 50%, according to her baseline assessment. Now she was at 25, 36, and 57%, which are very good values that represent progress, indicating she was decreasing percentage of time for the two inhibit frequency ranges and increasing percentage of time for the range set for reward. Note, too, that forehead EMG was at 3 μV (down from > 13) and she was keeping her breathing at about one breath every 10 seconds (six per minute). At this stage, she could not hold this state for more than about 2 minutes, but her comment was, "It feels wonderful."

Sessions 10–20: Intermediate Sessions Using NFB + Biofeedback

At this stage, Diane had sufficient self-control to experiment with producing and letting go of specific mental states. She was able to demonstrate to herself the precise frequencies that correlated with several mental states: (1) feeling emotionally intense and anxious versus feeling calm and relaxed; (2) feeling worried, with negative, circular, nonproductive ruminations versus a mind empty of distracting thoughts and focused on the problem at hand; (3) being focused on one intrusive thought after another versus an open focus that flexibly could concentrate on a single target in a calm mental state.

To accomplish this practice of being in and out of specific mental states, she used a sailboat screen, shown in Figure 9.17. We altered the screen so that the bar graph could

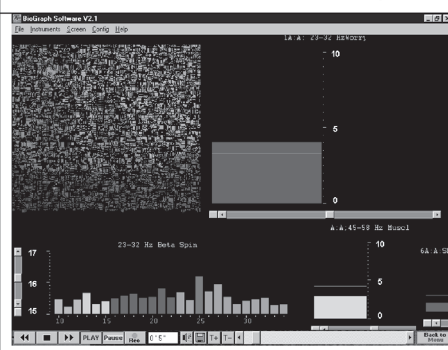
	Sailboat disappears 23–32 Hz high
Diane said that, if she *"emptied her mind"* of negative, circular thinking and felt calm and externally oriented, then she could maintain a low bar-graph and an intact sailboat. The moment she started worrying, the music would stop and the sailboat would disappear as is shown opposite. This occurred because EEG amplitude for 23–32 Hz is above the threshold. Note that the 45–58 Hz is low. It would reflect the effect of muscle activity on the EEG.	

FIGURE 9.17. Sailboat screen to gain control of ruminating.

represent different frequency bandwidths. Initially, we did not label the bar graph, and we tried not to even suggest to her what she might do in order to make the picture of the boat visible or make it disappear. Our goal was to make the feedback clear so that Diane could feel confident that what she experienced was being consistently reflected on the computer monitor. We let her discover which frequencies were related to which mental states.

Diane noted that if she felt that she was going into a panic at home she could calm herself by breathing at six breaths per minute while she emptied her mind. She said that practicing this in sessions and then practicing it while she drove home meant that she could attain the desired state quite quickly, and no panic attack ensued.

We do not doubt that different mental states do correspond to different bandwidths but note that it may be different frequencies for different individuals. (Age also plays a role, with frequencies moving higher with age. Pediatric alpha may be at 7 Hz but, in adults, the peak alpha frequency is around 10 Hz.)

Sessions 21–35

Diane interspersed one or more of the sailboat exercises (using various frequency bands) with her more general screens that gave her the opportunity to work on biofeedback goals (such as respiration, forehead or trapezius EMG, or peripheral skin temperature) while controlling one or more of the following: high-frequency beta, high alpha (11–12 Hz), SMR, and theta. The goal was self-regulation. After 30 sessions, when she really had voluntary control of combining emptying her mind (reduced high-frequency beta spindling) and breathing at six breaths per minute, she reported that, for the first time in as long as she could remember, she had not experienced a panic attack for a full week. Diane's training for the high-beta bandwidth was done at F2 (frontally and just to the right of the midline) and, at other times, at FCz, where beta spindling had been observed. (FCz is usually the site where activity originating from the anterior cingulate, BA 24, is observed.) The right frontal area is overactive in panic disorder, and these findings suggest that Diane had achieved some control of this episodic overactivity in the right frontal area. For Diane, the specific goal was the ability to sustain a mental state of feeling relaxed and calm, yet alert, completely free of unwanted thoughts and ruminations, and able to switch appropriately from broad external awareness to narrow focus on a problem-solving task.

Sessions 36–40

At this juncture, Diane was working to have control of all variables automatic and achievable, even while being asked questions similar to those that had previously been experienced in the stress-producing situation of oral examinations. In the screen shown in Figure 9.18, note that she has gone from the usual starting point for %-over-threshold calculations of 50 or 55% to 79% for the 11–15 Hz frequency band (she has increased SMR) and dropped the 25–36 Hz band to a remarkably low 17% of the time over threshold (less busy brain and muscle tension). Her forehead EMG had originally been above 12 and had now dropped to 2.6. She kept her breathing steady at six breaths per minute, which is her resonance frequency. She had learned to have a longer exhale compared to inhale. This is important because a longer exhale is associated with more parasympathetic activity and, additionally, Sterman's early research with animals showed that SMR is produced on the exhale.

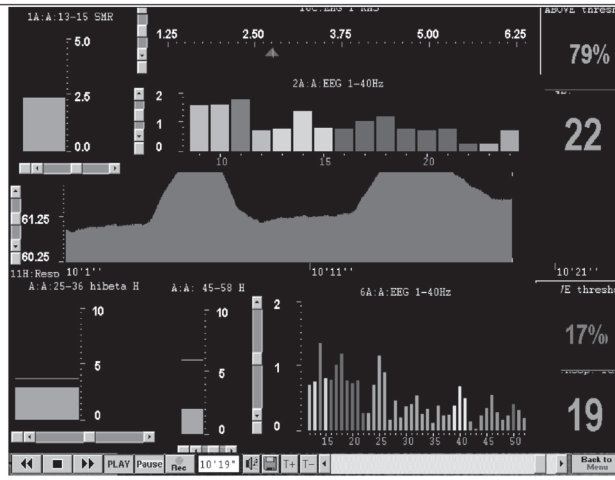
	13-–15 Hz Spectrum 8–24 Hz; peak at 11 Hz
Note also that the pink bar indicating maximum amplitude in the spectral width shown in each spectrum has moved from areas in the high-frequency beta range above 20 Hz to where she wanted to have it, at 11 Hz and 14 Hz respectively. More remarkable is that she could, for the most part, keep it in those general areas, though at times 25 Hz would rise (as is seen here) and she would have to remind herself to stop ruminating.	
	25–36 Hz 45–58 Hz 14 Hz highest

FIGURE 9.18. Screen showing EEG thresholds, spectrum, EMG, and respiration.

After 40 sessions of training, a reassessment demonstrated shifts in Diane's EEG patterns, improved performance on the CPT (from 2nd to 37th percentile for her Attention Quotient on the IVA), and decreased symptoms of ADD, anxiety, and depression on self-report questionnaires. Most important, Diane retook her practical examinations shortly thereafter and achieved passing grades. She subsequently set up practice in her profession. Her demeanor also changed, with the old anxiety, evidenced by averted gaze and stooped shoulders, replaced by direct looks, smiles, and posture that reflects confidence.

SUMMARY AND CONCLUSION

Both the brain and the body react to stress. A certain amount of stress can improve performance, with this amount of stress termed *eustress*. The normal stress response involves a return to baseline when the stress is over. If a person does not readily return to homeostasis, however, stress can build over time. Chronic stress can produce pathological alterations in normal brain, physiological, and biochemical functioning. Thus developing the ability to deal with stress and return to a healthy baseline after experiencing stress is imperative for healthy functioning.

Individuals are capable of identifying mental states that correlate to EEG frequency bands, as Joe Kamiya originally demonstrated with students who could identify when they were producing alpha (Kamiya, 1968, 1979). With NFB training, first developed by Sterman, a person can train his or her brain to shift the EEG patterns, becoming more flexible in his or her ability to find the correct mental state to fit the task demands of the moment. Individuals can also identify mental states that correlate with physiological changes, such as heart and respiratory rates, SC, temperature, and muscle tension. Using biofeedback from the EEG and other physiological variables, especially HRV, people can shift their responses to stressors and optimize their performance in academic, work, athletic, and social situations. The relative importance of different variables will differ between people and according to underlying strengths or pathologies.

Although it is reasonable to propose that a combined NFB and biofeedback approach is helpful in assisting individuals to deal with stress and optimize their performance, it should be recognized that this approach still requires more research. It should also be emphasized that NFB and biofeedback are part of an overall approach to lifestyle improvement. Such factors as nutrition, sleep, exercise, and evoking constructive patterns of viewing oneself and relating to others are additional components of a comprehensive training program.

Fine-tuning the ability to self-regulate both attention and emotions is achievable through NFB training. Adding HRV training and other biofeedback measures, such as temperature and electrodermal, to NFB makes the intervention particularly effective for stress management. A human being is one entity, not a mind–body dichotomy, so combining NFB and biofeedback to improve self-regulation skills is the most efficient way to build resilience that leads to effective management of the stresses of everyday life.

REFERENCES

Aich, P., Potter, A. A., & Griebel, P. J. (2009). Modern approaches to understanding stress and disease susceptibility: A review with special emphasis on respiratory disease. *International Journal of General Medicine, 2,* 19–32.

Arns, M., de Ridder, D., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*(3) 180–189.

Berger, H. (1929). Ueber das Electrenkephalogramm des Menschen. *Archiv fuer Psychiatrie und Nervenkrankheiten, 87,* 527–570.

Beauregard, M., & Levesque, J. (2006). Functional magnetic resonance imaging investigation of the effects of neurofeedback training on the neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 31*(1), 3–20.

Bluhm, R. L., Williamson, P. C., Osuch, E. A., Frewen, P. A., Stevens, T. K., Boksman, K., et al. (2009). Alterations in default mode network connectivity in posttraumatic stress disorder related to early-life trauma. *Journal of Psychiatry and Neuroscience, 34*(3), 187–194.

Cahill, L., Gorski, L., & Le, K. (2003). Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learning and Memory, 10*(4), 270–274.

Carmichael, S., & Stoddard, S. L. (1992). *The adrenal medulla, 1989–1991.* Boca Raton, FL: CRC Press.

Catani, M., & de Schotten, M. T. (2012). *Atlas of human brain connections.* New York: Oxford University Press.

Daniels, J. K., McFarlane, A. C., Bluhm, R. L., Moores, K. A., Clark, C. R., Shaw, M. E., et al. (2010). Switching between executive and default mode networks in posttraumatic stress disorder: Alterations in functional connectivity. *Journal of Psychiatry and Neuroscience, 35*(4) 258–266.

Davidson, R. J. (1995). Cerebral asymmetry, emotion and affective style. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 369–388). Cambridge, MA: MIT Press.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35,* 607–614.

Devinsky, O., Morrell, M., & Vogt, B. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain, 118,* 279–306.

Duffy, F. H., Hughes, J. R., Miranda, F., Bernad, P., & Cook, P. (1994). Status of quantitative

EEG (QEEG) in clinical practice. *Clinical Electroencephalography, 25,* vi–xxii.

Duffy, F. H., Iyer, V. G., & Surwillo, W. W. (1989). *Clinical electroencephalography and topographic brain mapping: Technology and practice.* New York: Springer-Verlag.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51,* 1–12.

Fisch, B. J. (1999). *Fisch and Spehlmann's EEG Primer: Basic principles of digital and analog EEG* (3rd ed., rev., & enlarged). New York: Elsevier.

Gagnon, S. A., & Wagner, A. D. (2016). Acute stress and episodic memory retrieval: Neurobiological mechanisms and behavioral consequences. *Annals of the New York Academy of Sciences, 1369*(1), 55–75.

Gevirtz, R. N. (2011) Biofeedback in the treatment of stress. *Cleveland Clinic Journal of Medicine: Proceedings of the 2010 Heart–Brain Summit, 78*(Suppl. 1), s59–s60.

Gevirtz, R., Shannon, S., Hong, D., & Hubbard, D. (1997). Myofacial trigger points show spontaneous needle EMG activity. *Pain, 69,* 65–73.

Ghaziri, J., Tucholka, A., Larue, V., Blanchette-Sylvestre, M., Reyburn, G., Gilbert, G., et al. (2013). Neurofeedback training induces changes in white and gray matter. *Clinical EEG and Neuroscience, 44*(4), 265–272.

Green, D. J., Barnea, A., Herzberg, K., Rassis, A., Neta, M., Raz, A., et al. (2008). Measuring attention in the hemispheres: The Lateralized Attention Network Test (LANT). *Brain and Cognition, 66*(1), 21–31.

Gruzelier, J. H., & Egner, T. (2003). Ecological validity of neurofeedback modulation of slow wave EEG enhances musical performance. *Neuroreport, 14*(1).

Hagedorn, D. (2015). Post-traumatic stress disorder. In M. Thompson & L. Thompson (Eds.), *The neurofeedback book: An introduction to basic concepts in applied psychophysiology* (2nd ed., pp. 684–690). Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Hammond, C., & Gunkelman, J. (2001). *The art of artifacting.* Retrieved from *www.ISNR.org.*

Hardt, J. V., & Kamiya, J. (1978). Anxiety change through electroencephalographic alpha feedback seen only in high anxiety subjects. *Science, 201,* 79–81.

Howe, L. L. S. (2009). Giving context to post-deployment post-concussive-like symptoms: Blast-related potential mild traumatic brain injury and comorbidities. *Clinical Neuropsychologist, 23*(8), 1315–1337.

Jasper, H. (1958). Report of the committee on methods of clinical examination in electroencephalography. *EEG and Clinical Neurophysiology, 10,* 374.

John, E. R., Easton, P., & Isenhart, R. (1997). Consciousness and cognition may be mediated by multiple independent coherent ensembles. *Consciousness and Cognition, 6,* 3–39.

Kamiya, J. (1968, April). Conscious control of brainwaves. *Psychology Today.*

Kamiya, J. (1979). Autoregulation of the EEG alpha rhythm: A program for the study of consciousness. In E. Peper, S. Ancoli, & M. Quinn (Eds.), *Mind body integration: Essential readings in biofeedback* (pp. 289–298). New York: Plenum Press.

Kennedy, S. H., Giacobbe, P., Rizvi, S. J., Placenza, F. M., Yasunori, N. Y., Mayberg, H. S., et al. (2011). Deep brain stimulation for treatment-resistant depression: Follow-up after 3 to 6 years. *American Journal of Psychiatry, 168*(5), 502–510.

Kerick, S., & Allender, L. E. (2006). Effects of cognitive workload on decision accuracy, shooting performance, and cortical activity of soldiers (Report). Aberdeen, MD: U.S. Army Research Laboratory, Human Research and Engineering Directorate, Aberdeen Proving Ground. Retrieved from *https://apps.dtic.mil/dtic/tr/fulltext/u2/a433487.pdf.*

Kerick, S. E., Douglass, L. W., & Hatfield, B. D. (2004). Cerebral cortical adaptations associated with visuomotor practice. *Medical Science in Sports and Exercise, 36*(1), 118–129.

Klimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews, 29,* 169–195.

Klimesch, W., Pfurtscheller, G., & Schimke, H. (1992). Pre- and post-stimulus processes in category judgement tasks as measured by event-related desynchronization (ERD). *Journal of Psychophysiology, 6,* 185–203.

Landers, D. M., Petruzzello, S. J., Salazar, W., Crews, D. J., Kubitz, K. A., Gannon, T. L., et al. (1991). The influence of electrocortical biofeedback on performance in pre-elite archers. *Medicine and Science in Sports and Exercise, 23*(1), 123–128.

Lanius, R. A., Bluhm, R. L., Coupland, N. J., Hegadoren, K. M., Rowe, B., Theberge, J., et al. (2010). Default mode network connectivity as a predictor of post-traumatic stress disorder symptom severity in acutely traumatized subjects. *Acta Psychiatrica Scandinavica, 121*(1), 33–40.

Lazar, S. W., Kerr, C. E., Wasserman, R. H., Gray, J. R., Greve, D. N., Treadway, M. T., et al. (2005). Meditation experience is associated with increased cortical thickness. *NeuroReport, 16,* 1893–1897.

Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback and Self-Regulation, 21*(1), 106–111.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback treatment for attention deficit/hyperactivity disorders. *Biofeedback and Self-Regulation, 16*(3), 201–225.

Lubar, J. F. (1997). Neocortical dynamics: Implications for understanding the role of neurofeedback and related techniques for the enhancement of attention. *Applied Psychophysiology and Biofeedback, 22*(2), 111–126.

Lubar, J. F., & Bahler, W. W. (1976). Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. *Biofeedback Self-Regulation, 7,* 77–104.

Lubar, J. F., Congedo, M., & Askew, J. H. (2003). Low-resolution electromagnetic tomography (LORETA) of cerebral activities in chronic depressive disorder. *International Journal of Psychophysiology, 49*(3), 175–185.

Lubar, J. F., & Lubar, J. (1999). Neurofeedback assessment and treatment for attention deficit/hyperactivity disorder. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 103–143). San Diego, CA: Academic Press.

Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioural changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback and Self-Regulation, 3,* 293–306.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in TOVA scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-Regulation, 21*(1), 83–99.

Mayberg, H. S. (2003). Modulating dysfunctional limbic-cortical circuits in depression: Towards development of brain-based algorithms for diagnosis and optimized treatment. *British Medical Bulletin, 65,* 193–207.

Mayberg, H. S., Brannan, S. K., Tekell, J. L., Silva, J. A., Mahurin, R. K., McGinnis, S., et al. (2000). Regional metabolic effects of fluoxetine in major depression: Serial changes and the relationship to clinical response. *Biological Psychiatry, 48*(8), 830–843.

Mayberg, H. S., Lozano, A. M., Voon, V., McNeely, H. E., Seminowicz, D., Hamani, C., et al. (2005). Deep brain stimulation for treatment-resistant depression. *Neuron, 45*(5), 651–660.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine, 153*(18), 2093–2101.

McKnight, J. T., & Fehmi, L. T. (2001). Attention and neurofeedback synchrony training: Clinical results and their significance. *Journal of Neurotherapy, 5*(1/2), 45–62.

Mikosch, P., Hadrawa, T., Laubreiter, K., Brandl, J., Pilz, J., Stettner, H., et al. (2010). Effectiveness of respiratory-sinus-arrhythmia biofeedback on state-anxiety in patients undergoing coronary angiography. *Journal of Advanced Nursing, 66*(5), 1101–1110.

Monastra, V. J., Lubar, J. F., & Linden, M. (2001). The development of a quantitative electroencephalographic scanning process for attention deficit-hyperactivity disorder: Reliability and validity studies. *Neuropsychology, 15*(1), 136–144.

Monastra, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., et al. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology, 13*(3), 424–433.

Pascual-Marqui, R. D., Esslen, M., Kochi, K., & Lehmann, D. (2002). Functional imaging with low resolution electromagnetic tomography (LORETA): A review. *Methods and Findings in Experimental and Clinical Pharmacology, 24*(Suppl. C), 91–95.

Patterson, S., Moran, P., Epel, E., Sinclair, E., Kemeny, M. E., Deeks, S. G., et al. (2013). Cortisol patterns are associated with T-cell activation in HIV. *PLOS ONE, 8*(7), e63429.

Plotkin, W. B., & Rice, K. M. (1981). Biofeedback as a placebo: Anxiety reduction facilitated by training in either suppression or enhancement of brain waves. *Journal of Consulting and Clinical Psychology, 49,* 590–596.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychiatry, 74,* 116–143.

Posner, M. I., Sheese, B. E., Odludas, Y., & Tang, Y. (2006). Analyzing and shaping human attentional networks [Special issue]. *Neural Networks, 19,* 1422–1429.

Prinsloo, G. E., Rauch, L., Lambert, M. I., Muench, F., Noakes, T. D., & Derman, W. E. (2011). The effect of short duration heart rate variability (HRV) biofeedback on cognitive performance during laboratory induced cognitive stress. *Applied Cognitive Psychology, 25*(5), 792–801.

Rabe, S., Beauducel, A., Zollner, T., Maercker, A., & Karl, A. (2006). Regional brain electrical activity in posttraumatic stress disorder after motor vehicle accident. *Journal of Abnormal Psychology, 115*(4), 687–698.

Randall, M. (2010, Fall). The physiology of stress: Cortisol and the hypothalamic-pituitary-adrenal axis. *Dartmouth Undergraduate Journal of Science.* Retrieved from *https://sites.dartmouth.edu/dujs/2011/02/03/the-physiology-of-stress-cortisol-and-the-hypothalamic-pituitary-adrenal-axis.*

Reiner, R. (2008). Integrating a portable biofeedback device into clinical practice for patients with anxiety disorders: Results of a pilot study. *Applied Psychophysiology and Biofeedback, 33,* 55–61.

Rice, K. M., Blanchard, E. B., & Purcell, M. (1993). Biofeedback of generalized anxiety disorder: Preliminary results. *Biofeedback and Self-Regulation, 18*(2), 93–105.

Robbins, J. (2000). *A symphony in the brain.* New York: Atlantic Monthly Press.

Rosso, I. M., Weiner, M. R., Crowley, D. J., Silveri, M. M., Rauch, S. L., & Jensen, J. E. (2014). Insula and anterior cingulate GABA levels in post-traumatic stress disorder: Preliminary findings using magnetic resonance spectroscopy. *Depression and Anxiety, 31*(2), 115–123.

Scott, J. C., Matt, G. E., Wrocklage, K. M., Crnich, C., Jordan, J., Southwick, S. M., et al. (2015). A quantitative meta-analysis of neurocognitive functioning in posttraumatic stress disorder. *Psychological Bulletin, 141*(1), 105–140.

Selye, H. (1976). *The stress of life* (rev. ed.). New York: McGraw-Hill.

Sheer, D. E. (1977). Biofeedback training of 40 Hz EEG and behavior. In N. Burch & H. I. Altshuler (Eds.), *Behavior and brain electrical activity.* New York: Plenum Press.

Shields, G., Sazma, M. A., & Yonelinas, A. P. (2016). The effects of acute stress on core executive functions: A meta-analysis and comparison with cortisol. *Neuroscience and Biobehavior Review, 68,* 651–668.

Smith-Pellettier, C. (2002, August). *The amygdala–hypothalamus–pituitary–adrenal axis.* Paper presented at the annual meeting of the Canadian Medical Association, Calgary, Alberta, Canada.

Sridharan, D., Levitin, J., & Menon, V. (2008). A causal role for the right fronto-insular cortex in switching between executive-control and default-mode networks. *Proceedings of the National Academy of Sciences of the USA, 105*(34), 12569–12574.

Steriade, M., & McCarley, R. W. (2005). *Brain control of wakefulness and sleep.* New York: Springer.

Sterman, M. B. (1999). *An atlas of topometric clinical displays.* Los Angeles: Sterman-Kaiser Imaging Labs.

Sterman, M. B. (2000a). EEG markers for attention deficit disorder: Pharmacological and neurofeedback applications. *Child Study Journal, 30*(1), 1–22.

Sterman, M. B. (2000b). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography, 31*(1), 45–55.

Sterman, M. B., & Thompson, L. (2014). Neurofeedback for seizure disorders. In D. S. Cantor & J. R. Evans (Eds.), *Clinical neurofeedback: Applications and techniques for treatment* (pp. 301–314). San Diego, CA: Academic Press.

Tan, G., Shaffer, F., Lyle, R., & Teo, I. (2016). *Evidence-based practice in biofeedback and neurofeedback* (3rd ed.). Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Tan, G., Thornby, J., Hammond, D. C., Strehl, U., Canady B., Arnemann, K., et al. (2009). Meta-analysis of EEG biofeedback in treating epilepsy. *Clinical EEG and Neuroscience, 40*(3), 173–179.

Tator, C. H. (2013). Concussions and their consequences: Current diagnosis, management and prevention. *Canadian Medical Association Journal, 185*(11), 975–979.

Thayer, J., Hansen, A., Saus-Rose, E., & Johnson, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine Publication of the Society of Behavioral Medicine, 37*(2), 141–153.

Thomas, J. E., & Sattlberger, E. (2001). Treatment of chronic anxiety disorder with neurotherapy: A case study. *Journal of Neurotherapy, 7*(2), 14–19.

Thompson, J., & Hagedorn, D. (2012). Multimodal analysis: New approaches to the concussion conundrum. *Journal of Clinical Sport Psychology, 6,* 22–46.

Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback, 23*(4), 243–263.

Thompson, L., Thompson, M., & Reid, A. (2010). Neurofeedback outcomes in clients with Asperger's syndrome. *Journal of Applied Psychophysiology and Biofeedback, 35*(1), 63–81.

Thompson, M. (1978). *A resident's guide to psychiatric education.* New York: Plenum Press.

Thompson, M., & Thompson, L. (2006, Fall). Improving attention in adults and children: Differing electroencephalograhy profiles and implications for training [Special issue]. *Biofeedback.*

Thompson, M., & Thompson, L. (2010). Functional neuroanatomy and the rationale for using EEG biofeedback for clients with Asperger's syndrome. *Journal of Applied Psychophysiology and Biofeedback, 35*(1), 39–61.

Thompson, M., & Thompson, L. (2011, Winter). Improving quality of life using biofeedback plus neurofeedback. *Neuroconnections, 6*(4), 18–21.

Thompson, M., & Thompson, L. (2015a). *The neurofeedback book: An introduction to basic concepts in applied psychophysiology* (2nd ed.). Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Thompson, M., & Thompson, L. (2015b). *Functional neuroanatomy: Organized with reference to networks, lobes of the brain, 10–20 sites, and Brodmann areas.* Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Thompson, M., Thompson, L., Thompson, J., & Hagedorn, D. (2011, Summer). Networks: A compelling rationale for combining neurofeedback, biofeedback and strategies. *NeuroConnections,* 8–17.

van der Kolk, B. A., Hodgdon, H., Gapen, M., Musicaro, R., Suvak, M. K., Hamlin, E., et al. (2016). A randomized controlled study of neurofeedback for chronic PTSD. *PLOS ONE, 11*(12), e0166752. Retrieved from *https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0166752.*

Weidmann, G., Pauli, P., Dengler, W., Lutzenburger, W., Birbaumer, N., & Buckkremer, G. (1999). Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Archives of General Psychiatry, 56,* 78–84.

Wiemers, U. S., Sauvage, M. M., Schoofs, D., & Hamacher-Dang, T. C. (2013). What we remember from a stressful episode. *Psychoneuroendocrinology, 8*(10), 2268–2277.

Wyrwicka, W., & Sterman, M. B. (1968). Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiology and Behavior, 3,* 703–707.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18,* 459–482.

CHAPTER 10

Biofeedback Training to Increase Heart Rate Variability

Paul M. Lehrer

THEORETICAL FOUNDATIONS

For many reasons, heart rate variability is an index of health. Aspects of it reflect parasympathetic activity and relaxation. Other characteristics, perhaps even more importantly, reflect the operation of various regulatory reflexes throughout the body. Heart rate variability biofeedback (HRVB) is a method that easily produces huge acute increases in heart rate variability (HRV), and regular practice of HRVB has been found to increase resting HRV, reflex strength of two regulatory reflexes (the baroreflex and respiratory sinus arrhythmia), improve various psychological and physical stress symptoms, and improve human performance. Evidence for these effects will be reviewed below.

The number of studies published on HRVB has shown a logarithmic increase since the early 1990s, when the first studies were published. Reasons for this include the very wide range of applications for the method, the ease of learning it, and the inexpensive cost of equipment needed to do it. Probable reasons for its wide range of effects is that it targets a variety of "control systems" throughout the body, by which the body adapts to physical and psychological stressors and maintains allostatic balance.

To understand how HRVB works, it is first important to understand what a control system is. A system, first of all, is a set of characteristics made up of various elements that is separate and distinct from the elements that compose it. Thus the cardiovascular system is made up of numerous nerve, vascular, and blood cells, but one cannot describe the behavior of the whole cardiovascular system just by describing cellular action. A system is separate and distinct from its component parts. "Control systems" are systems that have built-in mechanisms for functioning at a particular level such that, when activity goes "out of bounds," the mechanisms return the system to its optimal level. Such systems are routinely built into many of the machines we use in daily life, including automobiles, airplanes, and others. The cardiovascular system is a control system. One of the hallmarks of all stable control systems—biological, psychological, or otherwise—is a complex pattern of oscillation (Giardino, Lehrer, & Feldman, 2000; Lehrer & Eddie, 2013). Our heart rate, blood pressure, body temperature, and energy level, along with our mood, social relatedness, task performance, and other biological and behavioral systems, are all constantly in motion and never function at a constant level. The pattern of variability can be complex. It varies over months, weeks, days, hours, and even seconds, often at many overlapping frequencies simultaneously.

For years, psychologists, biologists, and psychophysiologists had looked at these oscillations as bothersome sources of noise that obscure the individual's "true" level of activity—for example, of heart rate, mood, or blood pressure. We now know that these oscillations represent self-regulatory reflexes. When the body is well regulated, these oscillations have a complex pattern and are often at a relatively high amplitude. Reductions in either amplitude or complexity can be a sign of vulnerability, indicating that the self-regulatory mechanisms are damaged or inefficient and unable to withstand the vicissitudes of stress, disease, injury, and so forth. Thus, when a function rises, a regulatory reflex is triggered that makes it fall again, and when it falls, a complementary reflex makes it rise. This sequence causes a pattern of continuing oscillation. In biology, multiple oscillations often occur simultaneously at differing but overlapping rates. For heart rate, this yields a complex pattern of HRV, a pattern that gives us a window to the body's processes for self-regulation and homeostasis.

Another way to think about this complex pattern is to understand that each body function can be controlled by multiple control reflexes. One could think of these as multiple backup systems for homeostatic control. Thus the complexity and amplitude of the oscillatory pattern reflect the resilience and health of the system. The complexity can be described mathematically as "chaotic": that is, complex but completely deterministic when sufficiently complex simultaneous formulas are applied. Oscillation patterns that are very simple or completely random (i.e., not deterministic) suggest dysfunction. Goldberger and colleagues as well as others have demonstrated this with respect to HRV and cardiac function (Goldberger, 1991; Goldberger, Peng, & Lipsitz, 2002; Goldberger & Rigney, 1990; Goldberger, Rigney, Mietus, Antman, & Greenwald, 1988), in which low levels of complexity in HRV predict a variety of poor outcomes: a low rate of survival among patients with heart failure (Arzeno, Kearney, Eckberg, Nolan, & Poon, 2007; Guzzetti et al., 2000; Ho et al., 1997), among people requiring resuscitation after sepsis or succumb to it (Brown et al., 2013; Ellenby et al., 2001), and among fetuses in at-risk pregnancies (Schnettler, Goldberger, Ralston, & Costa, 2016). Low complexity in HRV also is associated with depression (Byun et al., 2019; Kwon et al., 2019), anxiety (De la Torre-Luque, Balle, Fiol-Veny, Llabres, & Bornas, 2018; Dimitriev, Saperova, & Dimitriev, 2016; Fiol-Veny, De la Torre-Luque, Balle, & Bornas, 2018), and stress (Brindle et al., 2016; Schubert et al., 2009; Visnovcova et al., 2014).

The *amplitude* of HRV also predicts health and resilience. Reduced HRV is associated with a wide variety of diseases. Cardiovascular disorders showing this pattern include hypertension (Mancia, Ludbrook, Ferrari, Gregorini, & Zanchetti, 1978), particularly in frequencies reflecting baroreflex (BR) activity and vagus nerve function. Low HRV amplitude also is characteristic of ventricular arrhythmia (Matveev & Prokopova, 2002; Rozen et al., 2013; Yi et al., 2014), presence and severity of ischemic heart disease (Eller, 2007; Gautier et al., 2007; Huikuri & Makikallio, 2001; Tacoy, Acikgoz, Kocaman, Ozdemir, & Cengel, 2010; Watanabe et al., 2013; Zdrenghea, Sitar-Taut, & Pop, 2007), and risk of rejection after heart transplant (Chou, Kao, Kuo, Liu, & Chan, 1998;

Izrailtyan et al., 2000). It also is related to risk for sudden cardiac death (Bigger, Fleiss, Rolnitzky, & Steinman, 1992; Christensen, Dyerberg, & Schmidt, 1999; Farrell et al., 1991; Franco et al., 2008; Loguidice, Schutt, Horton, Minei, & Keeley, 2016; Maheshwari et al., 2016; Rother et al., 1987; Kenneback, Ericson, Tomson, & Bergfeldt, 1997; Wu, Jiang, Li, & Shu, 2014). Low amplitudes of HRV are also characteristic of people with mood and anxiety disorders (Agelink, Boz, Ullrich, & Andrich, 2002; Brown et al., 2018; Chalmers, Heathers, Abbott, Kemp, & Quintana, 2016; Gorman & Sloan, 2000; Harrewijn et al., 2018; Kawachi, Sparrow, Vokonas, & Weiss, 1995; Kidwell & Ellenbroek, 2018; Kuang et al., 2017; Mullen, Faull, Jones, & Kingston, 2012; Shinba, 2017; Williams et al., 2017; Yeragani, Balon, Pohl, & Ramesh, 1995) and of people undergoing stress (Brindle, Ginty, Phillips, & Carroll, 2014). HRV amplitude is negatively correlated with age among adults (Liao et al., 1995; O'Brien, O'Hare, & Corrall, 1986).

It is positively related to aerobic capacity (Byrd, Reuther, McNamara, DeLucca, & Berg, 2015; Carter, Banister, & Blaber, 2003; Choi et al., 2006; Fuller-Rowell et al., 2013; Hedelin, Wiklund, Bjerle, & Henriksson-Larsen, 2000; Pardo et al., 2000; Ray, Pyne, & Gevirtz, 2017; Russoniello, Zhirnov, Pougatchev, & Gribkov, 2013). HRV amplitude increases among patients undertaking a program of cardiac rehabilitation that includes a relaxation and/or exercise intervention (Caruso et al., 2015; Fallavollita et al., 2016; Lucini et al., 2002; Routledge, Campbell, McFetridge-Durdle, & Bacon, 2010; van Dixhoorn & White, 2005) and among people undertaking a program of aerobic exercise (De Meersman, 1993; Hallman et al., 2017). Thus one can think of HRV as a general measure of adaptability. Diminished amplitude and complexity of HRV both are signs of decreased resilience and increased vulnerability to physical and emotional stress.

HRV, Respiratory Sinus Arrhythmia, Vagus Nerve Function, and Paradoxes of Autonomic Balance

Respiratory sinus arrhythmia (RSA) is the variation in heart rate that accompanies breathing. Heart rate (HR) increases during inhalation, when parasympathetic stimulation of the heart is blocked and decreases during exhalation, when parasympathetic stimulation of the heart increases. Because of these vagal effects, RSA is sometimes used as an index of parasympathetic tone (Porges, 1995).

There are various ways to measure RSA. Respiratory-linked variations in HR usually occur in the frequency range of 0.15–0.4 Hz (9–24 cycles per minute) in the healthy human adult, the range of normal adult respiration rate. HR oscillations in this range are often referred to as *high-frequency* (HF) HR oscillations, and the spectral power within this range is often treated as synonymous with RSA and thus is frequently used as an index of *vagal tone* because it reflects RSA amplitude. This is a *frequency-domain* method of calculating HRV, which involves doing a spectral analysis, usually by means of a Fourier transformation, and looking at this specific frequency band. Because frequency-domain measures are complicated to assess, require longer measurement time, and assume *stationarity* (i.e., lack of change in oscillation frequency) during the measurement period, some *time-domain* measures also are used to assess RSA that do not have these limitations. One is simply average peak-to-trough amplitude in HR oscillations. Other measures look at amount of change in adjacent interbeat intervals, which usually reflects the fastest changing characteristics of HRV, a marker of RSA. These include the root mean square of successive differences (RMSSD), defined as the average squared difference in adjacent cardiac interbeat intervals, and the percentage of adjacent normal interbeat intervals that exceed 50 milliseconds in time (pnn50).

In describing various measures of HRV, the word *normal* connotes beats that are under neural control, excluding arrhythmic beats such as preventricular or preatrial contractions. These abnormalities must be edited out of all HRV calculations, usually by interpolation or filtering. For a discussion of methods for doing this, see Peltola (2012).

Cardiac vagal tone is often seen as an index of health. It decreases during periods of inflammation (Dennis et al., 2016; Rupprecht et al., 2019; Williams et al., 2019), although nonvagal influences on HRV also are depressed in acute experimentally induced inflammation, indicating complete autonomic withdrawal (Alamili, Rosenberg, & Gogenur, 2015; Lehrer et al., 2010). It is diminished in heart disease (Azuaje et al., 1999), hypertension (Drummond, 1990; Grossman, Brinkman, & de Vries, 1992; Masi, Hawkley, Rickett, & Cacioppo, 2007; Xie et al., 2018), diabetes (Masi et al., 2007), and pain (Barakat et al., 2012; Sturgeon, Yeung, & Zautra, 2014; Zamuner et al., 2016). It also has been found to be related to developmental maturity in preterm infants (Clark et al., 2012). It is inversely related to mortality in heart disease, to presence of cardiac arrhythmias (Dreifus et al., 1993; Sinnecker et al., 2016), and to fetal and sudden infant death (Hon & Lee, 1965; Rudolph, Vallbona, & Desmond, 1965). In an early study, we found decreased HRV among alcoholics (Lehrer & Taylor, 1974), perhaps reflecting cardiac malfunction. Decreased HRV in diabetes also was recognized quite early (Morguet & Springer, 1981).

RSA also decreases in emotional disorders, including depression (Austen & Wilson, 2001; Borelli, Smiley, Rasmussen, Gomez, Seaman, & Nurmi, 2017; Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014; Kovacs et al., 2017), anxiety (Licht, de Geus, van Dyck, & Penninx, 2009; Viana et al., 2017), and posttraumatic stress (Dennis et al., 2016; Hopper, Spinazzola, Simpson, & van der Kolk, 2006). RSA also is positively correlated with social relatedness (Gouin, Zhou, & Fitzpatrick, 2015; Grippo, Lamb, Carter, & Porges, 2007; Holt-Lunstad, Uchino, Smith, & Hicks, 2007; Kolacz, 2017), and is negatively related to autism and autistic behavior (Condy, Scarpa, & Friedman, 2019; McCormick et al., 2018; Patriquin, Hartwig, Friedman, Porges, & Scarpa, 2019).

However, elevated parasympathetic activity is not always a sign of health, and stress does not always depress parasympathetic activity. For example, in vasovagal syncope, which can be stress-induced, patients show elevated parasympathetic tuning (Piccirillo et al., 2004) but are sick and maladaptive nonetheless. This response tends to be characteristic in some individuals (Longin et al., 2008) and can be more generally elicited by passive stress coping (Engel, 1978; Lehrer et al., 1996), such as in the face of traumatic or life-threatening stress for which no active coping response is available (Schauer & Elbert, 2010). Similarly, HR tends to be depressed in nonassertive women faced with an assertion challenge (Lehrer & Leiblum, 1981) and in cognitively impaired patients with closed head injury when exposed to cognitively demanding tasks (Lehrer, Groveman, Randolph, Miller, & Pollack, 1989). Elevated vagal activity (including RSA) also is characteristic of people with chronic fatigue syndrome (Giardino, 2003) and among people with blood phobia (Angrilli, Sarlo, Palomba, Schincaglia, & Stegagno, 1997) when they are exposed to blood. People with blood phobia also have an attenuated sympathetic response when exposed to blood stimuli (Sarlo, Palomba, Angrilli, & Stegagno, 2002). Elevated parasympathetic responsiveness also occurs in asthma patients exposed to various stressors (Aboussafy, Campbell, Lavoie, Aboud, & Ditto, 2005; Miller, Wood, Lim, Ballow, & Hsu, 2009; Miller & Wood, 1994, 1997) and among defensive asthma patients, healthy individuals who tend to respond with vagal inhibition or sympathetic arousal (Feldman, Lehrer, Hochron, & Schwartz, 2002).

To understand these apparent paradoxes, it is important to think of autonomic balance as part of the body's "yin and yang" system of resilience and to appreciate the complexity of how the body controls itself. While the body mobilizes for fight-or-flight activity, for concentrative work, or for increased alertness, it simultaneously prepares systems to modulate these responses to prevent autonomic overload, autoimmune reactions, or fatigue of the very circuits that are simultaneously mobilizing. Thus increased sympathetic arousal automatically increases parasympathetic reactivity, which can produce fatigue, hunger, bronchoconstriction, and other parasympathetic reactions soon after the mobilization subsides, in "parasympathetic rebound" reactions (see Lehrer, Chapter 2, this volume, and Gellhorn & Loofbourrow, 1963). This reflex is so powerful that it sometimes can overwhelm the mobilization response itself and cause the parasympathetic stress responses we just described. In the immune system, we often see stress-induced anti-inflammatory processes, such as corticosteroid and anti-inflammatory cytokine production, just as the body is mobilizing the immune and inflammatory systems to resist invasion of foreign microbes from effects of anticipated injury (see Lehrer, Chapter 2, and Kusnekov, Norton, & Nissenbaum, Chapter 4, this volume). "Anticipation" is an important part of this equation, since psychological stressors rarely produce the kind of metabolic mobilization caused by a genuine physical fight or flight. Anticipation is the foundation of conditioning and learning (Balsam et al., 2009; Stepp, 2012). Patterns of learning and conditioning, as well as constitutional factors, will determine the relative strength of the mobilization versus modulatory tuning of any individual's response to stress, and allostatic overload could cause symptoms of stress-induced dysregulation, with both parasympathetic (and sympathetic) overactivity, as well as underactivity.

Other Measures of HRV

Other frequencies of HRV also reflect various allostatic functions. The *low-frequency* (LF) band of HR oscillations, between 0.05 and 0.15 Hz (between 0.3 and 9 cycles per minute), is heavily influenced by the cardiac portion of the BR (Rahman, Pechnik, Gross, Sewell, & Goldstein, 2011). This is discussed later in the chapter. This band, like the HF band, also seems to be primarily under control of the vagus nerve (Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013). Less frequently, researchers also look at the *very-low-frequency* (VLF) band, which is under alpha sympathetic control (Leor-Librach, Eliash, Kaplinsky, & Bobrovsky, 2003) and is influenced by the vascular tone component in BR (Leor-Librach, Bobrovsky, Eliash, & Kaplinsky, 2002; Vaschillo, Vaschillo, Buckman, Pandina, & Bates, 2012) and appears to play a role in thermal regulation (Thayer, Nabors-Oberg, & Sollers, 1997). This band is difficult to assess because it requires very long measurement periods, within which stationarity can rarely be achieved.

Time-domain measures of vagus nerve function have been discussed above. Another time-domain measure that is frequently used is the standard deviation of normal cardiac interbeat intervals (SDNN). This measure is useful because it combines all frequencies in HRV and thus may be considered to be a general measure of all control systems affecting the heart and, thus, an overall measure of resilience. For a complete discussion and the various measures of HRV, norms, interpretation, and editing procedures, see Shaffer and Ginsberg (2017).

Complexity of the Vagus Nerve, Autonomic Regulation, and the BR

Although RSA is vagally controlled and is sometimes used as a measure of cardiac vagal tone, RSA is not completely synonymous with cardiac vagal tone. Porges (2011) has described this complexity and ascribed two different functions to specific fibers in the vagus nerve. Myelinated fibers, controlled through nucleus tractus solitarius (NTS) in the brainstem, carry patterns of oscillatory activity, including RSA. These fibers and this pattern are present only in warm-blooded animals whose body temperature and metabolism are under internal control, and thus they represent an important autonomic control system. RSA, by this pathway, also appears to be characteristic of social animals, such as humans, dogs, and horses, but is small or absent in relatively nonsocial animals such as cats.

Unmyelinated vagal fibers, on the other hand, are controlled through the dorsal motor nucleus. They do not have regulatory functions and do not carry RSA patterns. These fibers produce changes in tonic parasympathetic functions such as mean HR or blood pressure and, in dysregulated states, are responsible for large swings in parasympathetic function, as may occur in stress-induced syncope. These two vagal functions often, but not always, work together. Decoupling of the two functions happens frequently and is a particular characteristic of HRVB. When doing breathing maneuvers such as those associated with HRVB, large increases in the amplitude of RSA routinely occur with little or no change in mean HR (Lehrer et al., 2003; Sargunaraj et al., 1996; Song & Lehrer, 2003). From this we conclude that HRVB specifically affects the regulatory functions of vagus nerve activity.

Another important regulatory function of the vagus nerve derives from its interaction with the immune system. A large body of evidence shows that increased vagus nerve activity decreases inflammatory activity (Tracey, 2002). Robust findings have been found for electrical stimulation of the vagus nerve (Pavlov & Tracey, 2012). HRVB has been found to reduce airway inflammation in asthma (Lehrer et al., 2018), although, in another study, HRVB did not reduce inflammation caused by experimental injection of an inflammatory cytokine (Lehrer et al., 2010), although it did modulate the decrease in HRV caused by this exposure (Lehrer et al., 2018).

It also is important to keep in mind that vagal tone and RSA are not synonymous with HF HRV. People can breathe more quickly or more slowly than 9–24 breaths per minute (the HF range), in which case RSA occurs at actual respiration rate, which may be outside the boundaries of the HF range. This happens systematically while doing HRVB, where people breathe at a rate closer to six breaths per minute. Also, some influences independent of vagal traffic can cause RSA to vary. One such influence is the rate of acetylcholine hydrolyzation (decomposition). Acetylcholine is released by vagus nerve activity during exhalation (causing RSA), but it may be hydrolized more fully during slow rather than fast respiration, in which case the effects of inhalation may diminish the effect of vagus nerve output on HRV (Taylor, Myers, Halliwill, Seidel, & Eckberg, 2001). Thus larger amplitudes of RSA during slower rates of breathing may not necessarily reflect differential quantities of vagus nerve traffic. Also, under some circumstances, sympathetic activity may even affect HF HRV (Pyetan & Akselrod, 2003; Taylor et al., 2001). Indeed, the imputation of vagal tone from RSA is complicated. At the very least, it is important to consider respiration rate while evaluating HRV as a measure of autonomic function. If increases in RSA occur independently of respiration rate and are coupled with decreases in HR, then one can confidently interpret these effects as reflecting increased overall vagal tone.

HRV in the LF range (0.05–0.15 Hz) has a more complex relationship with autonomic activity. LF HRV primarily reflects activity of the BR (Moak et al., 2007; Robbe et al., 1987). The BR helps control blood pressure swings, such that it causes HR to fall as blood pressure rises and rise as blood pressure falls. This loop of the BR is often called the "vagal BR," because most LF activity is controlled by the vagus nerve. It occurs at a rhythm of approximately 0.1 Hz (10 second cycles; Vaschillo, Lehrer, Rishe, &

Konstantinov, 2002), resulting in the well-known "Meyer wave" at this frequency in almost all HR assessments. Although LF HRV is mostly vagal (Elghozi & Julien, 2007; Houle & Billman, 1999), portions of it at the low end of the frequency spectrum may have some sympathetic influence (Berntson et al., 1997; Pyetan & Akselrod, 2003; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), though this remains controversial. Some investigators have used the LF:HF ratio as a measure of autonomic balance, although this use does not appear to be valid, particularly in the supine position (Eckberg, 2000; Moak et al., 2007; Myers, Cohen, Eckberg, & Taylor, 2001). Nevertheless, the LF:HF ratio has periodically been found to be higher in anxiety (Chang et al., 2019; Choi et al., 2019; Traina, Cataldo, Galullo, & Russo, 2011), depression (Choi et al., 2019; Wang et al., 2013; Yeh et al., 2016), and heart disease (Watanabe et al., 2013).

RSA and Respiratory Efficiency

Yasuma and Hayano (2004) have shown that RSA promotes respiratory efficiency by making more blood available when oxygen concentration in the alveoli is at a maximum, that is, during inhalation. Respiratory efficiency increases when people breathe at about six breaths per minute (Bernardi et al., 1998; Bernardi, Gabutti, Porta, & Spicuzza, 2001; Spicuzza, Gabutti, Porta, Montano, & Bernardi, 2000; Giardino, Chan, & Borson, 2004). It is only at this frequency that RSA is completely in phase with breathing, so that HR rises simultaneously with inhalation and falls simultaneously with exhalation (Vaschillo, Vaschillo, & Lehrer, 2004). Thus, when people breathe at this rate, HR is highest when the oxygen concentration in the alveoli is highest (during inhalation), thus causing increased gas exchange efficiency. Bernardi and his colleagues have observed that yogis also tend to breathe at approximately this same frequency. Breathing at this rate is accompanied by a decreased hypoxic ventilatory response, along with better oxygen saturation during the experimental challenge and better tolerance for exercise and high altitude, with some advantage to people who have engaged in long-term practice of yoga (Bernardi, Passino, et al., 2001; Spicuzza et al., 2000). Saying the rosary prayers and yogic meditation are both associated with breathing at approximately six times per minute and produce predictably large increases in HRV at that frequency (Bernardi, Sleight, et al., 2001).

Vaschillo et al. (2004) have shown that HRVB encourages the individual to breathe at the frequency at which HRV and respiration are exactly in phase with each other, a frequency that is usually near six breaths per minute, although often not exactly at this rate. HRVB thus should maximize respiratory efficiency. My own recent clinical observation of phase relationships between HRV and phase relationships between HR and breathing cast some doubt on this general observation. We therefore reanalyzed HRVB data from 26 adult patients with asthma and calculated phase among people with coherence > 0.8 between HR and breathing, indicating that they were breathing at a constant rate that should stimulate high amplitudes of HRV. The phase relationship between breathing and HR oscillations was 75°, which is only a little closer to "in phase" relationship (0°) than during normal breathing, where RSA usually oscillates with a 90° phase relationship with breathing and the beginning of inhalation occurs in the middle of each increase in HR. Higher phase angles tended to occur among older people, with close to 0° relationships occurring only among young adults, such as those studied in the early observations of Vaschillo et al. (2008). The meaning of this differential effect in some individuals is unclear. Perhaps it reflects changes in vascular flexibility with aging and consequent speed of HR-induced blood pressure changes.

Nevertheless, the fact that amplitude of RSA greatly increases during HRVB indicates that respiratory gas exchange is also expected to increase, with expected salutary effects on respiratory disease and possibly on respiratory drive and dysfunctional breathing. There is thus some reason to think that HRVB may help inoculate people against hyperventilation, a common pattern among people with panic symptoms and various psychosomatic disorders (Bass, 1997; Hegel & Ferguson, 1997; Studer, Danuser, Hildebrandt, Arial, & Gomez, 2011; Van Diest et al., 2005), just as Bernardi's data (cited above) show that people have a decreased hypotoxic ventiliatory response when they breathe at a frequency close to that taught in HRVB. Whether age of the individuals may modify these effects remains a topic for future research. In our own clinical work, we have found that the HRVB method, when it includes coaching in breathing shallowly, increases end-tidal CO_2 and stops or prevents panic symptoms both when challenged by deliberate hyperventilation and when the hyperventilation occurs spontaneously. These effects are similar to those found by Ritz and colleagues in a study of end-tidal CO_2 biofeedback among asthma patients, where CO_2 levels rose, anxiety decreased, and respiration rate fell to levels close to those associated with resonance frequency breathing (Ritz, Meuret, Wilhelm, & Roth, 2009; see also Meuret & Ritz, Chapter 11, this volume). Caution should be used in training, however, because paced breathing at any rate, as is done in HRVB, tends to produce a compensatory increase in ventilation and decrease in pCO_2 unless people are taught to breathe more shallowly than is apparently instinctual during paced breathing (Kox, Pompe, van der Hoeven, Hoedemaekers, & Pickkers, 2011; Lehrer et al., 1997; Pinna, Maestri, La Rovere, Gobbi, & Fanfulla, 2006; Szulczewski & Rynkiewicz, 2018). Interestingly, however, breathing at or near resonance frequency tends to prevent increased ventilation in response to various respiratory challenges (Bernardi, Schneider, Pomidori, Paolucci, & Cogo, 2006; Critchley et al., 2015).

The BRs and LF HRV

The BRs are important mechanisms for control of blood pressure (Eckberg & Sleight, 1992). The BRs are triggered from stretch receptors located in the aortic arch and carotid sinus ("baroreceptors") that are responsive to changes in blood pressure. When blood pressure increases, the BRs trigger decreases in HR and in vascular tone, thus producing subsequent mechanical homeostatic decreases in blood pressure after a delay caused by mechanics of the circulatory system. When blood pressure decreases, the BRs produce the opposite effects. Because of inertia in the blood and the varying effects of arterial compliance, a delay occurs between changes in HR and the mechanically induced changes in blood pressure that follow (because of changes in blood volume flowing through the relatively constant-sized vessels). This delay is close to 5 seconds for changes in HR, thus accounting for the approximately 10-second rhythm in HRV, by far the largest contributor to LF HRV. Because changes in vascular tone occur more slowly than changes in HR, the vascular tone component of the BR, mediated through the alpha sympathetic system, produces a slower rhythm, usually in the VLF component of HRV (Eckberg & Kuusela, 2005; Vaschillo et al., 2002; Vaschillo et al., 2018; Vaschillo et al., 2012). Autonomic effects on vascular tone and, therefore, the VLF component in HRV are mediated through the alpha sympathetic system (Crawford-Achour et al., 2016; Elghozi & Julien, 2007).

The strength or gain of the BRs is measured in milliseconds of change in cardiac interval, measured from the R-spike in the cardiogram, and therefore called the R–R interval (RRI) of the electrocardiogram (in milliseconds), per millimeters of mercury change in blood pressure (mm Hg). BR gain can be calculated by assessing the slope of increases in RRI against increases in systolic blood pressure and of decreases in RRI against decreases in blood pressure and by cross-spectral analysis of RRI and blood pressure. The last of these has been found to correlate highly with BR gain calculated from phenylephrine-induced blood pressure increases and with suction on the neck, which directly stimulates the carotid BR (James, Panerai, & Potter, 1998; Rudas et al., 1999). The cross-spectral and slope methods are particularly useful because they assess both aortic and carotid BRs, without the unacceptable side effects often produced by phenylephrine.

Hypertension is often accompanied by BR dysfunction (Eckberg, 1979; Freitas et al., 2017; Guo & Abboud, 1984; Laitinen, Hartikainen, Niskanen, Geelen, & Länsimies, 1999; Matsuguchi & Schmid, 1982; Pitzalis et al., 1999; Rau et al., 1994; Rau, Furedy, & Elbert, 1996; Robertson et al., 1993), as are anxiety and depression (Davydov, Shapiro, Cook, & Goldstein, 2007; Virtanen et al., 2003; Henze et al., 2008; Watkins, Grossman, Krishnan, & Blumenthal, 1999). BR gain also is related to adaptiveness of the cardiovascular system—indeed, to survival. La Rovere, Bigger, Marcus, Mortara, and Schwartz (1998) showed that, among patients recovering from myocardial infarction, those with subnormal BR gain have high risk of fatal cardiac events, especially if the patient also has low HRV. The linkage between BR impairment and mortality may partially reflect patients' autonomic responses to cardiac rhythm changes. Consistent with this, arterial perfusion pressures during ventricular tachycardia recover more rapidly in patients with stronger BRs (Hamdan et al., 1999). Ventricular tachycardia is a rapid rhythm that commonly precedes sudden death (Bayes de Luna, Coumel, & Leclercq, 1989), precipitously lowers arterial pressure, and increases muscle–sympathetic nerve activity (Smith, Ellenbogen, Beightol, & Eckberg, 1991), and reduces vagal–cardiac nerve activity (Huikuri et al., 1989). In an exercise-ischemia dog model of sudden cardiac death, ventricular fibrillation occurs when BRs are weak but do not occur when they are strong (Billman, Schwartz, & Stone, 1983).

Nerve stimulators have been used with some success to stimulate the BR as a treatment for hypertension, although with insufficient efficacy to be accepted as standard practice (Ewen, Bohm, & Mahfoud, 2017; Gordin et al., 2016). Because HRVB systematically stimulates the BR and increases BR gain acutely and, in some studies, chronically (Lehrer et al., 2003; Lin et al., 2012), it stands to reason that this completely noninvasive method should be adequately investigated as an alternative method.

Resonance in the System and a BR Mechanism for HRVB

The cardiovascular system is known to have resonance characteristics, with a first resonance frequency of ~0.1 Hz (DeBoer, Karemaker, & Strackee, 1987; Vaschillo et al., 2002). Characteristics of resonance include very-high-amplitude oscillations at a single frequency, eclipsing oscillations at all other frequencies, particularly when the system is stimulated by a rhythmical perturbation at the resonance frequency. The resonance characteristics of HRV appear to reflect BR influences (Vaschillo et al., 2002; Vaschillo, Vaschillo, & Lehrer, 2006). HRVB always influences the individual to breathe at the individual's specific BR-determined resonance HRV frequency, thus rhythmically perturbing the BR system at its resonance frequency, thus producing very-high-amplitude oscillations at this single frequency.

In their early research on resonance in HRV, Vaschillo and colleagues found that the phase relationship between HR and blood pressure oscillations at the resonance frequency (and *only* at this frequency) is exactly 180° (oscillating in opposite directions at the same frequency), whereas the phase relationship between HR oscillations and respiration is 0° (oscillating in phase with each other; Vaschillo et al., 2002; Vaschillo, Zingerman, Konstantinov, & Menitsky, 1983) with inhalation coinciding with HR accelerations. Thus

RSA and the BR stimulate HRV *in the same direction* at resonance frequency. Therefore, when breathing at resonance frequency, just as RSA causes HR to rise, the simultaneous decrease in blood pressure also causes HR to rise by BR action, with the opposite occurring during exhalation, causing a "push" to the system with every inhalation and every exhalation. The highest amplitudes of biofeedback-produced HRV were achieved when people breathed at this frequency. The 75° phase angle found in our more recent research still allows HRV to be stimulated rhythmically at the resonance frequency of the BR, although the breathing-induced perturbation in HR now occurs in the middle of a BR-induced oscillation rather than at the beginning of it (Lehrer, Vaschillo, & Vidali, 2020). Thus, when people breathe at their resonance frequency, whether in or out of phase, the amplitude of HRV is maximized, and the BR is greatly stimulated. Conversely, when people try to maximize the amplitude of their HRV using biofeedback, they automatically breathe at their resonance frequency and thus also stimulate the BR.

Another characteristic of resonance is that oscillations at the resonance frequency persist after initial stimulation. Imagine striking an object without resonance characteristics (e.g., a tree trunk). What happens? *Thunk:* an auditory event that immediately disappears. Then imagine striking an object with resonance, such as a bell. You then produce a sound with a long decay: bo-o-o-o-o-o-ng. The oscillation is big at first and gradually decays. When a resonant system is *rhythmically* stimulated at its resonance frequency, the external stimulation magnifies the persistent oscillations, thus greatly increasing total variability. Imagine pushing a swing at its resonance frequency. If you push each time the swing starts going up (i.e., at its resonance frequency), the oscillations in the swing do not just persist and decay; they grow in amplitude. This occurs even if we push the swing in the middle of its course through the air (close to a 75° phase) rather than at the apex of the swing movement (0° phase), it still stimulates the swing to go higher, as happens among older people in HRVB. Such is the case with HRV and breathing, where HRV is the swing and breathing is the push.

Thus breathing at resonance frequency of the BR system produces a very high amplitude in HRV. Even breathing *close* to resonance frequency produces a higher spectral peak at respiratory frequency, because this also is a characteristic of resonance. However, the spectral peak at the respiratory frequency is highest (reflecting a higher oscillation amplitude) at the *exact* resonance frequency (see Figure 10.1).

Our research has found that resonance frequency differs among individuals, between 4.5 and 7 cycles/minute among healthy adults, replicating previous research by Vaschillo and colleagues (Vaschillo et al., 1983; Vaschillo et al., 2002; Vaschillo et al., 2006). Each individual's resonance frequency is related to gender (higher in women) and, independently, inversely to height, but not to age or weight, suggesting that it may be related to the volume of blood in the vasculature, such that inertia will cause a greater delay in blood pressure changes after changes in HR when the mass of the blood supply is greater, thus producing slower oscillations in HR.

Effects of HRVB on the BR

Regular training of a reflex may be expected to produce chronic increases in reflex efficiency. This has been particularly well demonstrated for musculoskeletal reflexes (Bawa, 2002; Schalow, Blanc, Jeltsch, & Zach, 1996), and the process also has been demonstrated in the cardiovascular system (Zhao, Hintze, & Kaley, 1996). In HRVB, participants are instructed to practice biofeedback for several periods each day, so BR gain is greatly amplified on a regular basis by biofeedback practice. Data from our laboratory (Lehrer et al., 2003) show that this procedure produces *chronic* increases in BR gain

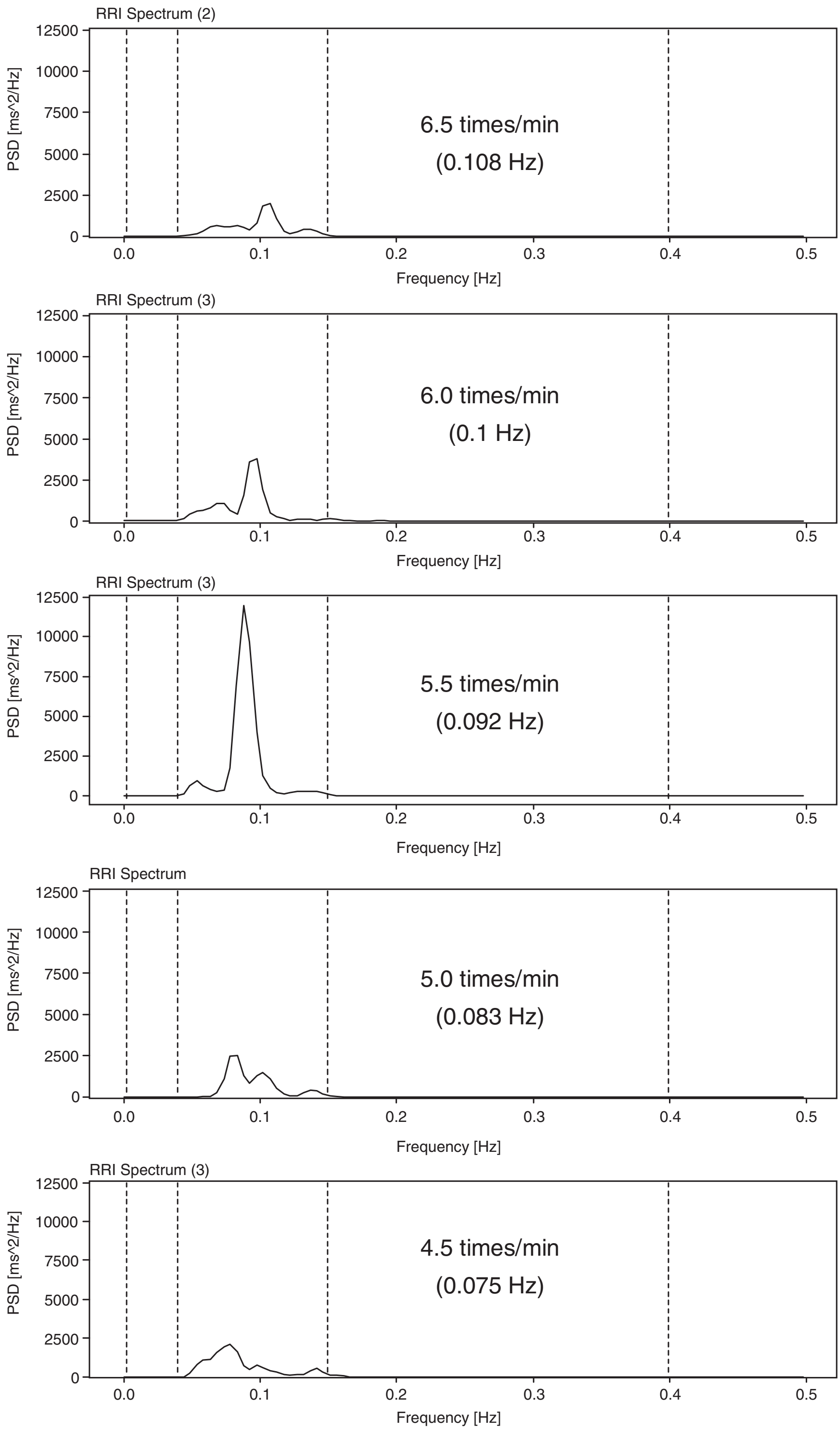


FIGURE 10.1. HRV spectra while breathing at various frequencies. From Vaschillo et al. (2006). Note that the spectral peak is highest at resonance frequency, which is 5.5/min for this individual.

among healthy people *at rest,* apparently reflecting greater BR efficiency resulting from exercising of the BRs from high-amplitude HR and blood pressure oscillations during frequent resonance frequency breathing.

In a study of the method among healthy individuals (Lehrer et al., 2003), we found that BR gain was significantly increased, both acutely and chronically, and that LF HRV (0.05–0.15 Hz), in which BR effects are most clearly observed (Robbe et al., 1987), is acutely effectively doubled. This has been replicated several times (Fonoberova et al., 2014; Lin et al., 2012; Nolan et al., 2012). Additionally, Vaschillo et al. (2002) showed that particularly great increases in BR gain occur at the specific frequency that reflects BR activity in each individual. In a clinical application of this, slow breathing, such as occurs during HRVB, also has been shown to increase BR gain and sensitivity among patients with chronic heart failure (Bernardi et al., 2002). HRVB also has been found to increase HRV in this population (Del Pozo, Gevirtz, & Guanieri, 2004).

Other Pathways for Resonance-Frequency Cardiovascular Stimulation

Note that resonance characteristics of HRV and the BR system can be stimulated by *any* repetitive activity or event that stimulates the system at its resonance frequency. In addition to breathing, the system can be stimulated by repetitive muscle tension-release sequences at resonance frequency (Lehrer, Vaschillo, Trost, & France, 2009), by repetitive exposure to emotional stimuli (Vaschillo et al., 2008), by repeated tasks (see Figure 10.2),

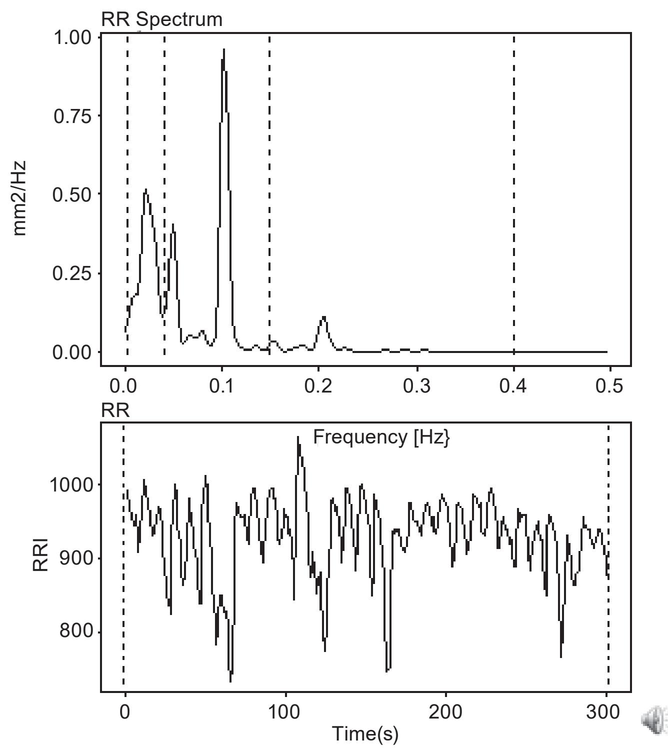


FIGURE 10.2. Heart rate variability in airline pilot performing simple maneuvers every 10 seconds in a flight simulator.

and others. The increases in RSA produced by repetitive muscle tension at resonance frequency presents some interesting characteristics for autonomic control. Because the skeletal muscles are controlled by the *sympathetic* system, this maneuver may not only stimulate BR control but may also tune the sympathetic system to facilitate BR activity. This may be particularly important for people with vasovagal overactivity, as in vasovagal syncope (Miranda & Silva, 2016; Piccirillo et al., 2004; Salameh et al., 2007; Vershuta et al., 2009), in which increased sympathetic arousal may decrease unmyelinated vagal activity and thus prevent fainting.

Also, we should note that the BR system has more than one resonance frequency in each individual. In addition to its effect on HR, as mentioned above, the BR system also affects vascular tone, such that increases in blood pressure produce vasodilation, whereas decreases in blood pressure produce vasoconstriction. This activity produces oscillations in the cardiovascular system within the VLF range, close to 2–3 cycles per minute (Hammer & Saul, 2005; Vaschillo et al., 2002; Vaschillo, Vaschillo, Buckman, Pandina, & Bates, 2011). This oscillation presumably has resonance characteristics that can also be stimulated by repetitive activity, thus strengthening the vascular tone component of the BR. This activity is also presumably mediated by the alpha sympathetic system, which is the main mechanism for autonomic control of vascular tone (Lundvall & Edfeldt, 1994; Panza, Epstein, & Quyyumi, 1991; Rodionov, Koshelev, Meshcheryakova, & Stavskaya, 1981). We do not know the effects of this kind of stimulation on various disorders, or on the human condition, but the effects of poor modulation of vascular tone on blood pressure are well known, so the possibilities of new forms of resonance frequency cardiovascular stimulation are worth exploring.

Other Possible Effects of HRVB

In a clinical trial of HRVB as a treatment for asthma, an interesting pattern of results emerged. We studied 97 patients with asthma, approximately half of whom were given training in HRVB. The rest were divided into two control conditions: frontal EEG alpha biofeedback and a waiting-list condition (Lehrer et al., 2004). We titrated asthma controller medications (primarily inhaled steroids) based on patients' asthma symptoms and pulmonary function. We found that, compared with the control groups, participants given HRVB were able to decrease their consumption of controller medication to a clinically significant level while maintaining, or improving, their clinical condition (i.e., fewer asthma exacerbations, decreased respiratory resistance, greater compliance of airway tissue, less inhomogeneity of resistance in the airways, and fewer asthma symptoms). However, none of these results correlated with changes in HRV, BR gain, or any other autonomic measures we studied. Additionally, although the cardiovascular effects of HRVB were greater among younger people, the effects on respiratory resistance were greater in older people (Lehrer et al., 2006).

So what mediated the biofeedback effects on asthma? Now we can only guess. Our forced oscillation pneumography findings suggested that tissues in the airways became more compliant, with less inhomogeneity in airway resistance among the various airway passages (Dubois, Brody, Lewis, & Burgess, 1956). These results suggest a decrease in airway inflammation, which causes generalized stiffness in the airways but tends to affect various air passages differentially. In a subsequent study (Lehrer et al., 2018), we found that HRVB did indeed decrease airway inflammation in asthma, as measured by exhaled nitric oxide. There is corroborating evidence that HRVB may decrease inflammation. There is a study showing an anti-inflammatory effect on C-reactive proteins among patients with hypertension (Nolan et al., 2012). The effects on inflammation should not be surprising, given the known anti-inflammatory effects of vagus nerve activity (Tracey, 2002).

Another possible mechanism for asthma improvement may be an indirect effect of improved respiratory gas exchange efficiency, as described earlier. There is some link between poor gas exchange and asthma, particularly during acute attacks (Rodriguez-Roisin, 1990; Young & Bye, 2011), one could speculate that muscular tone in the airways may be related to the amount of effort required to breathe and maintain normal oxygenation. HRVB may strengthen respiratory muscles, as shown by increases in peak expiratory flow among healthy people after HRVB (Lehrer et al., 2003). In these people, the airways are not impaired; so airway resistance should be minimal. Therefore, improvement in peak flow probably reflected increased strength of the respiratory muscles rather than changes in airway patency.

In another study, we assessed the effects of HRVB among healthy study participants who were administered lipopolysaccharide, a potent inflammatory endotoxin (Lehrer et al., 2010). Although we found no effects on various inflammatory markers, we found a marked increase in HRV, which had been severely depressed by the endotoxin, and a decrease in flu-like symptoms. Thus, although HRVB may not directly decrease all kinds of inflammation, it may decrease symptoms and impaired autonomic adaptability associated with it.

HRVB versus Paced Breathing without Biofeedback

There is some evidence for a larger therapeutic, as well as physiological, effect of breathing at exact resonance frequency as determined through biofeedback compared with standard instructions to breathe six times a minute (Lin et al., 2012; Steffen, Austin, DeBarros, & Brown, 2017). Nevertheless, a number of studies have found significant clinical effects for paced breathing at rates similar to those characteristic of typical resonance frequencies (Botha et al., 2015; Carpenter et al., 2013; Lin et al., 2012; Stromberg, Russell, & Carlson, 2015; Wahbeh, Goodrich, Goy, & Oken, 2016). More research is needed to determine the clinical importance of breathing at the exact resonance frequency. An interesting pilot study comparing paced breathing at resonance frequency determined by our protocol, as detailed below, with resonance frequency determined by a more exact procedure, found a small increase in resulting HRV when people were alternately instructed to breathe at the resonance frequencies determined by each of these methods (Fisher, 2020).

Brain Mechanisms for HRVB Effects

As described above, HRVB has large and direct effects on strengthening the BR and hence modulating autonomic reactivity, while also strengthening respiratory control reflexes. However, as shown in Table 10.1 and discussed below, HRVB biofeedback also appears to have salutary effects on modulation of emotion. The mechanism for this may be on the close connection between the BRs and brain structures that generate and modulate emotion (Henderson et al., 2004; Mather & Thayer, 2018). The BRs are known to be mediated through the NTS in the brainstem. The NTS is both anatomically and physiologically closely connected to the insula and amygdala, which are involved in control of emotion. Some functional magnetic resonance imaging (fMRI) data now are available showing very large 6/min oscillations in various limbic structures, as well as cortical modulatory structures, and modulatory pathways when people breathe at that frequency in the fMRI (Vaschillo, Vaschillo, Buckman, & Bates, 2019). Mather (2019) has also reported increased activity in brain structures that modulate and control emotion and mediate increases in RSA, including the left insula and medial prefrontal orbital cortex, where study participants were at rest after five weeks of training.

Additional evidence for HRVB effects on the brain come from studies of HRVB effects on cardiac evoked potentials. Heartbeats are known to produce evoked potentials (Schandry, Sparrer, & Weitkunat, 1986; Pollatos, Kirsch, & Schandry, 2005; Gray et al., 2007). There is evidence that breathing close to resonance frequency increases the amplitude of these potentials (Huang, Gevirtz, Onton, & Criado, 2014; MacKinnon, Gevirtz, McCraty, & Brown, 2013). The pathway could be through BR effects, or through effects on the brain from vagal afferents affected by biofeedback through subdiaphragmatic pathways (Brown & Gerbarg, 2005; Porges, 2011).

In addition to salutary effects on emotional conditions such as stress reactions, anxiety, substance craving, depression, and anger, a listing of controlled trials in Table 10.1 shows that HRVB also appears to enhance neuromuscular performance, as evidenced in athletic and dance performance. It lessens pain, improves sleep, may decrease pregnancy complications, lessen symptoms of functional or stress-related gastrointestinal disorders, and improve symptoms and quality of life for patients with heart failure. An uncontrolled study of patients with chronic obstructive pulmonary disease showed improvement in symptoms, the 6-minute walk distance test, and quality of life (Giardino et al., 2004), perhaps due to phase relationships between breathing and heart rate, such that blood flow to the lung is maximal when heart rate is highest, thus maximizing efficiency of gas exchange. Greater availability of oxygen may also contribute to enhanced muscular and brain function.

HISTORY OF THE METHOD

The first documented studies of HRVB were reported in a Russian report by Vaschillo et al. (1983). They initially used the technique to assess autonomic function and found that biofeedback could easily be used to teach cosmonauts to produce high amplitudes of HRV and blood pressure variability at various frequencies. They then performed transfer function analyses in order to examine specific frequency characteristics of biofeedback-induced HRV and found that participants uniformly produced the highest frequency-specific HRV oscillations only at certain frequencies, usually at about 0.1 Hz (10-second cycles). By examining the phase relationships between HR and blood pressure oscillations, the researchers discovered the relationships among BR activity, HRVB, and resonance characteristics in the cardiovascular system, described earlier. This work was later reported in English (Vaschillo et al., 2002).

Because stimulation of an autonomic modulatory reflex was thought to have potentially important therapeutic implications, Vaschillo and his colleagues then began applying this biofeedback method to patients with various neurotic and psychosomatic problems (Chernigovskaya, Vaschillo, Petrash, & Rusanovsky, 1990). Smetankin founded the Biosvyaz Company in St. Petersburg that manufactured a freestanding HRVB biofeedback unit and operated a clinic devoted to treating pediatric asthma using this method (Lehrer, Smetankin, & Potapova, 2000). Influenced by this Russian research, Richard Gevirtz and I independently began programs of research in the United States to evaluate this method. The method has been adopted as part of standard biofeedback practice by many applied psychophysiologists.

However, the notion that salutary health effects can be obtained by breathing at particular rates has been with us for many centuries. Slow breathing underlies many of the Eastern meditative techniques, including yoga and Zen, with increases in HRV noted during the practice of these disciplines (Bhagat, Kharya, Jaryal, & Deepak, 2017; Lehrer, Sasaki, & Saito, 1999; Pascoe, Thompson, & Ski, 2017; Tyagi, Cohen, Reece, Telles, & Jones, 2016), suggesting that practitioners were breathing at a resonance frequency for the cardiovascular system. HRVB systematizes these findings and makes them readily learnable in very little time. Before our work started on HRVB, Song and Lehrer (2003) and Sargunaraj et al. (1996) reported greatly increased amplitudes of HRV when people breathed at rates close to six times a minute, with little change in mean heart rate.

ASSESSMENT

The level of empirical validation for HRVB in various applications is summarized in Table 10.1. Because it appears to strengthen an important allostatic reflex and various other control reflexes associated with it, we consider this method to be an efficacious procedure to improve general resilience. We have recently performed a meta-analysis of studies on HRVB and paced breathing at about average adult resonance frequencies. We included all outcome measures analyzed in each study, whether or not they related to the target symptoms of the population or were identified as primary outcome variables. Among 58 controlled studies, we found a highly significant ($p < .0005$) moderate to small effect size (Hedges's $g = 0.37$) (Lehrer et al., 2020). This effect is particularly notable considering that many of the outcome measures were not related to problems presented by study participants and were not specifically targeted in the research and that some of the control conditions included demonstrably effective treatments. We thus concluded that HRVB contributes significantly to improving overall resilience.

METHODS OF APPLYING HRVB

Although most research on biofeedback has been done using high-end instruments with capabilities needed for the exacting requirements of research, most clinicians tend to use simpler instruments. A number of smartphone-based HRVB applications have appeared recently, some of them from plethysmograph recordings taken through the flashlight and camera in the smartphone, with others taken using various commercially available sensors that record interbeat interval taken from the pulse in the earlobe or from the EKG. The latter measures both have some advantage over recordings from the finger with the smartphone camera. Because of movement artifact or vasoconstriction from cold or stress, the pulse often is not reliably detected in the finger, and, in our experience, characteristics of the dual cameras in some advanced smartphones tend to make the pulse less easily detectable in various software packages when taken using the smart phone's camera. Most researchers prefer to use the R-spike of the EKG over the blood volume pulse signal because the spike is sharper, thus allowing a more accurate detection of the interbeat interval. The BVP is even more inexact because it is affected by phasic changes in vascular tone, which may alter the time interval between each heartbeat and the site from which the pulse is measured. This would give some advantage to a BVP from the ear rather than from the finger, as it is closer to the heart, so changes in pulse wave velocity should have a smaller impact.

TABLE 10.1. Conditions Showing Efficacy for HRVB in Controlled Trials

Condition	Studies
Anxiety	Lee, Kim, & Wachholtz (2015); Meier & Welch (2016); Mikosch et al. (2010); Munafo, Patron, & Palomba (2016); Murphy (2009); Paul & Garg (2012); Strack, Gevirtz, & Sime (2003); Thurber (2007); van der Zwan, de Vente, Huizink, Bogels, & de Bruin (2015)
Asthma	Lehrer et al. (1997, 2004)
Cognitive performance	Sutarto, Wahab, & Zin (2013)
Depression	Patron et al. (2013); Rene (2008); van der Zwan et al. (2015); Zucker, Samuelson, Muench, Greenberg, & Gevirtz (2009)
Gastrointestinal functional problems	Hjelland, Svebak, Berstad, Flatabø, & Hausken (2007)
Heart failure	Swanson et al. (2009)
Hostility	Browne (2002); Lin et al. (2015)
Hypertension	Alabdulgader (2012); Lin et al. (2012); Reineke (2008)
Insomnia	Tsai, Kuo, Lee, & Yang (2015); van der Zwan et al. (2015); Yetwin, Marks, Bell, & Gold (2012); Zucker et al. (2009)
Pain	Berry et al. (2014); Hallman, Olsson, von Scheele, Melin, & Lyskov (2011); Soer, Vos, Hofstra, & Reneman (2014); Yetwin (2012)
Performance enhancement	Gruzelier, Thompson, Redding, Brandt, & Steffert (2014); Paul, Garg, & Sandhu (2012); Raymond, Sajid, Parkinson, & Gruzelier (2005); Strack, Gevirtz, & Sime (2003)
Pregnancy outcome	Cullins et al. (2013)
PTSD	Tan, Dao, Farmer, Sutherland, & Gevirtz (2011); Zucker et al. (2009)
Stress	Browne (2002); Cullins et al. (2013); Munafo et al. (2016); Prinsloo, Derman, Lambert, & Rauch (2013); Siepmann et al. (2014); Sutarto, Wahab, & Zin (2012); van der Zwan et al. (2015)
Substance dependence	Eddie, Kim, Lehrer, Deneke, & Bates (2014); Meule, Freund, Skirde, Vögele, & Kübler (2012); Penzlin, Siepmann, Illigens, Weidner, & Siepmann (2015)

However, neither method is perfect. To detect the exact intersection between the heartbeat and the central nervous system, the activity at the sinoatrial node of the heart should be measured. This is represented by the P-wave of the EKG rather than the R-spike, which represents ejection of blood from the heart, the timing of which may be affected by various physiological factors (Ferst & Chaitman, 1984; Keller & Williams, 1993). However, the P-wave is not easily detectable using current hardware and software, so it is not used in any hardware or software packages.

Despite all of these considerations, for most clinical purposes the methods are interchangeable, as HRV measures taken from them are highly correlated (Heilman, Handelman, Lewis, & Porges, 2008).

Other methods that train people to breathe at approximately resonance frequency may have similar effects. Schein and colleagues (2001) have received Food and Drug Administration (FDA) approval to market a biofeedback device for treating hypertension that helps to slow respiration to rates similar to those produced during HRVB. In their 2001 study, they reported that systolic pressure dropped by about 15 mm Hg, although respiration rate was higher than the average adult resonance frequency. In a study of 149 patients using this device alone (with no "live" training with a trainer or therapist), the same group (Elliot et al., 2004) found a decrease of approximately 15 mm Hg in systolic blood pressure among patients using the device more than 180 minutes in 8 weeks, but only approximately 9 mm Hg among other patients and among those randomly assigned to a control group. Joseph et al. (2005) trained 20 patients who had hypertension to breathe at six breaths/minute (similar to the rate of breathing in HRVB) and compared the results with those from people breathing at 15 times/minute (average normal respiration rate). They found that slow breathing decreased systolic and diastolic pressures in patients with hypertension (from 149.7 ± 3.7 to 141.1 ± 4 mm Hg, $p < .05$; and from 82.7 ± 3 to 77.8 ± 3.7 mm Hg, $p < .01$, respectively). Controlled breathing (15/minute) decreased systolic (to 142.8 ± 3.9 mm Hg; $p < .05$) but not diastolic blood pressure and decreased RRI ($p < .05$) without altering the BR.

SIDE EFFECTS AND CONTRAINDICATIONS

When people think about controlling their breathing, they often tend to hyperventilate. This frequently happens during the first few sessions of HRVB. As a result, the training protocol guides the trainee to breathe shallowly, even if slowly, and to be sensitive to symptoms of hyperventilation, such as lightheadedness, tingling, increased anxiety, or pounding heart. If available, monitoring end-tidal CO_2 with a capnometer during early sessions can be useful.

Also, we have found that some patients with frequent extrasystolic heartbeats sometimes show an increase in these events during HRVB, often toward the end of exhalation, when higher levels of pCO_2 may trigger preventricular contractions in vulnerable people. Irregular and missed beats can easily be detected with software that displays a Poincaré plot (cf. Thayer, Yonezawa, & Sollers, 2000), in which each RRI is plotted against the previous one. It also can usually be easily observed from an instantaneous cardiotachometer output. HRVB should be used with caution among people with an irregular pattern of heartbeats, particularly when these increase during HRVB. We do not know the long-term effect or risk of HRVB in this population, although some Russian researchers have used the method to decrease occurrence of certain cardiac arrhythmias (Chernigovskaya et al., 1990; Sidorov & Vasilevskii, 1995; Vasilevskii, Sidorov, & Suvorov, 1993). Still, it is not known whether increasing the frequency of extrasystolic beats by slow breathing might pose a cardiac risk. Although extrasystolic beats often occur in periods of fatigue (Parshuram, Dhanani, Kirsh, & Cox, 2004; Stamler, Goldman, Gomes, Matza, & Horowitz, 1992); stress (Lampert, 2015); or heavy doses of alcohol (Brunner et al., 2017; George & Figueredo, 2010), persistent occurrence of such events can be associated with more serious disease and warrant medical evaluation. Also, even though a Poincaré plot in the biofeedback display can help decrease the difficulty in following a cardiotachometer tracing in biofeedback, this approach may not allow accurate detection of resonance effects if there are many extrasystolic heartbeats.

THE METHOD

We have previously published two manuals for HRVB training (Lehrer et al., 2013; Lehrer, Vaschillo, & Vaschillo, 2000). The materials presented in this section include some minor modifications of that procedure. A study by McCraty, Atkinson, and Tomasino (2003) shows that HRVB can be easily and economically used by most patients and that patients with hypertension tend to utilize the method at home sufficiently frequently and well to produce significant decreases in blood pressure, after only a single session of training. In our experience, maximal control over HRV at the resonance frequency can be obtained in most people after approximately four sessions of training. The method tends to be appealing to people partly because it is so easy to learn. Most people can see large increases in HRV occur within the first few minutes of training, and often experience some salutary effects on health and performance within only a few days of use, although major changes in symptoms, in our experience, often require several months of daily practice.

Manual for Training

The therapist's interaction with the patient as a human being is just as important as following this manual. It is important that the patient feels that the therapist understands, sympathizes with, and respects his or her experiences, including demands that the treatment makes on the client's life (time, expense, travel, etc.). Respect, warmth, and caring on the part of the therapist can be just as important for clinical success as the proper use of specific biofeedback procedures.

Therefore, it is important and appropriate to chat a little at the beginning of each session about the client's experiences between sessions, discussing difficulties faced (e.g., in experiencing symptoms, taking measures, medications) or significant life experiences (births, deaths, illnesses, job or school changes or difficulties, etc.). Use an "active listening" approach: Ask the client to expand or clarify and repeat the patient's feeling statements, indicating that you understand how he or she feels. Try to avoid judgmental statements or giving advice, except regarding specific procedures in the training.

Try to be sensitive to changes in the client's mood. If the client appears to become sad, pessimistic, anxious, or fearful, or if he or she reports severe interpersonal or financial problems, discussing these may take precedence over biofeedback procedures.

Script for Introducing the Method

"Your heart rate goes up and down with your breathing. When you breathe in, your heart rate tends to go up. When you breathe out, your heart rate tends to go down. These changes in heart rate are called 'respiratory sinus arrhythmia,' or RSA. RSA triggers very powerful reflexes in the body that help it to control the whole autonomic nervous system (including your heart rate, blood pressure, and breathing). I will train you to increase the size of these heart rate changes. Increasing the size of the heart rate changes will better exercise these important reflexes and help them to control your body.

"As part of this treatment, I will measure your RSA and give you information about the swings in heart rate that accompany breathing. That will be the RSA biofeedback. You will use the information that these machines provide to teach yourself to increase your RSA. If you practice the technique regularly at home, you will strengthen the reflexes that regulate the autonomic nervous system. This should help improve your health and ability to manage everyday stress.

"Do you have any questions?"

Use a respiration pacer stimulus set to six breaths/minute. The stimulus may be visual (e.g., a bar rising and falling) or auditory (a tone rising or falling in pitch). Usually exhalation is paced at twice the length of inhalation, and there is some evidence that this ratio enhances the relaxing effects of HRVB (Van Diest et al., 2014).

"Breathe at the rate of this bar, moving up and down. Breathe in as the bar goes up and out as it goes down. Try it."

Give feedback about whether the client is accurately following instructions.

"Now continue to breathe at this rate. Do not breathe too deeply or you will hyperventilate. If this happens you may experience some lightheadedness or dizziness. You will be breathing slowly, so you will have to breathe a little more deeply than usual. If lightheadedness or dizziness occurs breathe more shallowly.

"Breathe out longer than you breathe in." Provide prompts both for inhalation and exhalation.

"In all breathing exercises I will teach you here, the *most important thing* is to breathe in a relaxed way. Breathe easily and comfortably. *Do not try too hard*."

Determining Resonance Frequency

"We will now find your 'resonance frequency,' the speed of breathing at which your RSA is the highest. In this procedure, I will ask you to breathe at various rates for periods of about 3 minutes each. You will breathe at rates of 6.5, 6, 5.5, 5, and 4.5 breaths per minute. You should not find this task difficult. However, if you feel uncomfortable at any time, you can simply stop the task. Do you have any questions?"

Using prompts from a respiratory pacing program, have the client breathe for 2 minutes at each of five frequencies (6.5, 6, 5.5, 5, and 4.5 breaths/minute), as prompted. Set the pacing stimulus manually, as prompted by the screen. Ask the participant to breathe at each frequency for one minute, before beginning the data acquisition part of the program. Record the height of the spectral frequency at the client's respiration rate for each frequency. Extend the measurement period if breathing or HRV patterns are unstable during the assessment period.

Ordinarily, the resonance frequency is characterized by (1) the highest LF spectral peak, (2) the highest LF power, (3) the highest peak-to-trough RSA amplitude, and (4) respiration and HRV in phase with each other. The normalized LF power can sometimes give a more accurate assessment of resonance than LF power alone and is similar to measures of "coherence" given by several hardware and software devices. Also, the HR tracing will show a smooth and even sine wave when the individual is breathing at resonance frequency. If these measures are discrepant, this will usually be between two specific frequencies. Repeat the process at those frequencies. If the measures remain discrepant, choose the frequency that yields the highest LF spectral peak. If there is capability for measuring beat-to-beat blood pressure, these measures will oscillate 180°

out of phase with HR at the resonance frequency. Also, finger temperature usually rises quickly while breathing at the resonance frequency. We usually repeat testing at the respiratory frequency yielding the highest LF spectral peak, along with adjacent frequencies, in order to confirm the finding, and we may eliminate higher or lower respiration rates if criteria for resonance have been met by midrange frequencies. Some programs provide a measure of "coherence," a measure reflecting the amount of time at which the heart rate pattern appears as a simple sine wave. Although this calculation is proprietary, it appears to be a reasonable assessment of resonance, since when stimulated at resonance frequency, almost all of the variability in a signal will be at a single frequency. When stimulated at other frequencies, RSA and BR activity will often appear as separate contributions to HRV, thus disturbing the sine wave form. This measure overlaps and is highly correlated with normalized LF activity, calculated as the ratio of the area under the curve for LF activity in a spectral graph divided by total variability or total variability–VLF variability.

Inform the client of his or her resonance frequency (i.e., the frequency of maximum amplitude). If a capnometer is available, the therapist should monitor CO_2 values and instruct the participant to breathe less deeply if values fall below initial CO_2 levels.

Second Training Session

Inquire about the client's experiences with resonant-frequency breathing within the past week. If symptoms of hyperventilation occurred, remind the client to breathe more shallowly. Help the client to troubleshoot problems with scheduling and motivation. Then proceed to training in relaxed abdominal breathing, as follows.

PROVIDE TRAINING IN RELAXED ABDOMINAL BREATHING

> "One of the things you will learn in biofeedback is relaxed breathing. When you are relaxed, your chest and your abdomen relax, and you begin to breathe more naturally, so that your abdomen expands when you inhale and contracts (goes back in) when you exhale. Let me show you what I mean."

Place one hand on your own chest and the other on the abdomen and demonstrate abdominal breathing.

> "When you breathe, your diaphragm moves down and seems to push out your abdomen, so it seems as though you are breathing from your abdomen. When your diaphragm moves down, a partial vacuum is created in your lungs, so your lungs fill up. Your lungs don't do anything during breathing. They are passive, like balloons. Movement of the diaphragm makes them fill, just like blowing into the balloon.
>
> "So, your chest doesn't do much work in relaxed breathing, although as mentioned in Chapter 12 of this volume by Van Dixhoorn, the whole body is involved in truly relaxed breathing. Nevertheless, in relaxed breathing, your diaphragm does most of the work. Your diaphragm is located here."

Point to the position of the diaphragm in your own body.

> "Usually, you seem to breathe primarily from your chest when muscles in your abdomen are tense. Then you breathe by using your chest muscles, because, when your

diaphragm moves down, there is no place to go—so the diaphragm cannot force air into the lungs. In relaxed breathing, as you inhale and exhale, the bottom hand moves up and down and the top hand doesn't move much at all."

Demonstrate with two or three inhalations.

"Do you see that? Why don't you try, just to get the feel of it? Relax and place one hand on your chest and the other on your abdomen."

Let the client try it a few times while you continue to model. If the client finds abdominal breathing too difficult, however, allow the client to breathe with some chest movement, and remind him or her to continue breathing slowly.

"Practice at home for about five minutes each day. First, try it lying down or standing in front of a mirror. You may find this easier than sitting. Then try doing it while sitting. Eventually you should be able to do abdominal breathing in all positions.

"Now inhale through the nose and exhale through pursed lips, like this."

Demonstrate. Give feedback to the client. Praise the client for doing it properly.

"Now go back to breathing out for longer than you breathe in. Follow the pacing bar on the screen. Continue to do pursed lips breathing when you exhale. Breathe abdominally. Combine all three styles of breathing, like this."

Demonstrate. Give feedback. Praise the client for good attempts.

"Remember: breathe easily and comfortably. Do not try too hard."

Answer questions about this procedure. Give feedback about abdominal pursed-lips breathing with longer exhalation than inhalation. Remind the client not to breathe too deeply.

"Remember: If you feel dizzy or lightheaded, you are breathing too deeply. You are hyperventilating. Breathe more shallowly and naturally. You will get the hang of it soon."

Instruct the client to practice relaxed abdominal pursed-lips breathing at the same resonance frequency as in the previous week.

Session 3

Review relaxed abdominal breathing and resonance frequency breathing. Troubleshoot problems with both procedures. Then have the client begin to breathe at resonance frequency while you show him or her a cardiotachometer display.

INSTRUCTION IN USING THE CARDIOTACHOMETER FOR BIOFEEDBACK

Instruct the client to maximize RSA using the cardiotachometer as biofeedback. Instruct the client to do this by breathing in phase with HR changes. Remind client not to breathe too deeply, particularly if experiencing dizziness or lightheadedness, and to continue breathing at a rate that is close to his or her resonance frequency.

> "Now breathe at your resonance frequency for about 30 seconds. Then shift to following your heart rate. Look at this red line." [*Point to cardiotachometer tracing.*] "When your heart rate goes up, this line goes up. When it goes down, the line goes down. Breathe in phase with your heart rate. When your heart rate goes up, breathe in. When your heart rate goes down, breathe out.
>
> "But first, just continue breathing at your resonance frequency."

After 30 seconds to 1 minute, depending on how well the client is doing the task, prompt the individual to follow his or her HR, using the cardiotachometer feedback signal. Turn the pacer signal off.

> "Breathe with your heart rate. Make your heart rate go up as far as possible and down as far as possible with each breath. Breathe in as your heart rate goes up and out as it goes down. You will find the right point during the heart rate swing to start the inhalation or exhalation. Some people will get the best results by breathing in as soon as the heart rate starts going up and out as soon as it starts going down. For others, the best results are obtained by breathing a few seconds after the heart rate begins to change."
>
> "Breathe easily, without tension. Breathe naturally. Don't try too hard. It should just flow almost automatically. Don't think too much about how to do it. Maybe it won't work right away. It will improve with time."

Allow the client to practice for 10 minutes using biofeedback. Give praise and indicate when a correction in technique is necessary. Monitor end-tidal CO_2 if a capnometer is available and coach the client to breathe more shallowly if hyperventilation occurs.

Session 4

Session 4 is similar to session 3. Here, the client may be encouraged to borrow or purchase a home trainer. Provide instructions to the client on the use of the particular hardware and software. Have the client practice with his or her own equipment in the office.

Subsequent Sessions

There are no specific changes from the fourth session. The primary task is to monitor progress and correct errors in technique.

CASE EXAMPLE

The client was a 34-year-old woman with a long-standing asthma condition, who was referred by her physician to a study evaluating the effect of HRVB as a treatment for asthma. Over a 2-month period, with weekly visits, a pulmonary specialist had attempted to optimize her clinical condition, using the minimum possible dose of steroid medication. At the time of her first session of training, she was taking a dose of asthma controller medication at the higher levels recommended for mild persistent asthma (National Heart, Lung, and Blood Institute, 2007): three puffs of triamcinolone acetonide (Azmacort) twice daily (600 micrograms/day). Her pulmonary function was slightly below the optimal value of 80% expected for FEV_1 (volume of air exhaled during the first second of a forced expiratory maneuver with maximum effort from full vital capacity): 73%. However, she reported frequent daytime and nighttime asthma symptoms at home, sufficient to be classified as severe asthma (National Heart Lung and Blood Institute, 2007). She took measures of her expiratory flow twice daily at home. She received 10 weekly sessions of HRVB, according to the protocol previously described. Medication was titrated upward or downward biweekly by the pulmonary physician based on symptoms and pulmonary function.

Measures of peak flow increased over weeks, and peak flow variability decreased. FEV_1 gradually increased across sessions, reaching 80% expected by the last treatment session, and her level of symptomatology decreased to the level of moderate asthma. These changes occurred despite a decrease in her level of controller medication to one puff of Azmacort twice daily (200 micrograms/day).

At a 1-month follow-up visit, she had completely stopped taking Azmacort. Her home peak flow values remained stable during this period, with a brief dip during a respiratory infection. Because of the infection, her average symptomatology increased, although her pulmonary function values at the office remained stable. The patient's BR gain also increased across sessions as assessed by cross-spectral analysis of HR and beat-to-beat blood pressure (from a Finapres unit).

The patient was informally followed up at 6 and 12 months. She continued practicing breathing at her resonance frequency, had not experienced any asthma flares, rarely used her bronchodilator, and had not returned to taking asthma controller medications.

In summary, the patient showed a clinically significant improvement in pulmonary function, along with complete elimination of high doses of inhaled steroid controller medication. Her level of symptoms remained high, either because of overperception of symptoms or because the assessment metrics included periods in which she had a respiratory infection. BR gain increased both immediately, while performing biofeedback procedures, and chronically, as measured during the 5-minute pretraining periods in the first, fourth, seventh, and tenth sessions of training.

SUMMARY AND CONCLUSIONS

HRVB strengthens an important autonomic control reflex (the BR), increases respiratory efficiency, and improves the function of brain pathways that regulate emotion. It improves signs and symptoms of a wide variety of disorders and improves human performance and general resilience. It appears it is easy to learn, using inexpensive and user-friendly equipment. It appears to be useful as an adjunct treatment for a variety of applications and has few and transient side effects.

REFERENCES

Aboussafy, D., Campbell, T. S., Lavoie, K., Aboud, F. E., & Ditto, B. (2005). Airflow and autonomic responses to stress and relaxation in asthma: The impact of stressor type. *International Journal of Psychophysiology, 57*(3), 195–201.

Agelink, M. W., Boz, C., Ullrich, H., & Andrich, J. (2002). Relationship between major depression and heart rate variability: Clinical consequences and implications for antidepressive treatment. *Psychiatry Research, 113,* 139–149.

Alabdulgader, A. A. (2012). Coherence: A novel nonpharmacological modality for lowering blood pressure in hypertensive patients. *Global Advance in Health and Medicine, 1,* 54–62.

Alamili, M., Rosenberg, J., & Gogenur, I. (2015). Day–night variation in heart rate variability changes induced by endotoxaemia in healthy volunteers. *Acta Anaesthesiologica Scandinavica, 59*(4), 457–464.

Angrilli, A., Sarlo, M., Palomba, D., Schincaglia, M., & Stegagno, L. (1997). Respiratory sinus arrhythmia in blood phobic subjects. *Perceptual and Motor Skills, 84*(2), 505–506.

Arzeno, N. M., Kearney, M. T., Eckberg, D. L., Nolan, J., & Poon, C. S. (2007, August). Heart rate chaos as a mortality predictor in mild to moderate heart failure. *Proceedings of the Annual International Conference of the IEEE Engineering in Medicine and Biology Society,* 5051–5054.

Austen, M. L., & Wilson, G. V. (2001). Increased vagal tone during winter in subsyndromal seasonal affective disorder. *Biological Psychiatry, 50*(1), 28–34.

Azuaje, F., Dubitzky, W., Lopes, P., Black, N., Adamson, K., Wu, X., et al. (1999). Predicting coronary disease risk based on short-term RR interval measurements: A neural network approach. *Artificial Intelligence in Medicine, 15*(3), 275–297.

Balsam, P., Sanchez-Castillo, H., Taylor, K., Van Volkinburg, H., & Ward, R. D. (2009). Timing and anticipation: Conceptual and methodological approaches. *European Journal of Neuroscience, 30*(9), 1749–1755.

Barakat, A., Vogelzangs, N., Licht, C. M., Geenen, R., MacFarlane, G. J., de Geus, E. J., et al. (2012). Dysregulation of the autonomic nervous system and its association with the presence and intensity of chronic widespread pain. *Arthritis Care and Research, 64*(8), 1209–1216.

Bass, C. (1997). Hyperventilation syndrome: A chimera? *Journal of Psychosomatic Research, 42*(5), 421–426.

Bawa, P. (2002). Neural control of motor output: Can training change it? *Exercise and Sport Sciences Reviews, 30,* 59–63.

Bayes de Luna, A., Coumel, P., & Leclercq, J. F. (1989). Ambulatory sudden cardiac death: Mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *American Heart Journal, 117,* 151–159.

Bernardi, L., Gabutti, A., Porta, C., & Spicuzza, L. (2001). Slow breathing reduces chemoreflex response to hypoxia and hypercapnia and increases baroreflex sensitivity. *Journal of Hypertension, 19,* 2221–2229.

Bernardi, L., Passino, C., Wilmerding, V., Dallam, G. M., Parker, D. L., Robergs, R. A., et al. (2001). Breathing patterns and cardiovascular autonomic modulation during hypoxia induced by simulated altitude. *Journal of Hypertension, 19,* 947–958.

Bernardi, L., Portta, C., Spicuzza, L., Bellwon, J., Spadacini, G., Frey, A. W., et al. (2002). Slow breathing increases the arterial baroreflex sensitivity in patients with chronic heart failure. *Circulation, 105,* 143–145.

Bernardi, L., Schneider, A., Pomidori, L., Paolucci, E., & Cogo, A. (2006). Hypoxic ventilatory response in successful extreme altitude climbers. *European Respiratory Journal, 27(1),* 165–171.

Bernardi, L., Sleight, P., Bandinelli, G., Cencetti, S., Fattorini, L., Wdowczyc-Szulc, J., et al. (2001). Effect of rosary prayer and yoga mantras on autonomic cardiovascular rhythms: Comparative study. *British Medical Journal, 323*(7327), 1446–1449.

Bernardi, L., Spadacini, G., Bellwon, J., Hajric, R., Roskamm, H., & Frey, A. W. (1998). Influence of breathing frequency and pattern on oxygen saturation and exercise performance. *Lancet, 351,* 1308–1311.

Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34,* 623–648.

Berry, M. E., Chapple, I. T., Ginsberg, J. P., Gleichauf, K. J., Meyer, J. A., & Nagpal, M. L. (2014). Non-pharmacological intervention for chronic pain in veterans: A pilot study of heart rate variability biofeedback. *Global Advances in Health and Medicine, 3*(2), 28–33.

Bhagat, O. L., Kharya, C., Jaryal, A., & Deepak, K. K. (2017). Acute effects on cardiovascular oscillations during controlled slow yogic breathing. *Indian Journal of Medical Research, 145*(4), 503–512.

Bigger, J. T., Fleiss, J. L., Rolnitzky, L. M., & Steinman, R. C. (1992). The ability of several short-term measures of RR variability to predict mortality after myocardial infarction. *Circulation, 88,* 927–934.

Billman, G. E., Schwartz, P. J., & Stone, H. L. (1983). Baroreceptor reflex control of heart rate: A predictor of sudden cardiac death. *Circulation, 66,* 874–880.

Borelli, J. L., Smiley, P. A., Rasmussen, H. F., Gomez, A., Seaman, L. C., & Nurmi, E. L.

(2017). Interactive effects of attachment and FKBP5 genotype on school-aged children's emotion regulation and depressive symptoms. *Behavioural Brain Research, 325*(Pt. B), 278–289.

Botha, C., Farmer, A. D., Nilsson, M., Brock, C., Gavrila, A. D., Drewes, A. M., et al. (2015). Preliminary report: Modulation of parasympathetic nervous system tone influences oesophageal pain hypersensitivity. *Gut, 64*(4), 611–617.

Brindle, R. C., Ginty, A. T., Phillips, A. C., & Carroll, D. (2014). A tale of two mechanisms: A meta-analytic approach toward understanding the autonomic basis of cardiovascular reactivity to acute psychological stress. *Psychophysiology, 51*(10), 964–976.

Brindle, R. C., Ginty, A. T., Phillips, A. C., Fisher, J. P., McIntyre, D., & Carroll, D. (2016). Heart rate complexity: A novel approach to assessing cardiac stress reactivity. *Psychophysiology, 53*(4), 465–472.

Brown, L., Karmakar, C., Gray, R., Jindal, R., Lim, T., & Bryant, C. (2018). Heart rate variability alterations in late life depression: A meta-analysis. *Journal of Affective Disorders, 235,* 456–466.

Brown, R. P., & Gerbarg, P. L. (2005). Sudarshan Kriya yogic breathing in the treatment of stress, anxiety, and depression: Part 1. Neurophysiologic model. *Journal of Alternative and Complementary Medicine, 11,* 189–201.

Brown, S. M., Tate, Q., Jones, J. P., Knox, D. B., Kuttler, K. G., Lanspa, M., et al. (2013). Initial fractal exponent of heart rate variability is associated with success of early resuscitation in patients with severe sepsis or septic shock: A prospective cohort study. *Journal of Critical Care, 28*(6), 959–963.

Browne, T. G. (2002). EEG theta enhancement and heart rate variability biofeedback and interactional stress in a clinical population. *Applied Psychophysiology and Biofeedback, 27*(4), 312.

Brunner, S., Herbel, R., Drobesch, C., Peters A., Massberg, S., Kaab, S., et al. (2017). Alcohol consumption, sinus tachycardia, and cardiac arrhythmias at the Munich Octoberfest: Results from the Munich Beer Related Electrocardiogram Workup Study (MunichBREW). *European Heart Journal, 38*(27), 2100–2106.

Bylsma, L. M., Salomon, K., Taylor-Clift, A., Morris, B. H., & Rottenberg, J. (2014). Respiratory sinus arrhythmia reactivity in current and remitted major depressive disorder. *Psychosomatic Medicine, 76*(1), 66–73.

Byrd, D. L., Reuther, E. T., McNamara, J. P., DeLucca, T. L., & Berg, W. K. (2015). Age differences in high frequency phasic heart rate variability and performance response to increased executive function load in three executive function tasks. *Frontiers in Psychology, 5,* 1470.

Byun, S., Kim, A. Y., Jang, E. H., Kim, S., Choi, K. W., & Jeon, H. J. (2019). Entropy analysis of heart rate variability and its application to recognize major depressive disorder: A pilot study. *Technology and Health Care, 27*(S1), 407–424.

Carpenter, J. S., Burns, D. S., Wu, J., Otte, J. L., Schneider, B., Ryker, K., et al. (2013). Paced respiration for vasomotor and other menopausal symptoms: A randomized, controlled trial. *Journal of General Internal Medicine, 28*(2), 193–200.

Carter, J. B., Banister, E. W., & Blaber, A. P. (2003). The effect of age and gender on heart rate variability after endurance training. *Medicine and Science in Sports and Exercise, 35*(8), 1333–1340.

Caruso, F. R., Arena, R., Phillips, S. A., Bonjorno, J. C., Jr., Mendes, R. G., Arakelian, V. M., et al. (2015). Resistance exercise training improves heart rate variability and muscle performance: A randomized controlled trial in coronary artery disease patients. *European Journal of Physical and Rehabilitation Medicine, 51*(3), 281–289.

Chalmers, J. A., Heathers, J. A., Abbott, M. J., Kemp, A. H., & Quintana, D. S. (2016). Worry is associated with robust reductions in heart rate variability: A transdiagnostic study of anxiety psychopathology. *BMC Psychology, 4,* 32.

Chang, H.-A., Fang, W.-H., Wan, F.-J., Tzeng, N.-S., Liu, Y.-P., Shyu, J.-F., et al. (2019). Attenuated vagally-mediated heart rate variability at rest and in response to postural maneuvers in patients with generalized anxiety disorder. *Psychological Medicine.* Available at *https://tinyurl.com/yd4m2jpb.*

Chernigovskaya, N. V., Vaschillo, E. G., Petrash, V. V., & Rusanovsky, V. V. (1990). Voluntary regulation of the heart rate as a method of functional condition correction in neurotics. *Human Physiology, 16,* 58–64.

Choi, J.-B., Hong, S., Nelesen, R., Bardwell, W. A., Natarajan, L., Schubert, C., et al. (2006). Age and ethnicity differences in short-term heart-rate variability. *Psychosomatic Medicine, 68*(3), 421–426.

Choi, K. W., Jang, E. H., Kim, A. Y., Fava, M., Mischoulon, D., Papakostas, G. I., et al. (2019). Heart rate variability for treatment response between patients with major depressive disorder versus panic disorder: A 12-week follow-up study. *Journal of Affective Disorders, 246,* 157–165.

Chou, C. L., Kao, N. T., Kuo, T. B., Liu, T. J., & Chan, R. C. (1998). Heart rate variability as an assessment of acute rejection after heart transplantation. *Chinese Medical Journal, 61*(10), 561–567.

Christensen, J. H., Dyerberg, J., & Schmidt, E. B. (1999). n-3 fatty acids and the risk of sudden cardiac death assessed by 24-hour heart rate variability. *Lipids, 34*(Suppl.), S197.

Clark, M. T., Rusin, C. G., Hudson, J. L., Lee, H., Delos, J. B., Guin, L. E., et al. (2012). Breath-by-breath analysis of cardiorespiratory interaction for quantifying developmental maturity in premature infants. *Journal of Applied Physiology, 112*(5), 859–867.

Condy, E. E., Scarpa, A., & Friedman, B. H. (2019). Restricted repetitive behaviors in autism spectrum disorder: A systematic review from the neurovisceral integration perspective. *Biological Psychology, 148,* 107739.

Crawford-Achour, E., Roche, F., Pichot, V., Celle, S., Barthelemy, J. C., & Chouchou, F. (2016). Sleep-related autonomic overactivity in a general elderly population and its relationship to cardiovascular regulation. *Heart and Vessels, 31*(1), 46–51.

Critchley, H. D., Nicotra, A., Chiesa, P. A., Nagai, Y., Gray, M. A., Minati, L., et al. (2015). Slow breathing and hypoxic challenge: Cardiorespiratory consequences and their central neural substrates. *PLOS ONE, 10*(5), e0127082.

Cullins, S. W., Gevirtz, R. N., Poeltler, D. M., Cousins, L. M., Harpin, R., & Muench, F. (2013). An exploratory analysis of the utility of adding cardiorespiratory biofeedback in the standard care of pregnancy-induced hypertension. *Applied Psychophysiology and Biofeedback, 38*(3), 161–170.

Davydov, D. M., Shapiro, D., Cook, I. A., & Goldstein, I. (2007). Baroreflex mechanisms in major depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 31*(1), 164–177.

De la Torre-Luque, A., Balle, M., Fiol-Veny, A., Llabres, J., & Bornas, X. (2018). Nonlinear Dynamics. *Psychology, and Life Sciences, 22*(3), 313–333.

De Meersman, R. E. (1993). Heart rate variability and aerobic fitness. *American Heart Journal, 125*(3), 726–731.

DeBoer, R. W., Karemaker, J. M., & Strackee, J. (1987). Hemodynamic fluctuations and baroreflex sensitivity in humans: A beat-to-beat model. *American Journal of Physiology: Heart and Circulatory Physiology, 253*(22), H680–H689.

Del Pozo, J., Gevirtz, R., & Guanieri, M. (2004). Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. *American Heart Journal, 147,* E11.

Dennis, P. A., Weinberg, J. B., Calhoun, P. S., Watkins, L. L., Sherwood, A., Dennis, M. F., et al. (2016). An investigation of vago-regulatory and health-behavior accounts for increased inflammation in posttraumatic stress disorder. *Journal of Psychosomatic Research. 83,* 33–39.

Dimitriev, D. A., Saperova, E. V., & Dimitriev, A. D. (2016). State anxiety and nonlinear dynamics of heart rate variability in students. *PLOS ONE, 11*(1), e0146131.

Dreifus, L. S., Agarwal, J. B., Botvinick, E. H., Ferdinand, K. C., Fisch, C., Fisher, J. D., et al. (1993). Heart rate variability for risk stratification of life-threatening arrhythmias. *Journal of the American College of Cardiology, 22,* 948–950.

Drummond, P. D. (1990). Parasympathetic cardiac control in mild hypertension. *Journal of Hypertension, 8*(4), 383–387.

Dubois, A., Brody, A., Lewis, D., & Burgess, B. (1956). Oscillation mechanics of lungs and chest in man. *Journal of Applied Physiology, 8,* 587–594.

Eckberg, D. L. (1979). Carotid baroreflex function in young men with borderline blood pressure elevation. *Circulation, 59,* 632–636.

Eckberg, D. L. (2000). Physiological basis for human autonomic rhythms. *Annals of Medicine, 32,* 341–349.

Eckberg, D. L., & Kuusela, T. A. (2005). Human vagal baroreflex sensitivity fluctuates widely and rhythmically at very low frequencies. *Journal of Physiology, 567*(Pt. 3), 1011–1019.

Eckberg, D. L., & Sleight, P. (1992). *Human baroreflexes in health and disease.* Oxford, UK: Clarendon Press.

Eddie, D., Kim, C., Lehrer, P., Deneke, E., & Bates, M. (2014). A pilot study of brief heart rate variability biofeedback to reduce craving in young adult men receiving inpatient treatment for substance use disorders. *Applied Psychophysiology and Biofeedback, 39*(3–4), 181–192.

Elghozi, J. L., & Julien, C. (2007). Sympathetic control of short-term heart rate variability and its pharmacological modulation. *Fundamental and Clinical Pharmacology, 21*(4), 337–347.

Ellenby, M. S., McNames, J., Lai, S., McDonald, B. A., Krieger, D., Sclabassi, R. J., et al. (2001). Uncoupling and recoupling of autonomic regulation of the heart beat in pediatric septic shock. *Shock, 16*(4), 274–277.

Eller, N. H. (2007). Total power and high frequency components of heart rate variability and risk factors for atherosclerosis. *Autonomic Neuroscience: Basic and Clinical, 131*(1–2), 123–130.

Elliot, W. J., Izzo, J. L., Jr., White, W. B., Rosing, D. R., Snyder, C. S., Alter, A., et al. (2004). Graded blood pressure reduction in hypertensive outpatients associated with use of a device to assist with slow breathing. *Journal of Clinical Hypertension, 6,* 553–561.

Engel, G. L. (1978). Psychologic stress, vasodepressor (vasovagal) syncope, and sudden death. *Annals of Internal Medicine, 89*(3), 403–412.

Ewen, S., Bohm, M., & Mahfoud, F. (2017). Long-term follow-up of baroreflex activation therapy in resistant hypertension: Another piece of the puzzle? *Hypertension, 69*(5), 782–784.

Fallavollita, L., Marsili, B., Castelli, S., Cucchi, F., Santillo, E., Marini, L., et al. (2016). Short-term results of a 5-week comprehensive cardiac rehabilitation program after first-time myocardial infarction. *Journal of Sports Medicine and Physical Fitness, 56*(3), 311–318.

Farrell, T. G., Bashir, Y., Cripps, T., Malik, M., Poloniecki, J., Bennett, E. D., et al. (1991). Risk stratification for arrhythmic events in postinfarction patients based on heart rate variability, ambulatory electrocardiographic variables and signal-averaged electrocardiogram. *Journal of the American College of Cardiology, 18,* 687–697.

Feldman, J. M., Lehrer, P. M., Hochron, S. M., & Schwartz, G. E. (2002). Defensiveness and individual response stereotypy in asthma. *Psychosomatic Medicine, 64,* 294–301.

Ferst, J. A., & Chaitman, B. R. (1984). The electrocardiogram and the athlete. *Sports Medicine, 1*(5), 390–403.

Fiol-Veny, A., De la Torre-Luque, A., Balle, M., & Bornas, X. (2018). Diminished heart rate complexity in adolescent girls: A sign of vulnerability to anxiety disorders? *Anxiety, Stress, and Coping, 31*(4), 375–386.

Fisher, L. (2020). Beyond stationary data collection: Investigation of a proposed method for determining heart rate variability resonance frequency. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 81*(9-B).

Fonoberova, M., Mezic, I., Buckman, J. F., Fonoberov, V. A., Mezic, A., Vaschillo, E. G., et al. (2014). A computational physiology approach to personalized treatment models: The beneficial effects of slow breathing on the human cardiovascular system. *American Journal of Physiology: Heart and Circulatory Physiology, 307*(7), H1073–H1091.

Franco, P., Groswasser, J., Scaillet, S., Lanquaif, J.-P., Benatar, A., Sastre, J.-P., et al. (2008). QT interval prolongation in future SIDS victims: A polysomnographic study. *Sleep: Journal of Sleep and Sleep Disorders Research, 31*(12), 1691–1699.

Freitas, I. M. G., de Almeida, L. B., Pereira, N. P., Mira, P. A. C., de Paula, R. B., Martinez, D. G., et al. (2017). Baroreflex gain and vasomotor sympathetic modulation in resistant hypertension. *Clinical Autonomic Research, 27*(3), 175–184.

Fuller-Rowell, T. E., Williams, D. R., Love, G. D., McKinley, P. S., Sloan, R. P., & Ryff, C. D. (2013). Race differences in age-trends of autonomic nervous system functioning. *Journal of Aging and Health, 25*(5), 839–862.

Gautier, C., Stine, L., Jennings, J. R., Sutton-Tyrrell, K., Muldoon, M. B., Kamarck, T. W., et al. (2007). Reduced low-frequency heart rate variability relates to greater intimal-medial thickness of the carotid wall in two samples. *Coronary Artery Disease, 18*(2), 97–104.

Gellhorn, E., & Loofbourrow, G. N. (1963). *Emotions and emotional disorders: A neurophysiological study.* Oxford, England: Harper & Row.

George, A., & Figueredo, V. M. (2010). Alcohol and arrhythmias: A comprehensive review. *Journal of Cardiovascular Medicine, 11*(4), 221–228.

Giardino, N. D. (2003). A controlled study of the effects of chemical and psychological stress on Persian Gulf War veterans with chronic fatigue. *Dissertation Abstracts International, 63*(10-B), 4901.

Giardino, N. D., Chan, L., & Borson, S. (2004). Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: Preliminary findings. *Applied Psychophysiology and Biofeedback, 29,* 121–133.

Giardino, N., Lehrer, P. M., & Feldman, J. (2000). The role of oscillations in self-regulation: Their contribution to homeostasis. In D.

Kenny, J. G. Carlson, F. J. McGuigan, & J. L. Sheppard (Eds.), *Stress and health: Research and clinical applications* (pp. 27–52). Newark, NJ: Harwood.

Goldberger, A. L. (1991). Is the normal heartbeat chaotic or homeostatic? *News in Physiological Science, 6,* 87–91.

Goldberger, A. L., Peng, C. K., & Lipsitz, L. A. (2002). What is physiologic complexity and how does it change with aging and disease? *Neurobiology of Aging, 23*(1), 23–26.

Goldberger, A. L., & Rigney, D. R. (1990). Sudden death is not chaos. In S. Krasner (Ed.), *The ubiquity of chaos* (pp. 23–34). Washington, DC: American Association for the Advancement of Science.

Goldberger, A. L., Rigney, D. R., Mietus, J., Antman, E. M., & Greenwald, S. (1988). Nonlinear dynamics in sudden cardiac death syndrome: Heart rate oscillations and bifurcations. *Experientia, 44,* 983–987.

Gordin, D., Vikatmaa, P., Vikatmaa, L., Groop, P. H., Alback, A., & Tikkanen, I. (2016). Baroreflex activation therapy in the treatment of resistant hypertension. *Duodecim, 132*(20), 1874–1881.

Gorman, J. M., & Sloan, R. P. (2000). Heart rate variability in anxiety and depressive disorders. *American Heart Journal, 140,* S77–S83.

Gouin, J.-P., Zhou, B., & Fitzpatrick, S. (2015). Social integration prospectively predicts changes in heart rate variability among individuals undergoing migration stress. *Annals of Behavioral Medicine, 49*(2), 230–238.

Gray, M. A., Taggart, P., Sutton, P. M., Groves, D., Holdright, D. R., Bradbury, D., et al. (2007). A cortical potential reflecting cardiac function. *Proceedings of the National Academy of Sciences of the USA, 104,* 6818–6823.

Grippo, A. J., Lamb, D. G., Carter, C. S., & Porges, S. W. (2007). Social isolation disrupts autonomic regulation of the heart and influences negative affective behaviors. *Biological Psychiatry, 62*(10), 1162–1170.

Grossman, P., Brinkman, A., & de Vries, J. (1992). Cardiac autonomic mechanisms associated with borderline hypertension under varying behavioral demands: Evidence for attenuated parasympathetic tone but not for enhanced beta-adrenergic activity. *Psychophysiology, 29*(6), 698–711.

Gruzelier, J., Thompson, T., Redding, E., Brandt, R., & Steffert, T. (2014). Application of alpha/theta neurofeedback and heart rate variability training to young contemporary dancers: State anxiety and creativity. *International Journal of Psychophysiology, 93*(1), 105–111.

Guo, G. B., & Abboud, F. M. (1984). Impaired central mediation of the arterial baroreflex in chronic renal hypertension. *American Journal of Physiology, 246*(5, Pt. 2), H720–H727.

Guzzetti, S., Mezzetti, S., Magatelli, R., Porta, A., De Angelis, G., Rovelli, G., et al. (2000). Linear and non-linear 24 h heart rate variability in chronic heart failure. *Autonomic Neuroscience: Basic and Clinical, 86*(1–2), 114–119.

Hallman, D. M., Holtermann, A., Sogaard, K., Krustrup, P., Kristiansen, J., & Korshoj, M. (2017). Effect of an aerobic exercise intervention on cardiac autonomic regulation: A worksite RCT among cleaners. *Physiology and Behavior, 169,* 90–97.

Hallman, D. M., Olsson, E. M., von Scheele, B., Melin, L., & Lyskov, E. (2011). Effects of heart rate variability biofeedback in subjects with stress-related chronic neck pain: A pilot study. *Applied Psychophysiology and Biofeedback, 36*(2), 71–80.

Hamdan, M. H., Joglar, J. A., Page, R. L., Zagrodzky, J. D., Sheehan, C. J., Wasmund, S. L., et al. (1999). Baroreflex gain predicts blood pressure recovery during simulated ventricular tachycardia in humans. *Circulation, 100,* 381–386.

Hammer, P. E., & Saul, J. P. (2005). Resonance in a mathematical model of baroreflex control: Arterial blood pressure waves accompanying postural stress. *American Journal of Physiology: Regulatory Integrative and Comparative Physiology, 288*(6), R1637–R1648.

Harrewijn, A., Van der Molen, M., Verkuil, B., Sweijen, S., Houwing-Duistermaat, J., & Westenberg, P. (2018). Heart rate variability as candidate endophenotype of social anxiety: A two-generation family study. *Journal of Affective Disorders, 237,* 47–55.

Hedelin, R., Wiklund, U., Bjerle, P., & Henriksson-Larsen, K. (2000). Pre- and post-season heart rate variability in adolescent cross-country skiers. *Scandinavian Journal of Medicine and Science in Sports, 10,* 298–303.

Hegel, M. T., & Ferguson, R. J. (1997). Psychophysiological assessment of respiratory function in panic disorder: Evidence for a hyperventilation subtype. *Psychosomatic Medicine, 59*(3), 224–230.

Heilman, K. J., Handelman, M., Lewis, G., & Porges, S. W. (2008). Accuracy of the StressEraser in the detection of cardiac rhythms. *Applied Psychophysiology and Biofeedback, 33*(2), 83–89.

Henderson, L. A., Richard, C. A., Macey, P. M., Runquist, M. L., Yu, P. L., Galons, J. P., et al. (2004). Functional magnetic resonance signal changes in neural structures to baroreceptor reflex activation. *Journal of Applied Physiology, 96*(2), 693–703.

Henze, M., Hart, D., Samarel, A., Barakat, J., Eckert, L., & Scrogin K. (2008). Persistent alterations in heart rate variability, baroreflex sensitivity, and anxiety-like behaviors during development of heart failure in the rat. *American Journal of Physiology: Heart and Circulatory Physiology, 295*(1), H29–H38.

Hjelland, I. E., Svebak, S., Berstad, A., Flatabø, G., & Hausken, T. (2007). Breathing exercises with vagal biofeedback may benefit patients with functional dyspepsia. *Scandinavian Journal of Gastroenterology, 42*(9), 1054–1062.

Ho, K. K., Moody, G. B., Peng, C. K., Mietus, J. E., Larson, M. G., Levy, D., et al. (1997). Predicting survival in heart failure case and control subjects by use of fully automated methods for deriving nonlinear and conventional indices of heart rate dynamics. *Circulation, 96*(3), 842–848.

Holt-Lunstad, J., Uchino, B. N., Smith, T. W., & Hicks, A. (2007). On the importance of relationship quality: The impact of ambivalence in friendships on cardiovascular functioning. *Annals of Behavioral Medicine, 33*(3), 278–290.

Hon, E. H., & Lee, S. T. (1965). Electronic evaluations of the fetal heart rate patterns preceding fetal death: Further observations. *American Journal of Obstetrics and Gynecology, 87,* 814–826.

Hopper, J. W., Spinazzola, J., Simpson, W. B., & van der Kolk, B. A. (2006). Preliminary evidence of parasympathetic influence on basal heart rate in posttraumatic stress disorder. *Journal of Psychosomatic Research, 60*(1), 83–90.

Houle, M. S., & Billman, G. E. (1999). Low-frequency component of the heart rate variability spectrum: A poor marker of sympathetic activity. *American Journal of Physiology, 276*(1, Pt. 2), H215–H223.

Huang, C., Gevirtz, R., Onton, J., & Criado, J. (2014, March). *Investigation of vagal afferent functioning using heart beat event related potentials.* Paper presented at the 45th annual meeting of the Association for Applied Psychophysiology and Biofeedback, Savannah, GA.

Huikuri, H. V., & Makikallio, T. H. (2001). Heart rate variability in ischemic heart disease. *Autonomic Neuroscience: Basic and Clinical, 90,* 95–101.

Huikuri, H. V., Zaman, L., Castellanos, A., Kessler, K. M., Cox, M., Glicksman, F., et al. (1989). Changes in spontaneous sinus node rate as an estimate of cardiac autonomic tone during stable and unstable ventricular tachycardia. *Journal of the American College of Cardiology, 13,* 646–652.

Izrailtyan, I., Kresh, J. Y., Morris, R. J., Brozena, S. C., Kutalek, S. P., & Wechsler, A. S. (2000). Early detection of acute allograft rejection by linear and nonlinear analysis of heart rate variability. *Journal of Thoracic and Cardiovascular Surgery, 120,* 737–745.

James, M. A., Panerai, R. B., & Potter, J. F. (1998). Applicability of new techniques in the assessment of arterial baroreflex sensitivity in the elderly: A comparison with established pharmacological methods. *Clinical Science, 94,* 245–253.

Joseph, C. N., Porta, C., Casucci, G., Casiraghi, N., Maffeis, M., Rossi, M., et al. (2005). Slow breathing improves arterial baroreflex sensitivity and decreases blood pressure in essential hypertension. *Hypertension, 46,* 714–718.

Kawachi, I., Sparrow, D., Vokonas, P. S., & Weiss, S. T. (1995). Decreased heart rate variability in men with phobic anxiety. *American Journal of Cardiology, 75,* 882–885.

Keller, C., & Williams, A. (1993). Cardiac dysrhythmias associated with central nervous system dysfunction. *Journal of Neuroscience Nursing, 25*(6), 349–355.

Kenneback, G., Ericson, M., Tomson, T., & Bergfeldt, L. (1997). Changes in arrhythmia profile and heart rate variability during abrupt withdrawal of antiepileptic drugs: Implications for sudden death. *Seizure, 6*(5), 369–375.

Kidwell, M., & Ellenbroek, B. A. (2018). Heart and soul: Heart rate variability and major depression. *Behavioural Pharmacology, 29*(2–3), 152–164.

Kolacz, J. (2017). Investigating infant crying persistence and cry acoustic features as early risk indicators for social adjustment: Developmental associations with infant vagal tone and attachment stress. *Dissertation Abstracts International, 78*(5-B(E)).

Kovacs, M., Yaroslavsky, I., Rottenberg, J., George, C. J., Kiss, E., Halas, K., et al. (2016). Maladaptive mood repair, atypical respiratory sinus arrhythmia, and risk of a recurrent major depressive episode among adolescents with prior major depression. *Psychological Medicine, 46*(10), 2109–2119.

Kox, M., Pompe, J. C., van der Hoeven, J. G., Hoedemaekers, C. W., & Pickkers, P. (2011). Influence of different breathing patterns on heart rate variability indices and reproducibility during experimental endotoxaemia in human subjects. *Clinical Science, 121*(5), 215–222.

Kuang, D., Yang, R., Chen, X., Lao, G., Wu, F., Huang, X., et al. (2017). Depression recognition according to heart rate variability using Bayesian networks. *Journal of Psychiatric Research, 95,* 282–287.

Kwon, H. B., Yoon, H., Choi, S. H., Choi, J. W., Lee, Y. J., & Park, K. S., (2019). Heart rate variability changes in major depressive disorder during sleep: Fractal index correlates with BDI score during REM sleep. *Psychiatry Research, 271,* 291–298.

La Rovere, M. T., Bigger, J. T., Jr., Marcus, F. I., Mortara, A., & Schwartz, P. J. (1998). Baroreflex sensitivity and heart-rate variability in prediction of total cardiac mortality after myocardial infarction. *Lancet, 351*(9101), 478–484.

Laitinen, T., Hartikainen, J., Niskanen, L., Geelen, G., & Länsimies, E. (1999). Sympathovagal balance is a major determinant of short-term blood pressure variability in healthy subjects. *American Journal of Physiology: Heart and Circulatory Physiology, 276,* H1245–H1252.

Lampert, R. (2015). ECG signatures of psychological stress. *Journal of Electrocardiology, 48*(6), 1000–1005.

Lee, J., Kim, J. K., & Wachholtz, A. (2015). The benefit of heart rate variability biofeedback and relaxation training in reducing trait anxiety. *Hanguk Simni Hakhoe Chi Kongang, 20*(2), 391–408.

Lehrer, P. M., Carr, R. E., Smetankine, A., Vaschillo, E. G., Peper, E., Porges, S. W., et al. (1997). Respiratory sinus arrhythmia versus neck/trapezius EMG and incentive inspirometry biofeedback for asthma: A pilot study. *Applied Psychophysiology and Biofeedback, 22*(2), 95–109.

Lehrer, P., & Eddie, D. (2013). Dynamic processes in regulation and some implications for biofeedback and biobehavioral interventions. *Applied Psychophysiology and Biofeedback, 38,* 143–155.

Lehrer, P. M., Groveman, A., Randolph, C., Miller, M. H., & Pollack, I. (1989). Physiological response patterns to cognitive testing in adults with closed head injuries. *Psychophysiology, 26*(6), 668–675.

Lehrer, P. M., Hochron, S., Carr, R., Edelberg, R., Hamer, R., Jackson, A., et al. (1996). Behavioral task-induced bronchodilation in asthma during active and passive tasks: A possible cholinergic link to psychologically induced airway changes. *Psychosomatic Medicine, 58*(5), 413–422.

Lehrer, P. M., Irvin, C. G., Lu, S. E., Scardella, A., Roehmheld-Hamm, B., Aviles-Velez, M., et al. (2018). Heart rate variability biofeedback does not substitute for asthma steroid controller medication. *Applied Psychophysiology and Biofeedback, 43*(1), 57–73.

Lehrer, P. M., Karavidas, M. K., Lu, S.-E., Coyle, S. M., Oikawa, L. O., Macor, M., et al. (2010). Voluntarily produced increases in heart rate variability modulate autonomic effects of endotoxin induced systemic inflammation: An exploratory study. *Applied Psychophysiology and Biofeedback, 35*(4), 303–315.

Lehrer, P., Kaur, K., Sharma, A., Shah, K., Huseby, R., Bhavsar, J., et al. (2020). Heart rate variability biofeedback improves emotional and physical health and performance: A systematic review and meta analysis. *Applied Psychophysiology and Biofeedback, 45*(3), 109–129.

Lehrer, P. M., & Leiblum, S. R. (1981). Physiological, behavioral, and cognitive measures of assertiveness and assertion anxiety. *Behavioral Counseling Quarterly, 1,* 261–274.

Lehrer, P. M., Sasaki, Y., & Saito, Y. (1999). Zazen and cardiac variability. *Psychosomatic Medicine, 61*(6), 812–821.

Lehrer, P. M., Smetankin, A., & Potapova, T. (2000). Respiratory sinus arrhythmia biofeedback therapy for asthma: A report of 20 unmedicated pediatric cases using the Smetankin method. *Applied Psychophysiology and Biofeedback, 25,* 193–200.

Lehrer, P. M., & Taylor, G. (1974). The effects of alcohol on cardiac reactivity in alcoholics and normal subjects. *Quarterly Journal of Studies on Alcohol, 35,* 1044–1052.

Lehrer, P., Vaschillo, E., Lu, S.-E., Eckberg, D., Vaschillo, B., Scardella, A., et al. (2006). Heart rate variability biofeedback: Effects of age on heart rate variability, baroreflex gain, and asthma. *Chest, 129,* 278–284.

Lehrer, P., Vaschillo, E., Trost, Z., & France, C. R. (2009). Effects of rhythmical muscle tension at 0.1 Hz on cardiovascular resonance and the baroreflex. *Biological Psychology, 81*(1), 24–30.

Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. *Applied Psychophysiology and Biofeedback, 25,* 177–191.

Lehrer, P. M., Vaschillo, E. G., Vaschillo, B., Lu, S.-E., Eckberg, D. L., Edelberg, R., et al. (2003). Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. *Psychosomatic Medicine, 65*(5), 796–805.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Lu, S. E., Scardella, A., Siddique, M., et al. (2004). Biofeedback treatment for asthma. *Chest, 126*(2), 352–361.

Lehrer, P. M., Vaschillo, E. G., & Vidali, V. (2020). Heart rate and breathing are not always in phase during resonance frequency breathing. *Applied Psychophysiology and Biofeedback. 45*(3), 145–152.

Lehrer, P., Vaschillo, B., Zucker, T., Graves, J., Katsamanis, M., Aviles, M., et al. (2013). Protocol for heart rate variability biofeedback training. *Biofeedback, 41,* 98–109.

Leor-Librach, R. J., Bobrovsky, B. Z., Eliash, S., & Kaplinsky, E. (2002). A common origin of the very low frequency heart rate and blood pressure variability: A new insight into an old debate. *Autonomic Neuroscience: Basic and Clinical, 96*(2), 140–148.

Leor-Librach, R. J., Eliash, S., Kaplinsky, E., & Bobrovsky, B. Z. (2003). Very low-frequency heart rate variability wave amplitude and sympathetic stimulation: Characterization and modeling. *IEEE Transactions on Biomedical Engineering, 50*(7), 797–803.

Liao, D., Barnes, R. W., Chambless, L. E., Simpson, R. J., Sorlie, P., & Heiss, G. (1995). Age, race, and sex differences in autonomic cardiac function measured by spectral analysis of heart rate variability: The ARIC study. *American Journal of Cardiology, 76,* 906–912.

Licht, C. M., de Geus, E. J., van Dyck, R., & Penninx, B. W. (2009). Association between anxiety disorders and heart rate variability in the Netherlands Study of Depression and Anxiety (NESDA). *Psychosomatic Medicine, 71*(5), 508–518.

Lin, G., Xiang, Q., Fu, X., Wang, S., Wang, S., Chen, S., et al. (2012). Heart rate variability biofeedback decreases blood pressure in prehypertensive subjects by improving autonomic function and baroreflex. *Journal of Alternative and Complementary Medicine, 18*(2), 143–152.

Lin, I. M., Fan, S. Y., Lu, H. C., Lin, T. H., Chu, C. S., Kuo, H. F., et al. (2015). Randomized controlled trial of heart rate variability biofeedback in cardiac autonomic and hostility among patients with coronary artery disease. *Behaviour Research and Therapy, 70,* 38–46.

Loguidice, M. J., Schutt, R. C., Horton, J. W., Minei, J. P., & Keeley, E. C. (2016). Heart rate variability as a predictor of death in burn patients. *Journal of Burn Care and Research, 37*(3), e227–e233.

Longin, E., Reinhard, J., von Buch, C., Gerstner, T., Lenz, T., & Konig, S. (2008). Autonomic function in children and adolescents with neurocardiogenic syncope. *Pediatric Cardiology, 29*(4), 763–770.

Lucini, D., Milani, R. V., Costantino, G., Lavie, C. J., Porta, A., & Pagani, M. (2002). Effects of cardiac rehabilitation and exercise training on autonomic regulation in patients with coronary artery disease. *American Heart Journal, 143,* 977–983.

Lundvall, J., & Edfeldt, H. (1994). Very large range of baroreflex sympathetic control of vascular resistance in human skeletal muscle and skin. *Journal of Applied Physiology, 76*(1), 204–211.

MacKinnon, S., Gevirtz, R., McCraty, R., & Brown, M. (2013). Utilizing heart beat evoked potentials to identify cardiac regulation of vagal afferents during emotion and resonant breathing. *Applied Psychophysiology and Biofeedback, 38,* 241–255.

Maheshwari, A., Norby, F. L., Soliman, E. Z., Adabag, S., Whitsel, E. A., Alonso, A., et al. (2016). Low heart rate variability in a 2-minute electrocardiogram recording is associated with an increased risk of sudden cardiac death in the general population: The Atherosclerosis Risk in Communities Study. *PLOS ONE, 11*(8), e0161648.

Mancia, G., Ludbrook, J., Ferrari, A., Gregorini, L., & Zanchetti, A. (1978). Baroreceptor reflexes in human hypertension. *Circulation Research, 43,* 170–177.

Masi, C. M., Hawkley, L. C., Rickett, E. M., & Cacioppo, J. T. (2007). Respiratory sinus arrhythmia and diseases of aging: Obesity, diabetes mellitus, and hypertension. *Biological Psychology, 74*(2), 212–223.

Mather, M. (2019, September). *Can we improve brain mechanisms of emotion regulation by* *increasing heart rate variability?* Paper presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

Mather, M., & Thayer, J. (2018). How heart rate variability affects emotion regulation brain networks. *Current Opinion in Behavioral Sciences, 19,* 98–104.

Matsuguchi, H., & Schmid, P. G. (1982). Pressor response to vasopressin and impaired baroreflex function in DOC-salt hypertension. *American Journal of Physiology, 242*(1), H44–H49.

Matveev, M., & Prokopova, R. (2002). Diagnostic value of the RR-variability indicators for mild hypertension. *Physiological Measurement, 23,* 671–682.

McCormick, C. E. B., Sheinkopf, S. J., Levine, T. P., LaGasse, L. L., Tronick, E., & Lester, B. L. (2018). Diminished respiratory sinus arrhythmia response in infants later diagnosed with autism spectrum disorder. *Autism Research, 11*(5), 726–731.

McCraty, R., Atkinson, M., & Tomasino, D. (2003). Impact of a workplace stress reduction program on blood pressure and emotional health in hypertensive employees. *Journal of Complementary and Alternative Medicine, 9,* 355–369.

Meier, N. F., & Welch, A. S. (2016). Walking versus biofeedback: A comparison of acute interventions for stressed students. *Anxiety Stress Coping, 29*(5), 463–478.

Meule, A., Freund, R., Skirde, A. K., Vögele, C., & Kübler, A. (2012). Heart rate variability biofeedback reduces food cravings in high food cravers. *Applied Psychophysiology Biofeedback, 37*(4), 241–251.

Mikosch, P., Hadrawa, T., Laubreiter, K., Brandl, J., Pilz, J., Stettner, H., et al. (2010). Effectiveness of respiratory-sinus-arrhythmia biofeedback on state-anxiety in patients undergoing coronary angiography. *Journal of Advances in Nursing, 66*(5), 1101–1110.

Miller, B. D., & Wood, B. L. (1994). Psychophysiologic reactivity in asthmatic children: A cholinergically mediated confluence of pathways. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*(9), 1236–1245.

Miller, B. D., & Wood, B. L. (1997). Influence of specific emotional states on autonomic reactivity and pulmonary function in asthmatic children. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(5), 669–677.

Miller, B. D., Wood, B. D., Lim, J., Ballow, M., & Hsu, C. (2009). Depressed children with asthma evidence increased airway resistance: "Vagal bias" as a mechanism? *Journal of Allergy and Clinical Immunology, 124*(1), 66–73.

Miranda, C. M., & Silva, R. (2016). Analysis of heart rate variability before and during tilt test in patients with cardioinhibitory vasovagal syncope. *Arquivos Brasileiros de Cardiologia, 107*(6), 568–575.

Moak, J. P., Goldstein, D. S., Eldadah, B. A., Saleem, A., Holmes, C., Pechnik, S., et al. (2007). Supine low-frequency power of heart rate variability reflects baroreflex function, not cardiac sympathetic innervation [Comment]. *Heart Rhythm, 4*(12), 1523–1529.

Morguet, A., & Springer, H. J. (1981). Microcomputer-based measurement of beat-to-beat intervals and analysis of heart rate variability. *Medical Progress through Technology, 8,* 77–82.

Mullen, R., Faull, A., Jones, E. S., & Kingston, K. (2012). Attentional focus and performance anxiety: Effects on simulated race-driving performance and heart rate variability. *Frontiers in Psychology, 3,* 426.

Munafo, M., Patron, E., & Palomba, D. (2016). Improving managers' psychophysical well-being: Effectiveness of respiratory sinus arrhythmia biofeedback. *Applied Psychophysiology and Biofeedback, 41*(2), 129–139.

Murphy, J. A. W. (2009). Comparison of relaxation techniques for group cognitive behavioral therapy for generalized anxiety disorder. *Dissertation Abstracts International, 70*(3-B), 1952.

Myers, C. W., Cohen, M. A., Eckberg, D. L., & Taylor, J. A. (2001). A model for the genesis of arterial pressure Mayer waves from heart rate and sympathetic activity. *Autonomic Neuroscience: Basic and Clinical, 91,* 62–75.

National Heart, Lung, & Blood Institute. (2007). *Expert Panel Report 3: Guidelines for the diagnosis and management of asthma: Update on selected topics* (NIH Publication No. 08-5846). Washington, DC: U.S. Department of Health and Human Services.

Nolan, R. P., Floras, J. S., Ahmed, L., Harvey, P. J., Hiscock, N., Hendrickx, H., et al. (2012). Behavioural modification of the cholinergic anti-inflammatory response to C-reactive protein in patients with hypertension. *Journal of Internal Medicine, 272*(2), 161–169.

O'Brien, I. A., O'Hare, P., & Corrall, R. J. (1986). Heart rate variability in healthy subjects: Effect of age and the derivation of normal ranges for tests of autonomic function. *British Heart Journal, 55,* 348–354.

Panza, J. A., Epstein, S. E., & Quyyumi, A. A. (1991). Circadian variation in vascular tone and its relation to alpha-sympathetic vasoconstrictor activity. *New England Journal of Medicine, 325*(14), 986–990.

Pardo, Y., Merz, C. N., Velasquez, I., Paul-Labrador, M., Agarwala, A., & Peter, C. T. (2000). Exercise conditioning and heart rate variability: Evidence of a threshold effect. *Clinical Cardiology, 23,* 615–620.

Parshuram, C. S., Dhanani, S., Kirsh, J. A., & Cox, P. N. (2004). Fellowship training, workload, fatigue and physical stress: A prospective observational study. *Canadian Medical Association Journal, 170*(6), 965–970.

Pascoe, M. C., Thompson, D. R., & Ski, C. F. (2017). Yoga, mindfulness-based stress reduction and stress-related physiological measures: A meta-analysis. *Psychoneuroendocrinology, 86,* 152–168.

Patriquin, M. A., Hartwig, E. M., Friedman, B. H., Porges, S. W., & Scarpa, A. (2019). Autonomic response in autism spectrum disorder: Relationship to social and cognitive functioning. *Biological Psychology, 145,* 185–197.

Patron, E., Benvenuti, S. M., Favretto, G., Valfre, C., Bonfa, C., Gasparotto, R., et al. (2013). Biofeedback assisted control of respiratory sinus arrhythmia as a biobehavioral intervention for depressive symptoms in patients after cardiac surgery: A preliminary study. *Applied Psychophysiology and Biofeedback, 38*(1), 1–9.

Paul, M., & Garg, K. (2012). The effect of heart rate variability biofeedback on performance psychology of basketball players. *Applied Psychophysiology and Biofeedback, 37*(2), 131–144.

Paul, M., Garg, K., & Sandhu, J. S. (2012). Role of biofeedback in optimizing psychomotor performance in sports. *Asian Journal of Sports Medicine, 3*(1), 29–40.

Pavlov, V. A., & Tracey, K. J. (2012). The vagus nerve and the inflammatory reflex: Linking immunity and metabolism. *Nature Reviews Endocrinology, 8*(12), 743–754.

Peltola, M. A. (2012). Role of editing of R–R intervals in the analysis of heart rate variability. *Frontiers of Physiology, 23*(3),148.

Penzlin, A. I., Siepmann, T., Illigens, B. M. W., Weidner, K., & Siepmann, M. (2015). Heart rate variability biofeedback in patients with alcohol dependence: A randomized controlled study. *Neuropsychiatric Disease and Treatment, 11,* 2619–2627.

Piccirillo, G., Naso, C., Moise, A., Lionetti, M., Nocco, M., Di Carlo, S., et al. (2004). Heart rate and blood pressure variability in subjects with vasovagal syncope. *Clinical Science, 107,* 55–61.

Pinna, G. D., Maestri, R., La Rovere, M. T., Gobbi, E., & Fanfulla, F. (2006). Effect of paced breathing on ventilatory and cardiovascular variability parameters during short-term investigations of autonomic function. *American Journal of Physiology: Heart and Circulatory Physiology, 290*(1), H424–H433.

Pitzalis, M. V., Passantino, A., Massari, F., Forleo, C., Balducci, C., Santoro, G., et al. (1999). Diastolic dysfunction and baroreflex sensitivity in hypertension. *Hypertension, 33,* 1141–1145.

Pollatos, O., Kirsch, W., & Schandry, R. (2005). Brain structures involved in interoceptive awareness and cardioafferent signal processing: Adipole source localization study. *Human Brain Mapping, 26,* 54–64.

Porges, S. W. (1995). Cardiac vagal tone: A physiological index of stress. *Neuroscience and Biobehavioral Reviews, 19,* 225–233.

Porges, S. (2011). *The polyvagal theory.* New York: Norton.

Prinsloo, G. E., Derman, W. E., Lambert, M. I., & Rauch, L. (2013). The effect of a single session of short duration biofeedback-induced deep breathing on measures of heart rate variability during laboratory-induced cognitive stress: A pilot study. *Applied Psychophysiology and Biofeedback, 38*(2), 81–90.

Pyetan, E., & Akselrod, S. (2003). Do the high-frequency indexes of HRV provide a faithful assessment of cardiac vagal tone?: A critical theoretical evaluation. *IEEE Transactions on Biomedical Engineering, 50*(6), 777–783.

Rahman, F., Pechnik, S., Gross, D., Sewell, L., & Goldstein, D. S. (2011). Low frequency power of heart rate variability reflects baroreflex function, not cardiac sympathetic innervation. *Clinical Autonomic Research, 21*(3), 133–141.

Rau, H., Brody, S., Larbig, W., Pauli, P., Vohringer, M., Harsch, B., et al. (1994). Effects of PRES baroreceptor stimulation on thermal and mechanical pain threshold in borderline hypertensives and normotensives. *Psychophysiology, 31,* 480–485.

Rau, H., Furedy, J. J., & Elbert, T. (1996). PRES- and orthostatic-induced heart rate changes as markers of labile hypertension: Magnitude and reliability measures. *Biological Psychology, 42,* 105–115.

Ray, J. M., Pyne, J. M., & Gevirtz, R. N. (2017).

Alcohol use disorder moderates the effect of age on heart rate variability in veterans with posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 205*(10), 793–800.

Raymond, J., Sajid, I., Parkinson, L. A., & Gruzelier, J. H. (2005). Biofeedback and dance performance: A preliminary investigation. *Applied Psychophysiology and Biofeedback, 30*(1), 64–73.

Reineke, A. (2008). The effects of heart rate variability biofeedback in reducing blood pressure for the treatment of essential hypertension. *Dissertation Abstracts International, 68*(7-B), 4880.

Rene, R. (2008). The efficacy of a portable HRV feedback device in conjunction with mental health treatment of clients with major depressive disorder enrolled in a county welfare-to-work program. *Dissertation Abstracts International, 69*(3-B), 2000.

Reyes del Paso, G., Langewitz, W., Mulder, L. J. M., van Roon, A., & Duschek, S. (2013). The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: A review with emphasis on a reanalysis of previous studies. *Psychophysiology, 50*(5), 477–487.

Ritz, T., Meuret, A. E., Wilhelm, F. H., & Roth, W. T. (2009). Changes in pCO2, symptoms, and lung function of asthma patients during capnometry-assisted breathing training. *Applied Psychophysiology and Biofeedback, 34*(1), 1–6.

Robbe, H. W., Mulder, L. J., Ruddel, H., Langewitz, W. A., Veldman, J. B., & Mulder, G. (1987). Assessment of baroreceptor reflex sensitivity by means of spectral analysis. *Hypertension, 10,* 538–543.

Robertson, D., Hollister, A. S., Biaggioni, I., Netterville, J. L., Mosqueda-Garcia, R., & Robertson, R. M. (1993). The diagnosis and treatment of baroreflex failure. *New England Journal of Medicine, 329,* 1449–1455.

Rodionov, I. M., Koshelev, V. B., Meshcheryakova, O. D., & Stavskaya, O. N. (1981). Efficacy of sympathetic influences as related to initial vascular tonicity. *American Journal of Physiology, 241*(5), H691–H699.

Rodriguez-Roisin, R. (1990). Gas exchange abnormalities in asthma. *Lung, 168*(Suppl.), 599–605.

Rother, M., Zwiener, U., Eiselt, M., Witte, H., Zwacka, G., & Frenzel, J. (1987). Differentiation of healthy newborns and newborns-at-risk by spectral analysis of heart rate fluctuations and respiratory movements. *Early Human Development, 15,* 349–363.

Routledge, F. S., Campbell, T. S., McFetridge-Durdle, J. A., & Bacon, S. L. (2010). Improvements in heart rate variability with exercise therapy. *Canadian Journal of Cardiology, 26*(6), 303–312.

Rozen, G., Kobo, R., Beinart, R., Feldman, S., Sapunar, M., Luria, D., et al. (2013). Multipole analysis of heart rate variability as a predictor of imminent ventricular arrhythmias in ICD patients. *Pacing and Clinical Electrophysiology, 36*(11), 1342–1347.

Rudas, L., Crossman, A. A., Morillo, C. A., Halliwill, J. R., Tahvanainen, K. U., Kuusela, T. A., et al. (1999). Human sympathetic and vagal baroreflex responses to sequential nitroprusside and phenylephrine. *American Journal of Physiology, 276,* H1691–H1698.

Rudolph, A. J., Vallbona, C., & Desmond, M. M. (1965). Cardiodynamic studies in the newborn: 3. Heart rate patterns in infants with idiopathic respiratory distress syndrome. *Pediatrics, 36,* 551–559.

Rupprecht, S., Finn, S., Hoyer, D., Guenther, A., Witte, O. W., Schultze, T., et al. (2019). Association between systemic inflammation, carotid arteriosclerosis, and autonomic dysfunction. *Translational Stroke Research, 11*(1), 50–59.

Russoniello, C. V., Zhirnov, Y. N., Pougatchev, V. I., & Gribkov, E. N. (2013). Heart rate variability and biological age: Implications for health and gaming. *Cyberpsychology, Behavior, and Social Networking, 16*(4), 302–308.

Salameh, E., Kadri, Z., Neemtallah, R., Azar, R., Badaoui, G., Jaoude, S. A., et al. (2007). [Heart rate variability and vasovagal syncope]. *Annales de Cardiologie et d'Angeiologie, 56*(2), 88–91.

Sargunaraj, D., Lehrer, P. M., Hochron, S. M., Rausch, L., Edelberg, R., & Porges, S. (1996). Cardiac rhythm effects of .125 hz paced breathing through a resistive load: Implications for paced breathing therapy and Porges' polyvagal theory. *Biofeedback and Self-Regulation, 21,* 131–147.

Sarlo, M., Palomba, D., Angrilli, A., & Stegagno, L. (2002). Blood phobia and spider phobia: Two specific phobias with different autonomic cardiac modulations. *Biological Psychology, 60,* 91–108.

Schalow, G., Blanc, Y., Jeltsch, W., & Zach, G. A. (1996). Electromyographic identification of spinal oscillator patterns and recouplings in a patient with incomplete spinal cord lesion: Oscillator formation training as a method to improve motor activities. *General Physiology and Biophysics, 15*(Suppl. 1), 121–220.

Schandry, R., Sparrer, B., & Weitkunat, R. (1986). From the heart to the brain: A study of heartbeat contingent scalp potentials. *International Journal of Neuroscience, 30,* 261–275.

Schauer, M., & Elbert, T. (2010). Dissociation following traumatic stress: Etiology and treatment. *Zeitschrift fur Psychologie/Journal of Psychology, 218*(2), 109–127.

Schein, M. H., Gavish, B., Herz, M., Rosner-Kahana, D., Naveh, P., Knishkowy, B., et al. (2001). Treating hypertension with a device that slows and regularizes breathing: A randomized, double-blind controlled study. *Journal of Human Hypertension, 15,* 271–278.

Schnettler, W. T., Goldberger, A. L., Ralston, S. J., & Costa, M. (2016). Complexity analysis of fetal heart rate preceding intrauterine demise. *European Journal of Obstetrics, Gynecology, and Reproductive Biology, 203,* 286–290.

Schubert, C., Lambertz, M., Nelesen, R. A., Bardwell, W., Choi, J. B., & Dimsdale, J. E. (2009). Effects of stress on heart rate complexity—a comparison between short-term and chronic stress. *Biological Psychology, 80*(3), 325–332.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. Frontiers in Public Health. Retrieved from *www.frontiersin.org/articles/10.3389/fpubh.2017.00258/full.*

Shinba, T. (2017). Major depressive disorder and generalized anxiety disorder show different autonomic dysregulations revealed by heart-rate variability analysis in first-onset drug-naive patients without comorbidity. *Psychiatry and Clinical Neurosciences, 71*(2), 135–145.

Sidorov, I. A., & Vasilevskii, N. N. (1995). The physiological problems of biofeedback control by the pulse rate. *Neuroscience and Behavioral Physiology, 25,* 252–256.

Siepmann, M., Hennig, U. D., Siepmann, T., Nitzsche, K., Muck-Weymann, M., Petrowski, K., et al. (2014). The effects of heart rate variability biofeedback in patients with preterm labour. *Applied Psychophysiology and Biofeedback, 39*(1), 27–35.

Sinnecker, D., Dommasch, M., Steger, A., Berkefeld, A., Hoppmann, P., Muller, A., et al. (2016). Expiration-triggered sinus arrhythmia predicts outcome in survivors of acute myocardial infarction. *Journal of the American College of Cardiology, 67*(19), 2213–2220.

Smith, M. L., Ellenbogen, K. A., Beightol, L. A., & Eckberg, D. L. (1991). Sympathetic neural responses to induced ventricular tachycardia. *Journal of the American College of Cardiology, 18,* 1015–1024.

Soer, R., Vos, D., Hofstra, B., & Reneman, M. F. (2014). Heart coherence training combined with back school in patients with chronic non-specific low back pain: First pragmatic clinical results. *Applied Psychophysiology and Biofeedback, 39*(3–4), 259–267.

Song, H.-S., & Lehrer, P. M. (2003). The effects of specific respiratory rates on heart rate and heart rate variability. *Applied Psychophysiology and Biofeedback, 28*(1), 13–23.

Spicuzza, L., Gabutti, A., Porta, C., Montano, N., & Bernardi, L. (2000). Yoga and chemoreflex response to hypoxia and hypercapnia. *Lancet, 356,* 1495–1496.

Stamler, J. S., Goldman, M. E., Gomes, J., Matza, D., & Horowitz, S. F. (1992). The effect of stress and fatigue on cardiac rhythm in medical interns. *Journal of Electrocardiology, 25*(4), 333–338.

Steffen, P. R., Austin, T., DeBarros, A., & Brown, T. (2017). The impact of resonance frequency breathing on measures of heart rate variability, blood pressure, and mood. *Frontiers in Public Health, 5,* 222.

Stepp, N. (2012). Anticipation and learning. In N. M. Seel (Ed.), *Encyclopedia of the sciences of learning.* Boston: Springer.

Strack, B., Gevirtz, R., & Sime, W. (2003). Effect of heart rate variability (HRV) biofeedback on batting performance in baseball. *Dissertation Abstracts International, 64*(3-B), 1540.

Stromberg, S. E., Russell, M. E., & Carlson, C. R. (2015). Diaphragmatic breathing and its effectiveness for the management of motion sickness. *Aerospace Medicine and Human Performance, 86*(5), 452–457.

Studer, R., Danuser, B., Hildebrandt, H., Arial, M., & Gomez, P. (2011). Hyperventilation complaints in music performance anxiety among classical music students. *Journal of Psychosomatic Research, 70*(6), 557–564.

Sturgeon, J. A., Yeung, E. W., & Zautra, A. J. (2014). Respiratory sinus arrhythmia: A marker of resilience to pain induction. *International Journal of Behavioral Medicine, 21*(6), 961–965.

Sutarto, A. P., Wahab, M. N., & Zin, N. M. (2012). Resonant breathing biofeedback training for stress reduction among manufacturing operators. *International Journal of Occupational Safety and Ergonomics, 18*(4), 549–561.

Sutarto, A. P., Wahab, M. N., & Zin, N. M. (2013). Effect of biofeedback training on operator's cognitive performance. *Work, 44*(2), 231–243.

Swanson, K. S., Gevirtz, R. N., Brown, M., Spira, J., Guarneri, E., & Stoletniy, L. (2009). The effect of biofeedback on function in patients with heart failure. *Applied Psychophysiology and Biofeedback, 34*(2), 71–91.

Szulczewski, M. T., & Rynkiewicz, A. (2018). The effects of breathing at a frequency of 0.1 Hz on affective state, the cardiovascular system, and adequacy of ventilation. *Psychophysiology, 55*(12), e13221.

Tacoy, G., Acikgoz, K., Kocaman, S. A., Ozdemir, M., & Cengel, A. (2010). Is there a relationship between obesity, heart rate variability and inflammatory parameters in heart failure? *Journal of Cardiovascular Medicine, 11*(2), 118–124.

Tan, G., Dao, T. K., Farmer, L., Sutherland, R. J., & Gevirtz, R. (2011). Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): A pilot study. *Applied Psychophysiology and Biofeedback, 36*(1), 27–35.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. *Circulation, 93,* 1043–1065.

Taylor, J. A., Myers, C. W., Halliwill, J. R., Seidel, H., & Eckberg, D. L. (2001). Sympathetic restraint of respiratory sinus arrhythmia: Implications for vagal–cardiac tone assessment in humans. *American Journal of Physiology: Heart and Circulatory Physiology, 280,* H2804–H2814.

Thayer, J. F., Nabors-Oberg, R., & Sollers, J. J. (1997). Thermoregulation and cardiac variability: A time-frequency analysis. *Biomedical Sciences Instrumentation, 34,* 252–256.

Thayer, J. F., Yonezawa, Y., & Sollers, J. J., III. (2000). A system for the ambulatory recording and analysis of nonlinear heart rate dynamics. *Biomedical Sciences Instrumentation, 36,* 295–299.

Thurber, M. R. (2007). Effects of heart-rate variability biofeedback training and emotional regulation on music performance anxiety in university students. *Dissertation Abstracts International, 68*(3-A), 889.

Tracey, K. (2002). The inflammatory reflex. *Nature, 420,* 19–26.

Traina, M., Cataldo, A., Galullo, F., & Russo, G. (2011). Effects of anxiety due to mental stress on heart rate variability in healthy subjects. *Minerva Psichiatrica, 52*(4), 227–231.

Tsai, H., Kuo, T. B., Lee, G.-S., & Yang, C. C. (2015). Efficacy of paced breathing for insomnia: Enhances vagal activity and improves sleep quality. *Psychophysiology, 52*(3), 388–396.

Tyagi, A., Cohen, M., Reece, J., Telles, S., & Jones, L. (2016). Heart rate variability, flow, mood and mental stress during yoga practices in yoga practitioners, non-yoga practitioners and people with metabolic syndrome. *Applied Psychophysiology and Biofeedback, 41*(4), 381–393.

van der Zwan, J. E., de Vente, W., Huizink, A. C., Bogels, S. M., & de Bruin, E. I. (2015). Physical activity, mindfulness meditation, or heart rate variability biofeedback for stress reduction: A randomized controlled trial. *Applied Psychophysiology and Biofeedback, 40*(4), 257–268.

Van Diest, I., De Peuter, S., Devriese, S., Wellens, E., Van de Woestijne, K. P., & Van den Bergh, O. (2005). Imagined risk of suffocation as a trigger for hyperventilation. *Psychosomatic Medicine, 67*(5), 813–819.

Van Diest, I., Verstappen, K., Aubert, A. E., Widjaja, D., Vansteenwegen, D., & Vlemincx, E. (2014). Inhalation/exhalation ratio modulates the effect of slow breathing on heart rate variability and relaxation. *Applied Psychophysiology and Biofeedback, 39*(3–4), 171–180.

Van Dixhoorn, J. V., & White, A. (2005). Relaxation therapy for rehabilitation and prevention in ischaemic heart disease: A systematic review and meta-analysis. *European Journal of Cardiovascular Prevention and Rehabilitation, 12,* 193–202.

Vaschillo, E. G., Bates, M. E., Vaschillo, B., Lehrer, P., Udo, T., Mun, E. Y., et al. (2008). Heart rate variability response to alcohol, placebo, and emotional picture cue challenges: Effects of 0.1-Hz stimulation. *Psychophysiology, 45*(5), 847–858.

Vaschillo, E. G., Lehrer, P. M., Rishe, N., & Konstantinov, M. (2002). Heart rate variability biofeedback as a method for assessing baroreflex function: A preliminary study of resonance in the cardiovascular system. *Applied Psychophysiology and Biofeedback, 27*(1), 1–27.

Vaschillo, E., Vaschillo, B., Buckman, J., & Bates, M. (2019, February). *New approach for brain stimulation.* Paper presented at the Third International Brain Stimulation Conference, Vancouver, British Columbia, Canada.

Vaschillo, E. G., Vaschillo, B., Buckman, J. F., Heiss, S., Singh, G., & Bates, M. E. (2018).

Early signs of cardiovascular dysregulation in young adult binge drinkers. *Psychophysiology, 55*(5), e13036.

Vaschillo, E. G., Vaschillo, B., Buckman, J. F., Pandina, R. J., & Bates, M. E. (2011). The investigation and clinical significance of resonance in the heart rate and vascular tone baroreflexes. In A. Fred, J. Filipe, & H. Gamboa (Eds.), *Biomedical engineering systems and technologies: Communications in computer and information science* (pp. 224–237). Heidelberg, Germany: Springer.

Vaschillo, E. G., Vaschillo, B., Buckman, J. F., Pandina, R. J., & Bates, M. E. (2012). Measurement of vascular tone and stroke volume baroreflex gain. *Psychophysiology, 49*(2), 193–197.

Vaschillo, E., Vaschillo, B., & Lehrer, P. (2004). Heartbeat synchronizes with respiratory rhythm only under specific circumstances. *Chest, 126,* 1385–1386.

Vaschillo, E. G., Vaschillo, B., & Lehrer, P. M. (2006). Characteristics of resonance in heart rate variability stimulated by biofeedback. *Applied Psychophysiology and Biofeedback, 31*(2), 129–142.

Vaschillo, E. G., Zingerman, A. M., Konstantinov, M. A., & Menitsky, D. N. (1983). Research of the resonance characteristics for cardiovascular system. *Human Physiology, 9,* 257–265.

Vasilevskii, N. N., Sidorov, I. A., & Suvorov, N. B. (1993). [Role of biorhythmologic processes in adaptation mechanisms and correction of regulatory dysfunctions]. *Fiziologii Cheloveka, 19,* 91–98.

Vershuta, E. V., Pevzner, A. V., Ermishkin, V. V., Kuchinskaia, E. A., Al'bitskaia, K. V., Kheimets, G. I., et al. (2009). [Spectral indices of heart rate variability in patients with vasovagal syncopes according to evidence of 5-min ECG]. *Terapevticheskii Arkhiv, 81*(4), 17–21.

Viana, A. G., Palmer, C. A., Zvolensky, M. J., Alfano, C. A., Dixon, L. J., & Raines, E. M. (2017). Children's behavioral inhibition and anxiety disorder symptom severity: The role of individual differences in respiratory sinus arrhythmia. *Behaviour Research and Therapy, 93,* 38–46.

Virtanen, R., Jula, A., Salminen, J. K., Voipio-Pulkki, L. M., Helenius, H., Kuusela, T., et al. (2003). Anxiety and hostility are associated with reduced baroreflex sensitivity and increased beat-to-beat blood pressure variability. *Psychosomatic Medicine, 65*(5), 751–756.

Visnovcova, Z., Mestanik, M., Javorka, M., Mokra, D., Gala, M., Jurko, A., et al. (2014). Complexity and time asymmetry of heart rate variability are altered in acute mental stress. *Physiological Measurement, 35*(7), 1319–1334.

Wahbeh, H., Goodrich, E., Goy, E., & Oken, B. S. (2016). Mechanistic pathways of mindfulness meditation in combat veterans with posttraumatic stress disorder. *Journal of Clinical Psychology, 72*(4), 365–383.

Wang, Y., Zhao, X., O'Neil, A., Turner, A., Liu, X., & Berk, M. (2013). Altered cardiac autonomic nervous function in depression. *BMC Psychiatry, 13,* 187.

Watanabe, S., Amiya, E., Watanabe, M., Takata, M., Ozeki, A., Watanabe, A., et al. (2013). Simultaneous heart rate variability monitoring enhances the predictive value of flow-mediated dilation in ischemic heart disease. *Circulation Journal, 77*(4), 1018–1025.

Watkins, L. L., Grossman, P., Krishnan, R., & Blumenthal, J. A. (1999). Anxiety reduces baroreflex cardiac control in older adults with major depression. *Psychosomatic Medicine, 61*(3), 334–340.

Williams, D. P., Feeling, N. R., Hill, L. K., Spangler, D. P., Koenig, J., & Thayer, J. F. (2017). Resting heart rate variability, facets of rumination and trait anxiety: Implications for the perseverative cognition hypothesis. *Frontiers in Human Neuroscience, 11,* 520.

Williams, D. P., Koenig, J., Carnevali, L., Sgoifo, A., Jarczok, M. N., Sternberg, E. M., et al. (2019). Heart rate variability and inflammation: A meta-analysis of human studies. *Brain, Behavior, and Immunity, 80,* 219–226.

Wu, L., Jiang, Z., Li, C., & Shu, M. (2014). Prediction of heart rate variability on cardiac sudden death in heart failure patients: A systematic review. *International Journal of Cardiology, 174*(3), 857–860.

Xie, L., Li, M., Dang, S., Li, C., Wang, X., Liu, B., et al. (2018). Impaired cardiorespiratory coupling in young normotensives with a family history of hypertension. *Journal of Hypertension, 36*(11), 2157–2167.

Yasuma, F., & Hayano, J. (2004). Respiratory sinus arrhythmia: Why does the heartbeat synchronize with respiratory rhythm? *Chest, 125*(2), 683–690.

Yeh, T.-C., Kao, L.-C., Tzeng, N.-S., Kuo, T. B. J., Huang, S.-Y., Chang, C.-C., et al. (2016). Heart rate variability in major depressive disorder and after antidepressant treatment with agomelatine and paroxetine: Findings from the Taiwan Study of Depression and Anxiety (TAISDA). *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 64,* 60–67.

Yeragani, V. K., Balon, R., Pohl, R., & Ramesh, C. (1995). Depression and heart rate variability. *Biological Psychiatry, 38,* 768–770.

Yetwin, A. K. (2012). Heart rate variability biofeedback therapy for children and adolescents with chronic pain. *Dissertation Abstracts International, 72*(12-B), 7704.

Yetwin, A., Marks, K., Bell, T., & Gold, J. (2012). Heart rate variability biofeedback therapy for children and adolescents with chronic pain [Abstract]. *Journal of Pain, 13*(4, Suppl.).

Yi, H. T., Hsieh, Y. C., Wu, T. J., Huang, J. L., Lin, W. W., Liang, K. W., et al. (2014). Heart rate variability parameters and ventricular arrhythmia correlate with pulmonary arterial pressure in adult patients with idiopathic pulmonary arterial hypertension. *Heart and Lung, 43*(6), 534–540.

Young, I. H., & Bye, P. T. (2011). Gas exchange in disease: Asthma, chronic obstructive pulmonary disease, cystic fibrosis, and interstitial lung disease. *Comprehensive Physiology, 1*(2), 663–697.

Zamuner, A. R., Forti, M., Andrade, C. P., Avila, M. A., & da Silva, E. (2016). Respiratory sinus arrhythmia and its association with pain in women with fibromyalgia syndrome. *Pain Practice, 16*(6), 704–711.

Zdrenghea, D., Sitar-Taut, A., & Pop, D. (2007). Comparison between heart rate variability and recovery in ischemic patients. *Romanian Journal of Internal Medicine, 45*(2), 171–175.

Zhao, G., Hintze, T. H., & Kaley, G. (1996). Neural regulation of coronary vascular resistance: Role of nitric oxide in reflex cholinergic coronary vasodilation in normal and pathophysiologic states. In M. Karmazyn (Ed.), *Myocardial ischemia: Mechanisms, reperfusion, protection* (pp. 1–19). Basel, Switzerland: Birkhauser.

Zucker, T. L., Samuelson, K. W., Muench, F., Greenberg, M. A., & Gevirtz, R. N. (2009). The effects of respiratory sinus arrhythmia biofeedback on heart rate variability and posttraumatic stress disorder symptoms: A pilot study. *Applied Psychophysiology and Biofeedback, 34*(2), 135–143.

CHAPTER 11

Capnometry-Assisted Respiratory Training

Principles and Findings

Alicia E. Meuret
Thomas Ritz

HISTORY OF THE METHOD

Hypoventilation therapy is aimed at reversing hypocapnia. Acute hypocapnia, or hyperventilation, is typically caused by breathing more than the organism's metabolic demand by breathing faster, deeper, or both, leading to low levels of arterial PCO_2 and disturbances of the acid–base balance (high pH levels, acute respiratory alkalosis). Adverse effects of hypocapnic breathing have long been recognized and have been linked experimentally to organ injury and several organic illnesses and mental disorders (Laffey & Kavanagh, 1999; Marhong & Fan, 2014). The clinical literature provides many observations of patients presenting with a miscellany of complaints for which no organic origin is apparent. Typically, patients feel symptoms of dyspnea, breathlessness, dizziness, chest pain, paresthesias, fatigue, and palpitations (Tavel, 1990). Hypocapnia and disturbance of the acid–base balance were proposed to underlie the origin of these symptoms (Lum, 1987), and clinical reports suggested a considerable suffering and reduced quality of life in these patients (Fried, 1993). First introduced by Kerr, Gliebe, and Dalton (1938), *hyperventilation syndrome* (HVS) became a common designation for this symptom constellation. Other terms used in the literature to describe such symptoms have been *dysfunctional breathing, overbreathing, behavioral breathlessness, neurocirculatory asthenia,* and *psychogenic dyspnea* (Howell, 1997; Jones, Harvey, Marston, & O'Connell, 2013; Thomas, McKinley, Freeman, & Foy, 2001). At least 5–10% of general medical outpatients have been thought to suffer from this condition (Magarian, 1982), but substantial controversy and lack of systematic research have been in the way of elevating HVS to a diagnostic status (Bass, 1997).

In acute hyperventilation, PCO_2 levels of 35 mm Hg or lower have been determined as the hypocapnic range (Oakes & Jones, 2017), but sustained or repeatedly low levels of

< 30 mm Hg are seen as a more conservative criterion for hyperventilation (Bass & Gardner, 1985). However, in chronic hyperventilation, pH levels can be normal due to buffer mechanisms in blood and kidneys, while PCO_2 remains low at the same time. Clinically relevant hyperventilation may exist without evidence of comorbid illness (chronic idiopathic hyperventilation; Jack et al., 2004) or in conjunction with a range of psychological or somatic conditions (Gardner, 1996). Particular attention has been directed to hyperventilation in the context of panic disorder and asthma. These two conditions are discussed in greater detail in this chapter because they have been the main areas of application of hypoventilation training.

While the benefits of reversing hypocapnia to achieve normocapnic levels have long been recognized (e.g., Laffey & Kavanagh, 2002), the earliest therapeutic approaches testing the utility of therapeutic capnography (the systematic manipulation and correction of hypocapnia) arose out of the HVS field. In the first pilot trial by pioneer Folgering and colleagues (Folgering, Lenders, & Rosier, 1980), the investigators tested whether participants without a history of pulmonary disease can be trained in increasing PCO_2. Across two sessions, 10 participants were asked to watch an oscilloscope that displayed their actual and reset values of PCO_2 (optic feedback). The task was to alter their ventilation to reach a preset PCO_2 value (+1.5 mm Hg above resting levels), which was rewarded by auditory feedback once reached (the procedure was repeated, with auditory feedback only followed by no feedback). The successful demonstration was followed up by a small, controlled trial of 20 HVS sufferers with PCO_2 levels in a hypocapnic range (≤ 35 mm Hg). Throughout seven weekly sessions, all patients assigned to respiratory training (RT) with in-laboratory PCO_2 feedback succeeded in increasing PCO_2, and more (but not all) achieved levels above the set goal of 37.5 mm Hg compared with RT only. The RT + CO_2 feedback also resulted in greater long-term maintenance and reduction in HVS symptoms (see Table 11.1 later in the chapter). The investigators speculated about relative increases in PCO_2 as a crucial factor in symptom reduction. Grossman, De Swart, and Defares (1985) followed up with another controlled HVS study demonstrating the successful increase in PCO_2 levels and HVS symptom reduction by using auditory respiration rate (RR)—feedback only. The controversy encircling the HVS diagnosis may have played a role in the lack of replication of these promising initial efforts to treating chronic hypocapnic breathing up until early 2000.

RATIONALE AND EMPIRICAL FOUNDATIONS OF MECHANISMS

Hypocapnia and Its Treatment in Panic Disorder

Respiratory abnormalities have been postulated as a central component in the development and maintenance of panic disorder based on patient reports of severe respiratory distress during panic/fear episodes and clinical and experimental observations (Nardi, Freire, & Zin, 2009; see Meuret & Ritz, 2010, for reviews). Along with palpitations and faintness, shortness of breath is the most frequent and distressing symptom reported by panic sufferers (Meuret et al., 2006). Theories on the origin of respiratory symptoms include Ley's *hyperventilation theory* and Klein's *suffocation false alarm theory*. The former viewed chronic and episodic states of hypocapnia as the origin of panic symptoms and attacks (Ley, 1985, 1991). Hyperventilation is also part of Klein's suffocation false alarm theory, but in this context, it constitutes part of a compensatory response to sudden sensations of dyspnea caused by overly sensitive chemoreceptors (Klein, 1993). In addition, hyperventilation may not be limited to the attack itself but may precede and follow it, giving rise to moderate sustained hypocapnia (for a review, see Meuret & Ritz, 2010; Meuret et al., 2011). Consequently, interventions aimed at reducing respiratory dysregulation, particularly hyperventilation, should be effective for these populations. Despite the widespread application of breathing training in anxious populations, the few studies designed to test the efficacy of traditional breathing training (e.g., Craske, Rowe, Lewin, & Noriega-Dimitri, 1997; Schmidt et al., 2000) have neither targeted nor evaluated PCO_2 as a key indicator of dysregulation. Furthermore, the respiratory instructions taught, such as slower breathing, likely perpetuate hyperventilation, thereby intensifying panic symptoms due to compensatory deeper breathing (e.g., Conrad et al., 2007; Meuret, Wilhelm, Ritz, & Roth, 2008; Meuret, Ritz, Wilhelm, Roth, & Rosenfield, 2018); or, as discussed by van Dixhoorn (Chapter 12, this volume), to a decrease in the proportion of dead space per minute volume of ventilation in slow deep breathing. Thus hyperventilation could increase even when minute ventilation does not increase. Similarly, instructions such as "Take a deep breath!" or "Breathe deeply!" not only exacerbate hypocapnia and the very symptoms the patient is trying to reduce (e.g., air hunger, shortness of breath), but also make little sense, as hypocapnia is not caused by too little but by too much ventilation. Predictably, studies on sole or combined traditional breathing training for panic disorder show mixed efficacy: Although some reports suggest benefits of traditional breathing training (Franklin, 1989; Bonn, Readhead, & Timmons, 1984; Hibbert & Chan, 1989), no added benefits were shown in others (Craske et al., 1997; Schmidt et al., 2000; see Meuret, Wilhelm, Ritz, & Roth, 2003, for a review).

Following up on the pioneering work of Folgering and colleagues in the early 1980s on utilizing PCO_2 feedback as a therapeutic tool for HVS (Folgering et al., 1980; van Doorn, Folgering, & Colla, 1982) and the one by Fried and colleagues (Fried, Rubin, Carlton, & Fox, 1984) on the successful alteration of hypocapnic levels in seizure sufferers with HVS (see Table 11.1), Meuret and colleagues in early 2000 (Meuret, Wilhelm, & Roth, 2001, 2004; Kim, Wollburg, & Roth, 2012) devised a novel intervention, coined *capnometry-assisted respiratory training* (CART), aimed at testing the merits of systematic manipulation of PCO_2 in panic sufferers.

Hypocapnia and Its Treatment in Asthma

Hypocapnia has also been a concern in asthma research. Studies have reported a heightened prevalence of symptoms that are suggestive of hypocapnia in asthma, although prevalence figures have varied widely across patient populations (e.g., D'Alba et al., 2015; de Groot, Duiverman, & Brand, 2013; Gridina, Bidat, Chevallier, & Stheneur, 2013; Thomas et al., 2001). The phenomenon has also been referred to as "dysfunctional breathing," but the validity of the questionnaire measure for asthma has been questioned (Ritz, Bobb, Edwards, & Steptoe, 2001; van Dixhoorn & Folgering, 2015). On a hyperventilation scale specifically developed for asthma, the Asthma Symptom Checklist (Kinsman, Luparello, O'Banion, & Spector, 1973), elevated report of hyperventilation symptoms (e.g., sensations of dizziness, pins and needles, numbness, tingling, nausea) has been associated with perceptions of a reduced health status over and above other asthma symptoms, and there is evidence that low perceived control of asthma mediates this relationship (Ritz, Rosenfield, Meuret, Bobb, & Steptoe, 2008). Thus patients with such symptoms may overuse medication that is designed to control their asthma rather than hyperventilation. However, self-report of such symptoms in asthma has not always correlated well with actual hypocapnia, making studies that have employed physiological measures more important. These have, indeed, reported lower PCO_2 levels in asthma compared with controls (Hormbrey, Jacobi, Patil, & Saunders, 1988; Osborne, O'Connor, Lewis, Kanabar, & Gardner, 2000). There is also evidence from laboratory studies that demonstrates the adverse effects of hyperventilation and hyperpnea on lung function (Sterling, 1968; van den Elshout, van Herwaarden, & Folgering, 1991). Additionally, individuals with asthma tend to respond more strongly with ventilation to physical challenges (Dal Negro & Allegra, 1989; Fujimori Satoh, & Arakawa, 1996; Ritz, Dahme, & Wagner, 1998), which can facilitate hypocapnia.

Breathing training has a long history of application for chronic respiratory diseases (see, e.g., Sutton, Pavia, Bateman, & Clarke, 1982; Zadai, 1990) and has been advocated as an adjunctive treatment in asthma. Various techniques, such as abdominal breathing, inspiratory muscle training, pursed-lip breathing, modifications of breathing pattern, or nasal breathing, have been used (for a critical discussion of treatment rationales and review of the earlier literature, see Ritz & Roth, 2003). Slow abdominal breathing exercises have been tested more recently in larger clinical trials with beneficial effects for symptom reduction and improvements in quality of life, whereas physiological indices such as lung function, airway inflammation, and airway hyperresponsiveness remained largely unchanged (Holloway & West, 2007; Thomas et al., 2009; Thomas et al., 2017). Other, smaller trials have shown effects on spirometric lung function (Grammatopoulo et al., 2011) or have not shown any benefits at all (Stanton, Vaughn, Carter, & Bucknall, 2008). Although potentially important for the overall well-being of patients, such interventions remain unsatisfactory due to their pragmatic character and the lack of a compelling psychobiological rationale. Despite the intention to reduce "dysfunctional breathing" and thus implicitly hyperventilation, direct manipulations of PCO_2 levels were seldom attempted, and, not too surprisingly, no substantial improvements in basal PCO_2 have been observed with a slow-breathing technique (Thomas et al., 2009).

More directly addressing hyperventilation, a breathing training method by the Russian physician Konstantin Buteyko, the "Buteyko method," has been widely advocated for complementary treatment of asthma (Stalmatski, 1999). The guiding assumption of the method is that low PCO_2 levels are the reason for several autonomic, endocrine, and metabolic disturbances, specifically contributing to asthma pathophysiology. To elevate PCO_2 levels, patients are instructed to breathe slower and more shallowly, breathe through the nose, and hold their breath intermittently. Entrainment of nose breathing by mouth taping is also sometimes added. Controlled trials have demonstrated improvements in quality of life and reduced bronchodilator use over control treatments (for a review, see Bruton & Lewith, 2005), but only one unpublished study suggested improvements in PCO_2. Taping the mouth at night alone for a median duration of 26 days did not yield any noticeable effect on symptoms or lung function compared with periods of normal breathing (Cooper et al., 2003).

Taken together, parallel developments for reducing hyperventilation using "standard" breathing training have been made (for reviews, see Ritz, Meuret, Wilhelm, & Roth, 2003, for asthma, and Meuret et al., 2003, for panic). However, even though prior interventions were aimed at reversing hypocapnia in both anxiety and asthma populations, all lacked direct manipulation of PCO_2, and only a few assessed PCO_2 (Meuret et al., 2003). The latter included studies by Grossman and colleagues (1985) for HVS; Salkovskis, Jones, and Clark (1986) for panic attacks; Meuret, Kroll, and Ritz (2017) for blood-injection-injury phobia; and Vaschillo, Vaschillo, and Lehrer (2006) and Feldman et al. (2016) for asthma. Although some demonstrated increases in PCO_2 in the absence of direct feedback (e.g., Meuret et al., 2017, Grossman et al., 1985), others did not (Feldman et al., 2016).

Therefore, evaluation of the efficacy or effectiveness of most breathing training is complicated due to the lack of this key measure. The lack of ecological capnometry devices was one reason for the obvious limitation; the ingrained belief that the manipulation of respiratory rate only (via slower breathing) would ameliorate hyperventilation may have been another. Fortunately, due to recent technological advances, user-friendly ambulatory capnometry equipment has opened new avenues for the development and implementation of therapeutic capnography.

EMPIRICAL VALIDATION OF APPLICATIONS

The CART Method

CART was designed to draw attention to and address the limitations of traditional breathing training by targeting hypocapnia directly. It is a brief, 4-week, five-session training that utilizes capnography (i.e., instant feedback of end-tidal PCO_2 via a portable capnometer) to help patients alter hypocapnic levels of PCO_2 and consequently distressing panic symptoms (e.g., dizziness and shortness of breath). The newer generation ambulatory capnometers feature instant, breath-by-breath feedback of PCO_2 in addition to respiratory rate, heart rate, and oxygen saturation. The latter could be particularly crucial for patients with suffocation fears (Figure 11.1). As illustrated in our case example later in the chapter, the alteration of depth and speed of breathing (i.e., more shallow and slower) can inevitably lead to strong sensations of dyspnea and can be interpreted catastrophically by panic sufferers (Clark, 1986). To avoid compensatory overbreathing in response to dyspnea sensations, oxygen saturation feedback can ensure patients that they indeed are not suffocating but are continuing to maintain oxygen levels in the normal range. Another advantage of portable capnometers is the real-time value of the recording and electronic storage, thus providing patients and therapists with a unique insight into their between-session practices. Real-life skill assessment recordings over “in-session snapshots” are associated with improved homework compliance and greater skill implementation and modification as they offer perspectives into patients’ physiology at divergent times of day, emotional states, and situations.

The CART Protocol

The CART training includes five individual sessions over 4 weeks. CART has four major components: (1) educating patients about the mechanisms of exacerbation of symptoms through hyperventilation and hyperpnea, (2) directing attention to potentially detrimental respiratory patterns, (3) teaching techniques to control respiration, and (4) training for in-home breathing exercises. Treatment integrity is maintained by a structured and manualized treatment protocol that details the specific goals and strategies. The core component of CART is the daily home-training exercises using a portable capnometer that samples the exhaled gas through a nasal cannula and offers immediate, breath-to-breath feedback for PCO_2 and respiratory rate. The breath-to-breath feedback allows patients to experience the immediate effects of changes in rate or depth of breathing on PCO_2. The therapist guides patients through a series of breathing maneuvers to further experience how changes in breathing affect physiology, symptoms, and mood. CART favors a capnometer model that saves the data as time-stamped exercises for in-session review and subsequent analysis. FDA-approved portable capnometers are reliable devices for measuring PCO_2 (Biedler et al., 2003). Most do not provide raw values but use internal algorithms to detect

FIGURE 11.1. Example of portable capnometer used in session. The display was set at providing breath-by-breath numeric feedback respiratory rate and CO_2, in addition to values of O_2 and heart rate. The device uses an internal algorithms to detect and extract CO_2 values.

CO_2 values, which are often provided as aggregated mean outputs of 6–7 breaths (Figure 11.1). An additional finger sensor (optional) can measure and provide feedback of SpO_2. Patients use PCO_2 and respiratory rate feedback and listen to guiding tones to regulate the speed and rhythm of their breathing. The tones correspond to a RR of 13 breaths/minute in the first week, and 10, 8, and 6 breaths/minute in the following weeks. The goal is to breathe with the tones while reaching and maintaining a PCO_2 of around 40 mm Hg (40 ± –3 mm Hg). The twice-daily, 17-minute exercises include three parts: (1) a quiet sitting baseline, (2) a 10-minute paced breathing phase, and (3) a 5-minute breathing phase without pacing tones during which patients maintain this breathing pattern without pacing information, but with continued PCO_2 feedback (see Figure 11.2 later in the chapter). Observation of SpO_2 levels informs patients that dyspnea is mostly a result of hypocapnia rather than oxygen deprivation. Patients receive a handout with an introduction to respiratory physiology and hypocapnia, the relevance of breathing to symptoms, and instructions for daily exercises. The weekly sessions are aimed at reviewing changes in respiration along with changes in symptoms and emotions. The case example given later provides an in-depth description of the treatment protocol, rationale, between-session illustration, and therapist interaction. Except for symptom specifics, CART uses the same rationale and skill-training protocol for both anxiety and asthma.

To date, six randomized controlled trials (RCTs) have tested the efficacy of CART, four for panic disorder, and two for asthma (Table 11.1). The majority of CART trials have been conducted by Meuret and her prior colleagues (Ritz, Roth, and Wilhelm) at Stanford University, where CART was originally conceived and tested. The only fully independent trial conducted is the one by Tolin, McGrath, Hale, Weiner, and Gueorguieva (2017).

CART Efficacy and Mechanism for Panic Disorder

In the first efficacy trial for CART, 37 patients with or without agoraphobia were randomly assigned to CART or to a delayed-treatment control group (Meuret et al., 2008). Panic symptom severity, anxiety sensitivity, depression, disability, respiratory rate, and end-tidal

PCO_2 were assessed at baseline, throughout treatment, posttreatment, at 2-month follow-up, and at 12-month follow-up. Patients were highly engaged and compliant, with a 100% session attendance and 91.3% between-session CART exercise completion rate (47.6 out of 52 exercises). Forty percent were panic-free at posttreatment (compared with 6% in the wait-list group), 62% at the 2-month follow-up, and 68% at the 12-month follow-up; 88% at 2-month follow-up and 96% at 12-month follow-up were either "much improved" or "very much improved" (Clinical Global Impression [CGI] > = 0.3). Recovery, defined as a 40% reduction in the initial Panic Disorder Severity Scale (PDSS) score (Shear et al., 1997), was met by 68% of the participants at posttreatment, and 79% and 93%, respectively, met remission status (≤ 2 on the PDSS) at 2-month and 12-month follow-up. Improvements were comparable to those of standard cognitive-behavioral therapy (Barlow, Gorman, Shear, & Woods, 2000), but in one-third of the time and with significantly lower patient attrition. Compared with the delayed group, PCO_2 increased significantly from a hypocapnic range to a normocapnic range at posttreatment and remained normocapnic at 2-month and 12-month follow-up. Respiratory rate was also significantly reduced. Daily exercise PCO_2 values showed a steady increase across exercises and weeks.

Despite the encouraging findings, CART, along with respiratory therapies more generally, was met with great skepticism and critique. Those points of critique pertained to the mechanism at core, which was viewed as improvements in perceived control or "rationale placebo" (e.g., Garssen, de Ruiter, & Van Dyck, 1992) (Critique A); symptom misappraisal (e.g., Salkovskis, Clark, & Gelder, 1996) (Critique B); respiratory rate, particularly at 6 breaths per minute, which has been shown to stimulate and exercise the baroreflex (e.g., Lehrer et al., 2004) (Critique C); self-induced, feedback-observed change in PCO_2 (e.g., Holroyd et al., 1984) (Critique D); and interoceptive exposure (e.g., Meuret et al., 2018) (Critique E). The critiques fostered follow-up studies that were designed to put those speculations to the test. In a second RCT (Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010), we aimed to address the first three critiques (A–C) by systematically testing the pathways of change of two theoretically distinct interventions (CART vs. cognitive therapy [CT]) using longitudinal, moderated mediation analyses. The interventions focused exclusively on the manipulation of the proposed mediator (symptom appraisal for CT and PCO_2 for CART), but also included a third, modality-nonspecific mediator, perceived control. Forty-one patients with panic disorder and agoraphobia were randomly assigned to receive 4 weeks of CART or CT. Between sessions, exercises comprised those mentioned above: twice-daily, 17-minute between-session exercises to raise PCO_2 (CART) or, during the same time period, identifying and restructuring panic-related catastrophic thoughts and cognitive errors associated with their thoughts (CT). For the latter, PCO_2 was assessed through the exercises, but measurements were unavailable to patients. Although both conditions demonstrated significant improvements in panic symptom severity, symptom misappraisal, and perceived control, only CART, but not CT, led to corrections from initially hypocapnic to normocapnic levels. And whereas PCO_2 in CART unidirectionally preceded changes in symptom appraisal, perceived control, and panic symptom severity, the association was bidirectional in CT, making it a theoretically weaker treatment. The mediational findings for PCO_2 and misappraisal duplicated those of the reanalysis of the 2008 study, which supported PCO_2 as the driver of change in CART, as earlier PCO_2 levels unidirectionally mediated later levels of symptom misappraisal and respiratory rate (Meuret, Rosenfield, Hofmann, Suvak, & Roth, 2009). CART efficacy (Tolin et al., 2017) and mediation (Davies, McGrath, Hale, Weiner, & Tolin, 2018) were independently replicated in an uncontrolled, community-based trial for panic disorder sufferers.

TABLE 11.1. Hypoventilation Trainings Using Direct PCO_2-Feedback Manipulation

Study	Subjects, *N*	Design	Findings
Folgering, Lenders, & Rosier (1980)	Nonclinical, *N* = 10	Uncontrolled, 2 in-lab PCO_2 feedback sessions with 2-level display of actual and targeted PCO_2 (+1.5 mm Hg, if reached auditory feedback)	First demonstration that PCO_2 can be increased using visual and auditory feedback
van Doorn, Folgering, & Colla (1982)	HVS with PCO_2 <35 mm Hg, *N* = 20	RCT, 7 weeks/7 sessions or PCO_2 >37.5 mm Hg for Ex and 4 sessions for Con, 3mFU, Ex = based on Folgering et al. (1980) with final goal of >37.5 mm Hg, HW = practice breathing skills w/o feedback; CON = slow, shallow abdominal breathing with 2x daily HW practice	Ex > Con reduction in HVS symptom complains at post-tx but not 3mFU, Ex = Con in PCO_2 increases
Fried, Rubin, Carlton, & Fox (1984)	Idiopathic, refractory seizures & HVS, *N* = 18	Uncontrolled, 7+ months, Ex = feedback-supported instructions in paced breathing at 12–14 bpm with goal to reach 5% PCO_2	RR/PCO_2 and RR improved, but pretx PCO_2 remained unchanged, reduction in seizure frequency
Meuret, Ritz, Wilhelm, & Roth (2007)	Asthma, *N* = 12	RCT, 4 weeks/5 sessions, 2 & 12-m FU, Ex = CART with 2x daily structured breathing exercises using portable capnometer, Con = WL	Ex > Con reduction in asthma frequency/distress symptoms, improved asthma control, mean PEF variability decreases, Ex but not Con increases from hypocapnic to normocapnic PCO_2 at post tx, 2-m FU (evidence for time-dependent change, Ritz et al., 2009)
Meuret, Wilhelm, Ritz, & Roth (2008)	Panic disorder with or without agoraphobia, *N* = 37	RCT, 4 weeks/5 sessions, 2 & 12-m FU, Ex = CART with 2x daily structured breathing exercises using portable capnometer, Con = WL	Ex > Con reduction in panic symptom severity and frequency, anxiety/depression, 68% panic free at 12-m FU, Ex but not Con increases from hypocapnic to normocapnic PCO_2 at post tx, 2-m & 6-m FU; PCO_2 mediated outcome (Meuret et al., 2009)

Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann (2010)	Panic disorder with agoraphobia, *N* = 41	RCT, 4 weeks/5 sessions, 2-m FU, Ex = CART with 2x daily 17-min breathing exercises using portable capnometer, Con = cognitive therapy with 2x daily 17-min cognitive-restructuring exercises	Ex = Con reduction in panic symptom severity and frequency, anxiety, perceived control, CART but not cognitive therapy increases from hypocapnic to normocapnic at post tx & 2-m FU; PCO_2 was CART-specific mediator
Kim, Wollburg, & Roth (2012)	Panic disorder with or without agoraphobia, *N* = 74	RCT, 4 weeks/5 sessions, 2 & 6-m FU, Ex1 = CART with 2x daily 17-min breathing exercises using portable capnometer, Ex2 = same as Ex1 but lowering of PCO_2, Con=WL	Ex1 = Ex2 reduction in panic symptom severity and frequency, anxiety, perceived control, 72.7% of Ex1 and 60.0% of Ex2 panic free at 6m FU, Ex1 but not Ex2 increases from hypocapnic to normocapnic at post tx, 2 & 6m FU
Ritz, Rosenfield, Steele, Millard, & Meuret (2014)	Asthma, *N* = 120	RCT, 4 weeks/5 sessions, 1 & 6-m FU, Ex = CART with 2x daily structured breathing exercises using portable capnometer, Con= same as CART but only RR-feedback	Ex = Con reduction in asthma control, quality of life, lung function, airways hyperreactivity, rescue inhaler use, Ex but not Con increases from hypocapnic to normocapnic PCO_2 at post tx, 1 & 6-m FU, Ex > Con reduction in respiratory impedance and asthma symptoms, lower distress during methacholine challenge
Tolin, McGarth, Hale, Weiner, & Gueorguiva (2017)	Panic disorder with or without agoraphobia, *N* = 69	Uncontrolled, multi-site non-academic clinical trial replication of Meuret et al. (2008), 4 weeks/5 sessions, 2 & 12-m FU, Ex = CART with 2x daily structured breathing exercises using portable capnometer	Reductions in panic symptom severity and frequency, anxiety, 78.8% panic free at 12-m FU, increases from hypocapnic to normocapnic PCO_2 at post tx, 2m & 6 mFU, PCO_2 mediated outcome (Davies, McGrath, Hale, Weiner, & Tolin, 2019)

Note. RCT = randomized controlled trial; mFU = months follow-up; Ex = experimental group; Con = control group; RR = respiratory rate; HW = homework; pretx = pretreatment; HVS = hyperventilation syndrome; HVT = hyperventilation test; CART = capnometry-assisted respiratory training; WL = wait-list control group; HRV = heart rate variability

Notably, respiratory rate was unrelated to symptom misappraisal in both analyses and unrelated to PCO_2 in the 2010 analysis. Together, the findings refute the ideas that corrections in misappraisal or perceived control drive therapeutic change in CART (Critiques A, B). Likewise, due to lack of mediational relation of respiratory rate to panic symptoms or even PCO_2, albeit successful, reduction in respiratory rate is an unlikely player in CART (see also Ritz, Rosenfield, Steele, Millard, & Meuret, 2014). Notably, although PCO_2 overall remained unchanged in CT, in those in which it did increase, PCO_2 mediated panic symptom severity. The finding refutes the argument of a simple expectancy effect caused by the feedback of CO_2 (Critique D), because patients receiving CT were unaware of their PCO_2 levels.

Finally, we examined the idea of CART acting as an interoceptive exposure (Meuret et al., 2018). The arguments hold strong face value given that CART exercises appear to increase, rather than decrease, symptoms, particularly symptoms of dyspnea. One reason, as speculated in Klein's suffocation alarm theory, could be the speculated oversensitivity to even the slightest rises in CO_2, which is countered with increased ventilation (and consequently hypocapnia). Indeed, during the daily exercises, PCO_2 dropped significantly, and dyspnea increased from the beginning to the end of the exercise. In support of the interoceptive exposure model (and Critique E), greater dyspnea during the daily CART exercises resulted in lower panicogenic cognitions in the subsequent treatment session across the 4 weeks of treatment, even after controlling for PCO_2.

The findings also argue against relaxation as one of the major effects of the treatment—within homework exercises, dyspnea on average increased, and these increases were also associated with increases in anxiety postexercise (although panic was reduced across weeks). It should be noted that relaxation effects of other breathing therapies may stem from their instructions to breathe slower *and* deeper, whereas the CART technique prescribes slower breathing with less compensation by depth to help elevate PCO_2. This causes dyspnea with associated anxiety, particularly in the initial phases of treatment (Meuret et al., 2018). Repeated deep breaths or sigh breaths reduce PCO_2 in a dose-dependent manner (Kroll et al., 2017) and have been suspected as a mechanism that sustains hyperventilation in panic disorder (Papp, Klein, & Gorman, 1993), making them a prime target for hypoventilation training. However, deep breaths may fulfill a function of providing the experience of relief from tension (Vlemincx, Van Diest, & Van den Bergh, 2016), and, by reducing them, a possible contributor to slow deep breathing relaxation effects is eliminated.

Following Klein's theory (1993) that the role of compensatory hyperventilation is to avoid CO_2 increases that trigger a low-threshold or hypersensitive suffocation alarm system, Kim and colleagues (2012) devised a study testing the therapeutic effects of hypocapnia training. Seventy-four patients with or without panic disorder were randomly assigned to CART or the identical protocol, except that PCO_2 was systematically lowered instead of increased. Both hyper- and hypoventilation training resulted in an analogous reduction in panic symptom severity and successful CO_2 manipulation. However, although the authors speculated on common factors driving improvements, outcome equality is not indicative of the same underlying mechanism and remains speculative in the absence of pathway testing. Even though hyperventilation could help patients bypass the feared suffocation symptoms and act as an interoceptive exposure, it is more likely that hypoventilation may serve to desensitize a hypersensitive suffocation alarm system.

CART Efficacy and Mechanism for Asthma

In an attempt to manipulate PCO_2 directly in asthma patients, CART training was adapted for this patient population (Meuret, Ritz, Wilhelm, & Roth, 2007; Ritz, Meuret, Wilhelm, & Roth, 2009). The methods and protocol largely followed those outlined for patients with panic disorder, but without any reference to the elicitation of anxiety or panic through hyperventilation. Instead, the therapy rationale outlined general health and asthma-specific adverse effects of overbreathing or hypocapnia, such as breathlessness symptoms and airway constriction. In addition to the capnometer, patients used a handheld electronic spirometer with diary functions for monitoring lung function and symptoms. Measurements of symptoms and lung function were scheduled before and after each exercise and during the five therapist-guided sessions. An illustrative case study is provided in Jeter, Kim, Simon, Ritz, and Meuret (2012).

In an initial feasibility study, 12 patients with asthma were randomly assigned either to a 4-week treatment or to a wait-list control (Meuret et al., 2007). Patients in treatment (n = 8) showed stable increases in PCO_2 and decreases in respiratory rate that was sustained at 2-month follow-up. Symptom frequency and distress were reduced, reported asthma control increased, and mean diurnal peak expiratory flow variability (as a distal marker of airway hyperreactivity) was reduced by the treatment, whereas patients in the control group remained stable across the 4-week wait period. Acceptance and credibility of the training were very high for the treatment group. Patients reported that the training improved their control over asthma symptoms, especially coughing. Further analysis showed that improvements in pCO_2, respiratory rate, and symptoms were gradual across the 4 weeks of training, suggesting that the best effects are achieved with full 4-week training (Ritz, Meuret, et al., 2009).

The CART technique was subsequently tested in a larger clinical trial, with the primary goal of establishing the specificity of PCO_2 changes relative to simple manipulation of respiratory rate (Ritz et al., 2014). For that, 120 patients with asthma were randomly assigned to CART or a slow-breathing training that used the capnometer for biofeedback of their respiratory rate instead of PCO_2. Patients in the latter group were taught to breathe more slowly and regularly, using this feedback, and to develop a heightened awareness of their breathing. Asthma control, PCO_2, and diurnal peak-flow variability were monitored as primary outcome measures by blind assessors across baseline, posttreatment, and 1-month and 6-month follow-up, together with symptoms, quality of life, bronchodilator use, and parameters of airway physiology (lung function by spirometry and forced oscillation, airway inflammation by exhaled nitric oxide, and airway hyperreactivity by methacholine challenge). Results showed improvements in most clinical indices in both interventions that were sustained throughout the 6-month follow-up. Slow breathing initially elevated PCO_2 (d = 0.64) at posttreatment, although to a smaller extent than CART (d = 1.45). Compared with CART, changes were not sustained at follow-up. CART showed superiority in reducing respiratory impedance during treatment, and patients in that group reported less distress during methacholine challenge at posttreatment, as well as a greater reduction in asthma symptoms at follow-up. Thus both device-guided methods have comparable clinical efficacy, with some additional benefits of CART.

An interesting added benefit of CART for asthma seems to lie in its potential to address comorbid anxiety (Meuret, Rosenfield, Steele, Millard, & Ritz, 2014). Although the protocol for this patient group did not address anxiety and panic, analyses of anxious mood (measured by the Hospital Anxiety and Depression Scale) and anxiety sensitivity (measured by the Anxiety Sensitivity Index) showed a drastic reduction in these measures across treatment and follow-up in the CART condition only. Because asthma shows elevated comorbidity with anxiety and panic (for reviews, see Meuret et al., 2017), CART may be a convenient approach to combining an adjunctive asthma treatment with a biobehavioral treatment for comorbid anxiety. These findings also demonstrate the specificity of PCO_2 elevations for targeting anxiety, excluding potential placebo effects or reporting biases, as patients were not taught that the training would alleviate their anxiety in this version of CART.

CAVEATS AND CONTRAINDICATIONS

Although the advantage of CART lies in its brevity compared with other interventions for anxiety, it is also very work-intensive, from both the patients' and the therapists' perspectives. Home-training assignments require at least 17 minutes of free time twice per day, which may not always be easy for patients to implement. Therapists need to download up to 14 weekly exercises at the beginning of each session to gain a quick overview of progress and identify requirements for additional instruction and in-session practice. Notwithstanding and in line with other research (e.g., Roque et al., 2018), homework compliance in CART (defined as the number of homework assignments completed) was related to greater reductions in panic symptom severity (Meuret, Hofmann, & Rosenfield, 2010). The need for a capnometer is an additional limitation that may deter therapists from implementing the treatment, although affordable handheld equipment in the range of $1,700 to $4,000 is currently marketed, and costs are recovered quickly because it can be reused for multiple patients. Nevertheless, it may be desirable to test future variants of the intervention that are limited to in-session monitoring of PCO_2 as done by Folgering and colleagues (van Doorn et al., 1982). It remains to be shown that a suitable set of breathing instructions practiced in session, combined with rigorous home training without a capnometer, can achieve similar tonic changes in PCO_2.

Capnometry training targets tonic levels of PCO_2 in a protocol over weeks, with each of the 4 training weeks providing additional gains (Ritz, Meuret, et al., 2009). However, training patients in suitable breathing patterns to avoid *situationally triggered, phasic* hyperventilation may allow for a shorter protocol. For example, patients suffering from blood-injection-injury phobia often show pronounced hyperventilation when confronted with their feared stimuli (Ritz, Wilhelm, Kullowatz, Gerlach, & Roth, 2005; Ayala, Meuret, & Ritz, 2010), with deep breaths being the major factor in reducing PCO_2 levels (Ritz, Wilhelm, Meuret, Gerlach, & Roth, 2009). Due to its strong capacity to constrict cerebral blood vessels, hyperventilation could be a prime contributor to fainting episodes typically seen in these patients. It has been shown that individuals with blood-injection-injury phobia profit from an ultra-brief, 20-minute video-supported instructional session in slow and shallow abdominal breathing (Meuret et al., 2017). When this is applied in the exposure situation as a simple coping skill, patients show increased levels of PCO_2 and blood pressure, which could reduce their risk of fainting. Furthermore, although earlier breathing therapies with paced breathing instructions showed therapeutic PCO_2 changes for patients with HVS (Grossman et al., 1985) or phobic or nonphobic panic attacks (Salkovskis et al., 1986), others failed to do so (Feldman et al., 2016). Interventions targeting hyperventilation that occurs only situationally could profit from such abbreviated protocols, although their long-term efficacy in reducing phobia, anxiety, and its corollaries awaits further evaluation.

Needless to say, interventions manipulating PCO_2 levels must ensure that PCO_2 levels do not get too high and cause acidosis or oxygen desaturation. Upper limits should be carefully observed and enforced, with values around 45 mm Hg as a maximum. Usually not an issue with lung-healthy individuals, patients with chronic obstructive pulmonary disease, who may suffer from hypercapnia, especially with more advanced or severe clinical presentation, would not qualify for hypoventilation treatment until more evidence on risks and benefits has been generated. Additional transcutaneous O_2 measurements, which are often combined with capnometry, can serve as a safety measure to detect rare cases that may respond to hypoventilation exercises with pronounced and longer lasting drops in oxygen saturation.

Hypocapnia can also be associated with a wide range of organic diseases and conditions, including cardiovascular disease, other pulmonary diseases than asthma (chronic bronchitis, emphysema, or pneumonia), chest or abdominal pain, severe liver or kidney disease, or central nervous system lesions (Cherniack, 1988; Gardner, 1996). Signs of life-threatening diseases such as diabetic ketoacidosis or hypoglycemia, where hyperventilation may occur as a secondary process, can also be misattributed to functional hyperventilation. Responsible treatment of hyperventilation with breathing training requires an exclusion or medically well-controlled status of problematic comorbidities. In the case of asthma, the ideal scenario for training initiation would be a disease status well controlled by maintenance medication for severity higher than intermittent. Medication needs could then gradually be adjusted by regular physician review and an asthma action plan that includes physician-issued criteria for stepping medication up or down and instructions for emergency measures in response to acute exacerbations (National Heart, Lung, & Blood Institute, 2007).

CASE EXAMPLE

In the following, we provide a case description that consists of a composite of several patients to preserve patient anonymity. This approach allows us to illustrate a wide variety of responses to treatment. The dialogue describes the typical conversations observed in CART between a patient and his or her therapist.

Sandra was a 38-year-old female who was first diagnosed with panic disorder in her late teens. At the time, she was treated with diazepam, which she felt had helped her cope a bit better with her physical symptoms. She discontinued her medication 6 months later because she felt fine and did not experience any further attacks until recently. She reported that, 4 months ago, while in a high-stakes business meeting, she suddenly felt her throat tightening. She tried to disregard the sensation as being just a sign of anxiety. However, after starting to feel acute shortness of breath, dizziness, and heart racing, Sandra left the meeting. That day, she sought help from her primary physician, who ensured her that there were no signs of respiratory or cardiac problems. In the months following this event, Sandra started to become apprehensive about having another episode. To cope with her fears, she had to have her old prescription bottle of diazepam with her at all times. She also started to avoid meetings whenever possible. On a few more occasions, she felt overcome with dyspnea, sweating, and racing heart while shopping or at a movie theatre.

Consequently, she avoided enclosed places with crowds. At the same time, she started to feel down and depressed. The inability to meet friends spontaneously made her feel isolated and helpless. She gave up on her exercise routine, started eating more, and began having difficulties concentrating or making decisions at work. Before her appointment, she suffered multiple nocturnal attacks, which greatly interrupted her ability to function during the day. At this point, she decided that she needed to seek help. During the diagnostic assessment, Sandra was diagnosed with panic disorder (American Psychiatric Association, 2013). She also met criteria for agoraphobia and major depressive disorder (American Psychiatric Association, 2013). Her baseline levels of PCO_2 were in a hypocapnic range at 30 mm Hg, and her respiratory rate was at 17 breaths per minute. Sandra was prescribed a 4-week, five-session course of CART.

The first session had a threefold agenda: first, to educate Sandra about the role of respiration and panic/fear; second, to have her experience this connection firsthand; and third, to instruct her in the between-session exercises. To start, the therapist educated Sandra about the fight-or-flight response and respiration, acute and sustained hyperventilation, and the role of CO_2 in respiratory regulation. To deepen understanding, the therapist used a portable capnometer and let Sandra experience the relation between different depth and rates of breathing firsthand. Here is an excerpt:

THERAPIST: Sandra, what do you believe is most important for proper breathing?

SANDRA: Oxygen.

THERAPIST: This is, indeed, what most people believe. Interestingly, though, while most people think oxygen must be the determining factor in breathing, the body actually uses carbon dioxide or CO_2 as its indicator of proper breathing. Let's test that out and see, for example, what happens when you hold your breath for about 15 seconds. (*pause*) How did that feel?

SANDRA: Terrible. I felt I was suffocating, and I really felt I needed to take a deep breath in.

THERAPIST: Yes, that is indeed what the natural response should be. But why is that? Why do you get this urge to take a deep breath?

SANDRA: Is it because my oxygen decreased?

THERAPIST: Let's take a look at your oxygen value and see what it says (pointing at the number on capnometer).

SANDRA: It says 99%.

THERAPIST: This means your oxygen is perfectly normal; in fact, almost 100%. So this cannot be the reason why you felt that air hunger. The reason, in fact, is that the chemoreceptors, which are in charge of monitoring even the tiniest changes in CO_2, started to fire and sent an alarm that you should continue breathing. If you did, in fact, have insufficient oxygen, this alarm would have helped prevent you from suffocating. For example, this effect takes place in people with sleep apnea. When they pause their breathing in their sleep, CO_2 builds up, and, once a certain threshold is passed, the brain initiates a deep breath. But your oxygen level was normal. Now, let's see if you do the opposite, taking several deep and fast breaths.

SANDRA: (*Performs maneuver.*) This is strange. I kind of felt the same as before, when I was holding my breath, like not getting enough air. But my oxygen is 100%. How is this possible?

THERAPIST: You are so right! This is really strange. You are breathing a lot of air—thus the 100% oxygenation—and yet you feel like you're not getting enough air. One could think of this being a biological error—feeling the same when breathing too much and breathing too little! Another term for overbreathing is "hyperventilation" or "hypocapnia." Your symptoms indicate a level of carbon dioxide that is below normal, caused by breathing more than metabolic demand. The way you can think about this is as if you are standing at a red light and you leave your foot on the gas pedal. The motor will be flooded with excessive gasoline which you don't use. But if you were driving at 65 miles on the highway, it would be OK to have that extra gasoline, because you are actually using it. The same applies to breathing. If you are in a resting (or freezing) state, which most people are when they have panic attacks, there is no point in breathing as if you were fighting or fleeing a threat. But why do we do it anyway?

SANDRA: Maybe because it feels like we are not getting enough air?

THERAPIST: You are absolutely right! Internal or external cues can signal danger, even when it is not there. For instance, mild shortness of breath can be such a cue. In response, you may start breathing more to prepare for action. But, because there is nothing to fight or flee from, you now have too much air in your system. As we saw before, breathing too much can lead to air hunger and more breathing.

SANDRA: I understand, but what can I do about it, particularly when I feel I am not getting enough air?

THERAPIST: This is what CART is designed for. It helps you regulate your respiration in a way so that you counter hyperventilation. How do you think we can do that?

SANDRA: By breathing slower?

THERAPIST: Yes, that is what most people think, and in fact most kinds of breathing training are based on that premise. But they are missing an important element: the depth at which you breathe, or, in other words, how much air (volume) you inhale. Why is this important? It is important because hypocapnia (or low CO_2) is a product of rate and volume of breathing. Let's understand that better. Why don't you follow these pacing tones which are set at nine breaths per minute for a few minutes? While you do that, please pay attention to what happens to your CO_2 level and respiratory rate (*pointing at CO_2 and RR on capnometer*).

SANDRA: (*performing maneuver*) Hmmm . . . I guess I was able to hold my respiratory rate at nine breaths because the number stayed at 9. But the CO_2 value went down from 32 to 25. Why is that?

THERAPIST: That is because you started to breathe in more air (or deeper). This happens a lot in patients with panic, particularly when they already mildly hyperventilate as you do. What would you need to do to increase CO_2 toward a more normal range of 37–45 mm Hg?

SANDRA: I guess I need to breathe less air? But that is so strange because everyone is telling me to take a deep breath when anxious or feeling short of breath!

THERAPIST: You are right—this is a common lay belief, and it likely stems from what we observed before. Both overbreathing and breathing too little lead to sensations of air hunger, dyspnea, and shortness of breath. However, acute oxygen deprivation is rare and tied to diseases like chronic obstructive pulmonary disease. Let's see what happens if you breathe more shallowly while pacing your respiratory rate.

SANDRA: I'm trying, but I'm not sure what you mean by breathing more shallowly.

THERAPIST: Imagine a full glass of water. Instead of drinking the entire glass in one huge gulp, you could take little sips by reducing the amount of water you were taking in with each drink. Now apply the same principle to air, so, instead of taking a huge gulp of air, you want to take little sips. Breathing only through your nose will also reduce volume. To reduce the amount of air you inhale, you can also imagine you are breathing through a beverage straw. If you hear yourself breathe, you are inhaling too much air.

SANDRA: That is helpful, but feels very unnatural.

THERAPIST: This feeling of shortness of breath is normal because your body is not used to this breathing pattern. You can compare it to drinking too much coffee, or starting to exercise after a long pause. If you start drinking less, your body will crave more, even though you're doing something that is healthier. After a while, the craving will subside because your body gets used to it. Similarly, if your body is used to taking in large amounts of air, it will feel unnatural or even wrong to breathe less. You will feel short of breath, which is kind of like a craving for more air. However, that sensation is not caused by a lack of oxygen but rather by too-low levels of PCO_2. As you can see, when looking at the values on your device, your levels of oxygen are, in fact, in the normal range. If you take deep breaths, you may continue feeling the same sensation in spite of taking in more air because your gas levels will be out of balance.

SANDRA: (*performing maneuver*) CO_2 increased a little bit, but I feel miserable. This is not at all relaxing.

THERAPIST: I understand, this is very common. Think about it this way. If someone drinks 10 cups of coffee every day, he or she will feel pretty bad when suddenly reducing it to 2 cups. The same happens to overbreathing. Your organism has gotten very used to breathing much more air than you need. That's in fact why you don't notice that you hyperventilate on a chronic basis. Breathing much less, even though it will restore your normal gas exchange, initially will not feel natural. Your chemoreceptors are accustomed to lower basal levels of CO_2, and they now start firing even when you increase the level a little bit. In most people, this will trigger an acute sense of air hunger. You can ensure yourself that this is not due to oxygen deprivation—looking at the oxygen value on the capnometer, it is due to too-low CO_2.

SANDRA: This is very interesting.

THERAPIST: So, what you will learn over the next four weeks is to gradually decrease the amount of air—or coffee cups—you inhale. I want to emphasize that the normalization of CO_2 is a gradual process that takes time and daily practice. Expect to crave more air initially. With continued practice, the craving will subside, because your body gets used to it, and eventually taking a deep breath will feel unnatural.

Lastly, Sandra was shown how to operate the portable capnometer, which she took home for her twice-daily, 17-minute practices. The therapist provided her with a daily monitoring diary to record her mood and symptoms before and after the exercise and explained to her that all exercises would be recorded with a date and time stamp. Upon her return to the following sessions, all data would be downloaded and printed out as graphs. The therapist then explained that each exercise was composed of three parts. The beginning and end of each part were announced on an audio recording. The first part of the exercise was a baseline recording during which Sandra was instructed to breathe naturally without monitoring her values on the capnometer. The data from this part would allow the therapist to see whether her CO_2 would normalize throughout therapy. During the second part of the exercise, Sandra had to breathe following pacing tones for

10 minutes. The rate was set at 13 breaths per minute (b/m) during the first week, 11 b/m during the second, 9 b/m during the third week, and 6 b/m during the last week of training. The therapist explained that the primary purpose was to keep her rate constant so she could focus on increasing her CO_2 levels. The pacing would also help her to develop a sense of slower and more regular breathing. However, because research has shown that overbreathing is primarily driven by volume, not the rate, of breathing, the major focus would always be on volume, not rate. The third part of the exercise was aimed at keeping the respiratory rate constant, without the aid of the pacing tone, while again directing the attention toward more shallow breathing to increase CO_2 levels. Sandra performed a full in-session exercise to ensure understanding and compliance.

The subsequent four therapy sessions were aimed at reinforcing Sandra's understanding of the relation between overbreathing and panic/fear symptoms, analyzing her between-session exercises, and discussing ways of implementing her skills in daily life.

Here is an excerpt of her second therapy session (see Figure 11.2):

THERAPIST: I downloaded your exercises from last week and printed them out as graphs. This is the print-out of the first exercise you did. The first box is the values for PCO_2, the second is respiratory rate (Resp), the third is oxygen (SpO2), and the fourth is heart rate (Pulse). Let's look at your baseline recording first [points are the values between 0–2 minutes; Figure 11.2]. What do you see?

SANDRA: Hmm . . . it looks like my PCO_2 is below 30 mm Hg and my rate starts around 20. Oxygen levels are around 98% and heart rate at around 70.

THERAPIST: Yes, heart rate and oxygen are in a perfectly normal range, whereas respiratory rate is quite high, and PCO_2 is below the normal range. In your diary, you indicate not experiencing many physical symptoms. Dizziness, shortness of breath, and heart rate are all below 3 [on a scale from 0–10]. Let's look at the pacing phase now [points are the values between 2–12 minutes]. What do you observe?

SANDRA: Wow—there is this major drop in PCO_2. My respiratory rate is almost a straight line.

THERAPIST: Yes, this is correct. This means already in this first exercise you were very successful in following the pacing tones. This is why, except for a few deep inhalations, you were breathing at the targeted rate of 13 breaths per minute. However, PCO_2 is indeed decreasing quite a bit—to around 21 mm Hg. We would consider that substantial hyperventilation. The low PCO_2, but mostly steady respiratory rate of 13 breath per minute, was maintained also during the transfer period [points at the values of the last 5 minutes]. How, do you think, did that happen?

SANDRA: I guess I must have taken a deeper breath.

THERAPIST: Yes, that is correct. Did you notice the drop?

SANDRA: Yes, I did, and it was frustrating. I tried to do what you said and breath more shallowly like taking small sips of air, but it did not help. I felt I was not getting enough air.

THERAPIST: I hear you and can assure you that this is a very common, and, yes, a really frustrating experience. Here are some other suggestions that have helped clients. Lie down on your bed when you do the exercise. When we lie, it's harder to overbreathe—place your hand on your chest and abdomen. You should

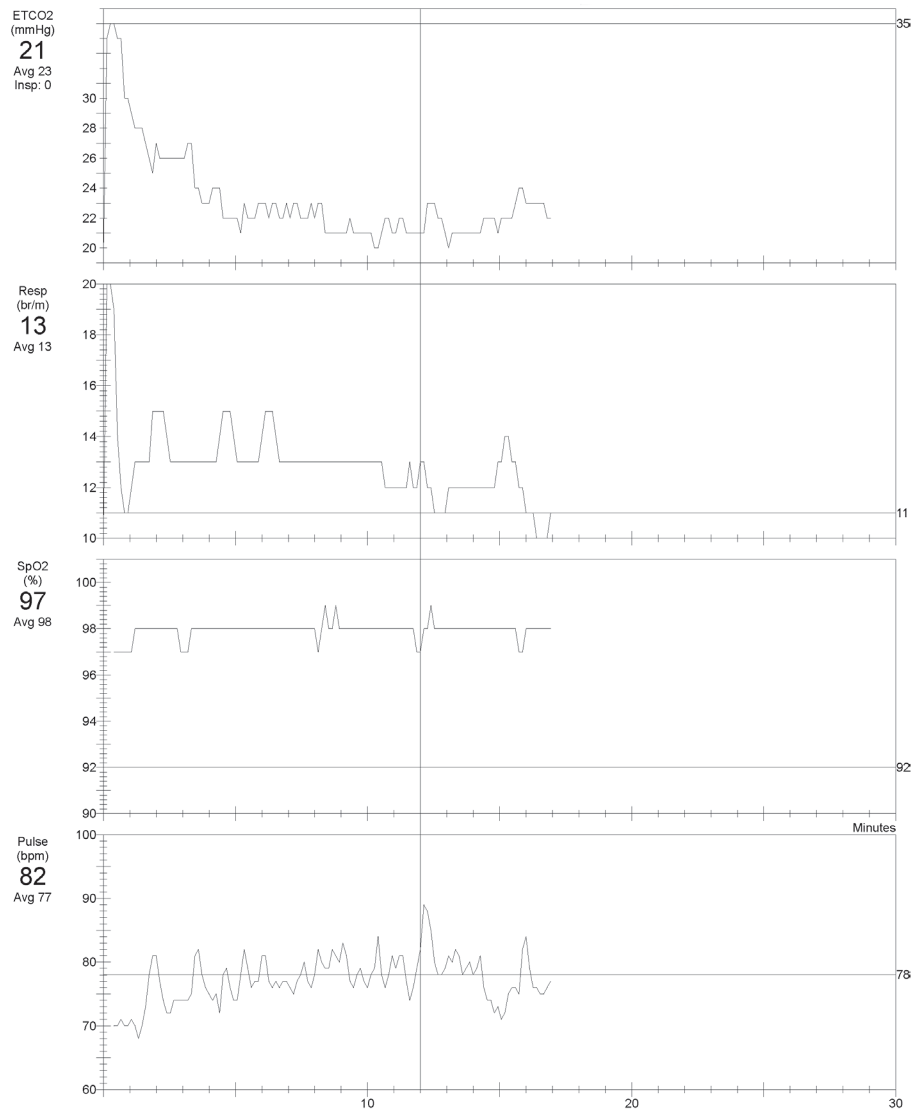


FIGURE 11.2. Between-session exercise (first week, first exercise). The figure illustrates a common problem of the significant drop in PCO_2 while breathing at pacing tones of 13 breaths/minute.

virtually feel no movement. Imagine the sea on a very calm day. The water just slides in and out, with very little effort. Similarly, the air you inhale should just slide in and out effortlessly, in a very small quantity. Remember, renormalizing your respiration is like starting to exercise after months or years of pausing. It won't feel good, but it gets easier if you keep doing it. Does this help?

SANDRA: Yes, this is helpful. I guess I just need to be more patient with myself. How often should I look at the capnometer numbers?

THERAPIST: Initially, and particularly when your PCO_2 is low, try to look as little as possible. Try to visualize your breath and get a sense of slow, shallow, and calm breathing. Once you feel you have better control over your rhythm, sleep, and volume of your breath, you can start checking your PCO_2 occasionally. If you feel you are not getting enough air, reassure yourself that this is not the case by looking at the oxygen. Let's practice that here with the new tones set at 11 breaths per minute!

Sandra's PCO_2 values slowly increased over the course of the four weeks of treatment. Overall, she was very compliant with her exercises but found it challenging at times to do them during her work, particularly when she was stressed. The therapist pointed out that these would be the best times to practice. She noted that she should expect to struggle more to achieve the same numbers as when not stressed but that this would provide her with an opportunity to understand her respiratory responses during stress or anxiety. Sandra also somewhat struggled with the progressively slower pace of the tones.

The following conversation is from her last treatment session.

THERAPIST: Sandra—do you remember your first exercise (*showing her the printout*)? Let's now see what your final exercise looks like. What differences do you see? [points at Figure 11.3.]

SANDRA: I think it looks really good!

THERAPIST: Yes, it really does! Fantastic work indeed! Throughout the pacing and transfer period [points at minute 5–15], you maintain a PCO_2 in a normal range (around 39 mm Hg), and your respiratory rate is around 6–7 breaths per minute. Let's also take a look at your baseline levels when you weren't trying to

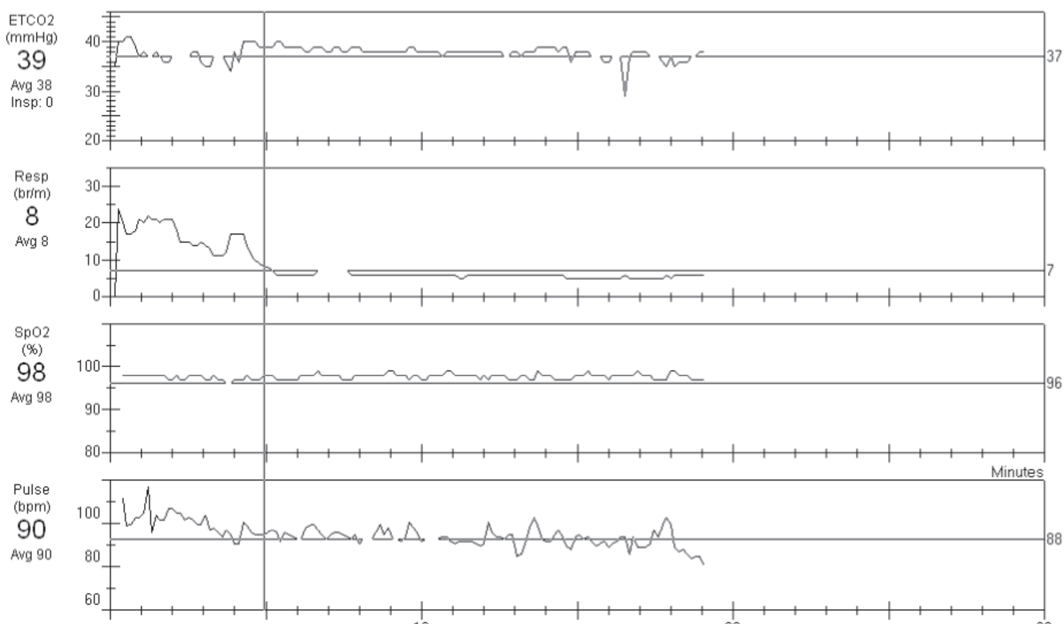


FIGURE 11.3. Between-session exercise (fourth week, last exercise). PCO_2 is in a normal range and targeted 6 breath/minute goal is achieved and maintained during pacing and nonpacing periods.

control your breathing [points at values minutes 3–5]. What can you tell me from this printout?

SANDRA: My PCO_2 is in the normal range, and my respiration rate is around 13. I can see that it starts off a bit higher but that does not seem to affect my PCO_2.

THERAPIST: Yes, indeed! You have learned not only to maintain a normal rate and depth of breathing when you are paying attention to your breathing but, just as importantly, when you are not. What have you noticed about the frequency or severity of your panic symptoms?

SANDRA: I have not had any panic symptoms or panic attacks. I also stopped avoiding places where I had symptoms before. I now understand how uncomfortable symptoms are produced and that I can influence them if I want.

REFERENCES

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Ayala, E. S., Meuret, A. E., & Ritz, T. (2010). Confrontation with blood and disgust stimuli precipitates respiratory dysregulation in blood-injury-injection phobia. *Biological Psychology, 84*(1), 887–897.

Barlow, D. H., Gorman, J. M., Shear, M. K., & Woods, S. W. (2000). Cognitive-behavioral therapy, imipramine, or their combination for panic disorder: A randomized controlled trial. *Journal of the American Medical Association, 283*(19), 2529–2536.

Bass, C. (1997). Hyperventilation syndrome: A chimera? *Journal of Psychosomatic Research, 42*(5), 421–426.

Bass, C., & Gardner, W. N. (1985). Respiratory and psychiatric abnormalities in chronic symptomatic hyperventilation. *British Medical Journal, 290,* 1387–1390.

Biedler, A. E., Wilhelm, W., Kreuer, S., Soltesz, S., Bach, F., Mertzlufft, F. O., et al. (2003). Accuracy of portable quantitative capnometers and capnographs under prehospital conditions. *American Journal of Emergency Medicine, 21,* 520–524.

Bonn, J. A., Readhead, C. A., & Timmons, B. (1984). Enhanced adaptive behavioural response in agoraphobic patients pretreated with breathing retraining. *Lancet, 324*(8404), 665–669.

Bruton, A., & Lewith, G. T. (2005). The Buteyko breathing technique for asthma: A review. *Complementary Therapies in Medicine, 13*(1), 41–46.

Cherniack, N. S. (1988). Hyperventilation syndromes. In J. F. Murray & J. A. Nadel (Eds.), *Textbook of respiratory medicine* (pp. 1861–1866). Philadelphia: Saunders.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*(4), 461–470.

Conrad, A., Müller, A., Doberenz, S., Kim, S., Meuret, A. E., Wollburg, E., et al. (2007). Psychophysiological effects of breathing instructions for stress management. *Applied Psychophysiology and Biofeedback, 32*(2), 89–98.

Cooper, S., Oborne, J., Newton, S., Harrison, V., Coon, J. T., Lewis, S., et al. (2003). Effect of two breathing exercises (Buteyko and pranayama) in asthma: A randomised controlled trial. *Thorax, 58*(8), 674–679.

Craske, M. G., Rowe, M., Lewin, M., & Noriega-Dimitri, R. (1997). Interoceptive exposure versus breathing retraining within cognitive-behavioural therapy for panic disorder with agoraphobia 1. *British Journal of Clinical Psychology, 36*(1), 85–99.

Dal Negro, R., & Allegra, L. (1989). Blood gas changes during and after nonspecific airway challenge in asthmatic and normal subjects. *Journal of Applied Physiology, 67*(6), 2627–2630.

D'Alba, I., Carloni, I., Ferrante, A. L., Gesuita, R., Palazzi, M. L., & de Benedictis, F. M. (2015). Hyperventilation syndrome in adolescents with and without asthma. *Pediatric Pulmonology, 50*(12), 1184–1190.

Davies, C. D., McGrath, P. B., Hale, L. R., Weiner, D. N., & Tolin, D. F. (2018). Mediators of change in capnometry guided respiratory intervention for panic disorder. *Applied Psychophysiology and Biofeedback, 44*(2), 97–102.

de Groot, E. P., Duiverman, E. J., & Brand, P.

L. (2013). Dysfunctional breathing in children with asthma: A rare but relevant comorbidity. *European Respiratory Journal, 41*(5), 1068–1073.

Feldman, J. M., Matte, L., Interian, A., Lehrer, P. M., Lu, S. E., Scheckner, B., et al. (2016). Psychological treatment of comorbid asthma and panic disorder in Latino adults: Results from a randomized controlled trial. *Behaviour Research and Therapy, 87,* 142–154.

Folgering, H., Lenders, J., & Rosier, L. (1980). Biofeedback control of $PaCO_2$: A prospective therapy in hyperventilation. In H. Herzog et al. (Eds.), *Asthma: Progress in respiratory research* (Vol. 14, pp. 26–30). Basel, Switzerland: Karger.

Franklin, J. A. (1989). A 6-year follow-up of the effectiveness of respiratory retraining, in-situ isometric relaxation, and cognitive modification in the treatment of agoraphobia. *Behavior Modification, 13*(2), 139–167.

Fried, R. (1993). *The psychology and physiology of breathing: In behavioral medicine, clinical psychology, and psychiatry.* New York: Plenum Press.

Fried, R., Rubin, S. R., Carlton, R. M., & Fox, M. C. (1984). Behavioral control of intractable idiopathic seizures: I. Self-regulation of end-tidal carbon dioxide. *Psychosomatic Medicine, 46*(4), 315–331.

Fujimori, K., Satoh, M., & Arakawa, M. (1996). Ventilatory response to continuous incremental changes in respiratory resistance in patients with mild asthma. *Chest, 109*(6), 1525–1531.

Gardner, W. N. (1996). The pathophysiology of hyperventilation disorders. *Chest, 109*(2), 516–534.

Garssen, B., de Ruiter, C., & Van Dyck, R. (1992). Breathing retraining: A rational placebo? *Clinical Psychology Review, 12*(2), 141–153.

Grammatopoulou, E. P., Skordilis, E. K., Stavrou, N., Myrianthefs, P., Karteroliotis, K., Baltopoulos, G., et al. (2011). The effect of physiotherapy-based breathing retraining on asthma control. *Journal of Asthma, 48*(6), 593–601.

Gridina, I., Bidat, E., Chevallier, B., & Stheneur, C. (2013). Prevalence of chronic hyperventilation syndrome in children and teenagers. *Archives de Pediatrie, 20*(3), 265–268.

Grossman, P., De Swart, J. C. G., & Defares, P. B. (1985). A controlled study of a breathing therapy for treatment of hyperventilation syndrome. *Journal of Psychosomatic Research, 29*(1), 49–58.

Hibbert, G. A., & Chan, M. (1989). Respiratory control: Its contribution to the treatment of panic attacks: A controlled study. *British Journal of Psychiatry, 154*(2), 232–236.

Holloway, E. A., & West, R. J. (2007). Integrated breathing and relaxation training (the Papworth method) for adults with asthma in primary care: A randomised controlled trial. *Thorax, 62*(12), 1039–1042.

Holroyd, K. A., Penzien, D. B., Hursey, K. G., Tobin, D. L., Rogers, L., Holm, J. E., et al. (1984). Change mechanisms in EMG biofeedback training: Cognitive changes underlying improvements in tension headache. *Journal of Consulting and Clinical Psychology, 52*(6), 1039–1053.

Hormbrey, J., Jacobi, M. S., Patil, C. P., & Saunders, K. B. (1988). CO_2 response and pattern of breathing in patients with symptomatic hyperventilation, compared to asthmatic and normal subjects. *European Respiratory Journal, 1*(9), 846–851.

Howell, J. B. (1997). The hyperventilation syndrome: A syndrome under threat? *Thorax, 52*(Suppl. 3), S30–S34.

Jack, S., Rossiter, H. B., Pearson, M. G., Ward, S. A., Warburton, C. J., & Whipp, B. J. (2004). Ventilatory responses to inhaled carbon dioxide, hypoxia, and exercise in idiopathic hyperventilation. *American Journal of Respiratory and Critical Care Medicine, 170*(2), 118–125.

Jeter, A. M., Kim, H. C., Simon, E., Ritz, T., & Meuret, A. E. (2012). Hypoventilation training for asthma: A case illustration. *Applied Psychophysiology and Biofeedback, 37*(1), 63–72.

Jones, M., Harvey, A., Marston, L., & O'Connell, N. E. (2013). Breathing exercises for dysfunctional breathing/hyperventilation syndrome in adults. *Cochrane Database of Systematic Reviews,* (5).

Kerr, W. J., Gliebe, P. A., & Dalton, J. W. (1938). Physical phenomena associated with anxiety states: The hyperventilation syndrome. *California and Western Medicine, 48*(1), 12–16.

Kim, S., Wollburg, E., & Roth, W. T. (2012). Opposing breathing therapies for panic disorder: Randomized controlled trial of lowering vs raising end-tidal pCO_2. *Journal of Clinical Psychiatry, 73*(7), 931–939.

Kinsman, R. A., Luparello, T., O'Banion, K., & Spector, S. (1973). Multidimensional analysis of the subjective symptomatology of asthma. *Psychosomatic Medicine, 35*(3), 250–267.

Klein, D. F. (1993). False suffocation alarms, spontaneous panics, and related conditions: An integrative hypothesis. *Archives of General Psychiatry, 50*(4), 306–317.

Kroll, J. L., Meuret, A. E., Adangwa, P. J., Battaglini, N., Fessler, A., Banerjee, N., et al. (2017, September). *How many deep breaths are needed to hyperventilate?* Poster presented at the 24th annual meeting of the International Society for the Advancement of Respiratory Psychophysiology, Lille, France.

Laffey, J. G., & Kavanagh, B. P. (1999). Carbon dioxide and the critically ill: Too little of a good thing? *Lancet, 354*(9186), 1283–1286.

Laffey, J. G., & Kavanagh, B. P. (2002). Hypocapnia. *New England Journal of Medicine, 347*(1), 43–53.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Lu, S. E., Scardella, A., Siddique, M., et al. (2004). Biofeedback treatment for asthma. *Chest, 126*(2), 352–361.

Ley, R. (1985). Agoraphobia, the panic attack and the hyperventilation syndrome. *Behaviour Research and Therapy, 23*(1), 79–81.

Ley, R. (1991). The efficacy of breathing retraining and the centrality of hyperventilation in panic disorder: A reinterpretation of experimental findings. *Behaviour Research and Therapy, 29*(3), 301–304.

Lum, L. C. (1987). Hyperventilation syndromes in medicine and psychiatry: A review. *Journal of the Royal Society of Medicine, 80*(4), 229–231.

Marhong, J., & Fan, E. (2014). Carbon dioxide in the critically ill: Too much or too little of a good thing? *Respiratory Care, 59,* 1597–1605.

Magarian, G. J. (1982). Hyperventilation syndromes: Infrequently recognized common expressions of anxiety and stress. *Medicine, 61*(4), 219–236.

Meuret, A. E., Hofmann, S. G., & Rosenfield, D. (2010). Catastrophic appraisal and perceived control as moderators of treatment response in panic disorder. *International Journal of Cognitive Therapy, 3*(3), 262–277.

Meuret, A. E., Kroll, J. L, & Ritz, T. (2017). Panic disorder comorbidity with medical conditions and treatment implications. *Annual Review of Clinical Psychology, 13,* 209–240.

Meuret, A. E., & Ritz, T. (2010). Hyperventilation in panic disorder and asthma: Empirical evidence and clinical strategies. *International Journal of Psychophysiology, 78*(1), 68–79.

Meuret, A. E., Ritz, T., Wilhelm, F. H., & Roth, W. T. (2007). Targeting pCO_2 in asthma: Pilot evaluation of a capnometry-assisted breathing training. *Applied Psychophysiology and Biofeedback, 32*(2), 99–109.

Meuret, A. E., Ritz, T., Wilhelm, F. H., Roth, W. T., & Rosenfield, D. (2018). Hypoventilation therapy alleviates panic by repeated induction of dyspnea. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 3*(6), 539–545.

Meuret, A. E., Rosenfield, D., Hofmann, S. G., Suvak, M. K., & Roth, W. T. (2009). Changes in respiration mediate changes in fear of bodily sensations in panic disorder. *Journal of Psychiatric Research, 43*(6), 634–641.

Meuret, A. E., Rosenfield, D., Seidel, A., Bhaskara, L., & Hofmann, S. G. (2010). Respiratory and cognitive mediators of treatment change in panic disorder: Evidence for intervention specificity. *Journal of Consulting and Clinical Psychology, 78,* 691–704.

Meuret, A. E., Rosenfield, D., Steele, A. M., Millard, M., & Ritz, T., (2014, September). *Pilot study of hypoventilation treatment on anxiety in patients with asthma.* Paper presented at the 21st annual meeting of the International Society for the Advancement of Respiratory Psychophysiology, New Brunswick, NJ.

Meuret, A. E., Rosenfield, D., Wilhelm, F. H., Zhou, E., Conrad, A., Ritz, T., et al. (2011). Do unexpected panic attacks occur spontaneously? *Biological Psychiatry, 70*(10), 985–991.

Meuret, A. E., White, K. S., Ritz, T., Roth, W. T., Hofmann, S. G., & Brown, T. A. (2006). Panic attack symptom dimensions and their relationship to illness characteristics in panic disorder. *Journal of Psychiatric Research, 40*(6), 520–527.

Meuret, A. E., Wilhelm, F. H., Ritz, T., & Roth, W. T. (2003). Breathing training for treating panic disorder: Useful intervention or impediment? *Behavior Modification, 27*(5), 731–754.

Meuret, A. E., Wilhelm, F. H., Ritz, T., & Roth, W. T. (2008). Feedback of end-tidal pCO2 as a therapeutic approach for panic disorder. *Journal of Psychiatric Research, 42*(7), 560–568.

Meuret, A. E., Wilhelm, F. H., & Roth, W. T. (2001). Respiratory biofeedback-assisted therapy in panic disorder. *Behavior Modification, 25*(4), 584–605.

Meuret, A. E., Wilhelm, F. H., & Roth, W. T. (2004). Respiratory feedback for treating panic disorder. *Journal of Clinical Psychology, 60*(2), 197–207.

Nardi, A. E., Freire, R. C., & Zin, W. A. (2009). Panic disorder and control of breathing. *Respiration Physiology and Neurobiology, 167*(1), 133–143.

National Heart, Lung, & Blood Institute. (2007). *Expert Panel Report 3: Guidelines for the diagnosis and management of asthma: Full report.* Washington, DC: Department of

Health and Human Services, National Asthma Education and Prevention Program.

Oakes, D. F., & Jones, S. (2017). *Oakes' respiratory care pocket guide* (9th ed.). Old Town, MN: Health Educator.

Osborne, C. A., O'Connor, B. J., Lewis, A., Kanabar, V., & Gardner, W. N. (2000). Hyperventilation and asymptomatic chronic asthma. *Thorax, 55*(12), 1016–1022.

Papp, L. A., Klein, D. F., & Gorman, J. M. (1993). Carbon dioxide hypersensitivity, hyperventilation, and panic disorder. *American Journal of Psychiatry, 150,* 1149–1157.

Ritz, T., Bobb, C., Edwards, M., & Steptoe, A. (2001). The structure of symptom report in asthma: A re-evaluation. *Journal of Psychosomatic Research, 51*(5), 639–645.

Ritz, T., Dahme, B., & Wagner, C. (1998). Effects of static forehead and forearm muscle tension on total respiratory resistance in healthy and asthmatic participants. *Psychophysiology, 35*(5), 549–562.

Ritz, T., Meuret, A., Wilhelm, F., & Roth, W. T. (2003) End-tidal pCO2 levels in asthma patients in the laboratory and at home [Abstract]. *Biological Psychology, 62,* 233–244.

Ritz, T., Meuret, A. E., Wilhelm, F. H., & Roth, W. T. (2009). Changes in pCO_2, symptoms, and lung function of asthma patients during capnometry-assisted breathing training. *Applied Psychophysiology and Biofeedback, 34*(1), 1–6.

Ritz, T., Rosenfield, D., Meuret, A. E., Bobb, C., & Steptoe, A. (2008). Hyperventilation symptoms are linked to a lower perceived health in asthma patients. *Annals of Behavioral Medicine, 35*(1), 97–104.

Ritz, T., Rosenfield, D., Steele, A. M., Millard, M., & Meuret, A. E. (2014). Controlling asthma by training of capnometry-assisted hypoventilation (CATCH) versus slow breathing: A randomized controlled trial. *Chest, 146*(5), 1237–1247.

Ritz, T., & Roth, W. T. (2003). Behavioral interventions in asthma: Breathing training. *Behavior Modification, 27*(5), 710–730.

Ritz, T., Wilhelm, F. H., Kullowatz, A., Gerlach, A., & Roth, W. T. (2005). End-tidal pCO_2-levels in blood phobia during viewing of emotional and disease-relevant films. *Psychosomatic Medicine, 67,* 661–668.

Ritz, T., Wilhelm, F. H., Meuret, A., Gerlach, A., & Roth, W. T. (2009). Do blood phobia patients hyperventilate during exposure by breathing faster, deeper, or both? *Depression and Anxiety, 26,* E60–E67.

Roque, A., Rosenfield, D., Smits, J. A. J., Simon, N., Otto, M. W., Marques, L., et al. (2018). Does d-cycloserine facilitate the effects of CBT in homework-compliant patients with social anxiety disorder? *Journal of Anxiety Disorders, 53,* 85–90.

Salkovskis, P. M., Clark, D. M., & Gelder, M. G. (1996). Cognition-behaviour links in the persistence of panic. *Behaviour Research and Therapy, 34*(5–6), 453–458.

Salkovskis, P. M., Jones, D. R., & Clark, D. M. (1986). Respiratory control in the treatment of panic attacks: Replication and extension with concurrent measurement of behaviour and pCO_2. *British Journal of Psychiatry, 148*(5), 526–532.

Schmidt, N. B., Woolaway-Bickel, K., Trakowski, J., Santiago, H., Storey, J., Koselka, M., et al. (2000). Dismantling cognitive-behavioral treatment for panic disorder: Questioning the utility of breathing retraining. *Journal of Consulting and Clinical Psychology, 68*(3), 417–424.

Shear, M. K., Brown, T. A., Barlow, D. H., Money, R., Sholomskas, D. E., Woods, S. W., et al. (1997). Multicenter Collaborative Panic Disorder Severity Scale. *American Journal of Psychiatry, 154*(11), 1571–1575.

Stalmatski, A. (1999). *Freedom from asthma: Buteyko's revolutionary treatment.* London: Kyle Cathie.

Stanton, A. E., Vaughn, P., Carter, R., & Bucknall, C. E. (2008). An observational investigation of dysfunctional breathing and breathing control therapy in a problem asthma clinic. *Journal of Asthma, 45*(9), 758–765.

Sterling, G. M. (1968). The mechanism of bronchoconstriction due to hypocapnia in man. *Clinical Science, 34*(2), 277–285.

Sutton, P. P., Pavia, D., Bateman, J. R., & Clarke, S. W. (1982). Chest physiotherapy: A review. *European Journal of Respiratory Diseases, 63*(3), 188–201.

Tavel, M. E. (1990). Hyperventilation syndrome: Hiding behind pseudonyms? *Chest, 97*(6), 1285–1289.

Thomas, M., Bruton, A., Little, P., Holgate, S., Lee, A., Yardley, L., et al. (2017). A randomised controlled study of the effectiveness of breathing retraining exercises taught by a physiotherapist either by instructional DVD or in face-to-face sessions in the management of asthma in adults. *Health Technology Assessment, 21*(53).

Thomas, M., McKinley, R. K., Freeman, E., & Foy, C. (2001). Prevalence of dysfunctional breathing in patients treated for asthma in primary care: Cross-sectional survey. *British Medical Journal, 322*(7294), 1098–1100.

Thomas, M., McKinley, R. K., Mellor, S., Watkin, G., Holloway, E., Scullion, J., et al. (2009). Breathing exercises for asthma: A randomised controlled trial. *Thorax, 64*(1), 55–61.

Tolin, D. F., McGrath, P. B., Hale, L. R., Weiner, D. N., & Gueorguieva, R. (2017). A multisite benchmarking trial of capnometry guided respiratory intervention for panic disorder in naturalistic treatment settings. *Applied Psychophysiology and Biofeedback, 42*(1), 51–58.

van den Elshout, F. J., van Herwaarden, C. L., & Folgering, H. T. (1991). Effects of hypercapnia and hypocapnia on respiratory resistance in normal and asthmatic subjects. *Thorax, 46*(1), 28–32.

van Dixhoorn, J., & Folgering, H. (2015, May 15). The Nijmegen Questionnaire and dysfunctional breathing. *European Respiratory Journal Open Research, 1*(1).

van Doorn, P., Folgering, H., & Colla, P. (1982). Control of the end-tidal PCO_2 in the hyperventilation syndrome: Effects of biofeedback and breathing instructions compared. *Bulletin Europeen de Physiopathologie Respiratoire, 18*(6), 829–836.

Vaschillo, E. G., Vaschillo, B., & Lehrer, P. M. (2006). Characteristics of resonance in heart rate variability stimulated by biofeedback. *Applied Psychophysiology and Biofeedback, 31*(2), 129–142.

Vlemincx, E., Van Diest, I., & Van den Bergh, O. (2016). A sigh of relief or a sigh to relieve: The psychological and physiological relief effect of deep breaths. *Physiology and Behavior, 165,* 127–135.

Zadai, C. C. (1990). Therapeutic exercise in pulmonary disease and disability. In J. V. Basmajian & S. L. Wolf (Eds.), *Therapeutic exercise* (5th ed., pp. 405–430). Baltimore: Williams & Wilkins.

CHAPTER 12

Whole-Body Breathing

A Systems Perspective on Respiratory Retraining

Jan van Dixhoorn

This chapter describes a breathing method that has been developed over the past 50 years in Europe. It includes elements of direct respiratory retraining, as well as indirect approaches to modify respiration by way of its connections to the whole body. It is combined with a systems perspective by taking mental and physical tension states into account.

HISTORY OF THE METHOD

One of the roots of the method is the work of voice teachers in Germany and the Netherlands: Gerard Meyer, Bram Balfoort, and the German laryngologist Bernhard Ulrich (1928). They had similar experiences with the medical profession to those of Carl Stough in the same time period in the United States (Stough, 1970). Bram Balfoort worked in an academic hospital from the 1950s until the 1970s, and he successfully treated all sorts of serious lung patients, including those with chronic obstructive pulmonary disease (COPD). Although the patients felt better, improvement did not show in the lung function measurements. Stough had the same experience and described his frustration in his book.

I cooperated with Balfoort for several years. This resulted in a popular book (Balfoort & Van Dixhoorn, 1979) and further development of the techniques. By applying them in the context of cardiac rehabilitation and studying the effects of various procedures using biofeedback, I shifted the emphasis from breathing technique to a wider perspective of relaxation, body awareness, and tension regulation. An initial treatment protocol was the basis of a clinical trial of breathing therapy in the early 1980s on patients who had experienced a myocardial infarction. Clear benefits appeared from adding relaxation to exercise training (Van Dixhoorn & Duivenvoorden, 1989; Van Dixhoorn, Duivenvoorden, Staal, & Pool, 1989), and also in the hard measurements (such as ST-deviations).

This outcome was accepted by cardiologists and was therefore included in the Netherlands national guidelines for cardiac rehabilitation. Thus it had far-reaching effects on cardiac rehabilitation practice in the Netherlands (Committee for the Rehabilitation of Cardiac Patients, 1996; Kruyssen et al., 1996; Revalidatiecommissie Nederlandse Vereniging Voor Cardiologie [NVvC], 2011). Now, about one-third of the hospitals that conduct cardiac rehabilitation programs include relaxation therapy using the present method (de Vries, Kemps, van Engen-Verheul, Kraaijenhagen, & Peek, 2015).

I applied the concepts of Edmund Jacobson (see McGuigan & Lehrer, Chapter 7, this volume) to systematically reduce residual, unnecessary muscle tension and effort associated with breathing. This was supplemented by elements from the dynamic system of Feldenkrais (1972), who defined effort as muscle tension that is in excess relative to its function in moving the bony structure of the body. As a result, I developed a model for breathing patterns in relation to skeletal movement, particularly the spinal column. Instructions were designed that influence breathing movement throughout the body, from the head to the feet, in a way that is, in principle, broadly accessible.

PROCESS-ORIENTED APPROACH

The main function of therapy is the self-regulation of tension. I used the concept of Lum (1976) to describe habitual tension as the key element of treatment: Many people have become accustomed to effort, and therefore excess muscle tension, in their breathing. When complicated instructions in breathing are given, following these instructions can cause even more tension, although the extra effort may disappear over time. I took upon myself the task to develop a set of instructions that were both effective and easy to follow. In our treatment protocol, we give an instruction and then take time to let the client describe his or her experiences. Was it easy to do? Did the person notice a change afterward? Did he or she like the change? We discuss this, note this down, and then move on to the next instruction. We adapt the next instructions to these experiences. We give two to four instructions per session and ask the trainee to practice them at home (see the Case Example later in this chapter). At the following session, we discuss the home experiences, and then choose how to proceed: offer new instructions or repeat the ones we did previously and clarify them. In three to four sessions, both the client and the practitioner get a clear idea of whether the treatment is working for the client.

Readers are recommended to do the same: simply to try the instructions given (see the "Instructions" subsection in the "Treatment Manual" section later in this chapter). If there is no more than habitual tension, the experiences are usually positive. However, if the client's circumstances continue to produce stress, or he or she is emotionally severely disturbed or intrinsically unmotivated to do something to help him- or herself, the experience is significantly less positive. This procedure will detect "responsive" clients, for whom the instructions work and can solve the habitual tension.

I call this a *process-directed* approach: to detect early on the presence of "blocking stressors" that complicate treatment (Van Dixhoorn, Scheffers, & Bussbach, 2017).

The method has been described in a manual (Van Dixhoorn, 1998b) that has become the basis of a 3-year part-time training program in breathing therapy for professionals. Participants in this program organized as a union (VDV; Van Dixhoorn Vereniging) and a foundation for quality control (Adem-en Ontspanningstherapie Stichting [AOS]). They have arranged to be reimbursed by major insurance companies as a complementary therapy, and they apply the method to a wide range of health problems.

THEORETICAL FOUNDATIONS

Basics of Respiration

Respiration is a rhythmic contraction and expansion of the body, as a result of which air flows in and out of the lungs. It can be represented as a curve, going up as the air flows in with inhalation and down as the air flows out with exhalation (Figure 12.1). Normal inhalation time is about 40% of total cycle length, and exhalation is about 60%. There are slight pauses or breath holds at the end of inhalation (about 3–5%) and at the end of exhalation (5–10%), when the flow reverses. However, there is great variation in the timing of respiration. Total cycle length determines the rate of respiration per minute, or cycles per minute (CPM), which also shows great variation and can be anywhere between very low frequencies of 3–6 CPM and higher frequencies of 16–24 CPM, or even higher. The amount of air that flows in and out of the lungs is another variable that determines breathing pattern. It is called tidal volume, V_t, and varies greatly as well. In resting situations, it is normally between 0.3 and 0.5 liters. The minute volume (MV) is the total amount of air that passes in and out of the lungs per minute. It is the product of V_t and CPM.

The action of air passing in and out of the lungs is called *ventilation*. The outside air that comes in supplies fresh oxygen, and the air that is expelled from the lungs contains carbon dioxide (CO_2). Both gases are essential for (aerobic) metabolism, which provides the energy that the living system requires. Oxygen combines with nutrient substances to produce energy and leaves water and CO_2 as waste products. Thus the metabolism in living tissues produces CO_2, which is passed to venous blood and carried to the lungs. In the small air sacs, or *alveoli,* the blood capillaries allow diffusion of the gases between the blood and the air. The high concentration of CO_2 in the blood creates diffusion to the alveoli, which contain the outside air with much lower concentration of CO_2. At the end of expiration, the concentration of CO_2 in the expired air almost equals the venous concentration. This end-tidal CO_2 ranges between 37 and 43 mm Hg. Carbon dioxide is important for many reasons, including its determination of the acidity of the blood, or pH. The pH is vital for homeostasis and needs to be regulated within a strictly delimited range. When CO_2 drops, the blood becomes less acidic; when CO_2 rises, the blood becomes more acidic.

By contrast, oxygen concentration is higher in inspired air than in venous blood, and oxygen diffuses in the alveoli into the capillary blood vessels. The oxygenated blood circulates back into the heart and from there throughout the body. Almost all the oxygen

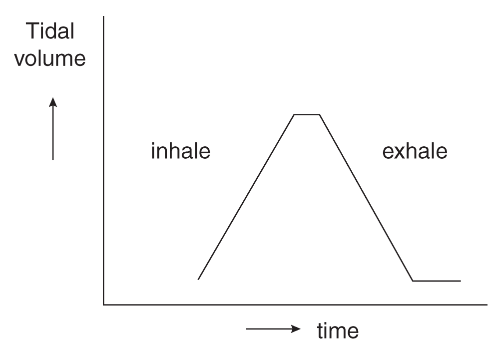


FIGURE 12.1. Respiration curve.

is bound to hemoglobin molecules in the blood, which carry and store it. A small percentage is dissolved in the blood and has sufficient pressure to allow it to pass to the tissues that need it. Under normal conditions, the hemoglobin in arterial blood is almost 100% saturated with oxygen. When the pressure of the dissolved oxygen (pO_2) drops, the saturation decreases slowly but leaves sufficient oxygen in storage. However, when the acidity of the blood decreases because of low CO_2 levels, oxygen is bound more tightly to the hemoglobin and passes less easily to the tissues. This is called the *Bohr effect* (Lumb, 2000).

Normally, minute ventilation is regulated automatically by the requirements for gas exchange. When the body becomes more active and requires more energy, metabolism increases, more oxygen is required, and more CO_2 is produced. Mostly on the basis of CO_2 levels, the body then increases ventilation. Hypoventilation can occur when the lungs function insufficiently because of lung disease—for example, in COPD. In that situation, oxygen levels can drop (hypoxemia), CO_2 levels rise, and the need for ventilation cannot be met, which results in dyspnea, or the sense of air hunger or laborious breathing. In patients who are prone to hypoxemia, it is therefore recommended to monitor pO_2 in the blood during exercise training and only to increase physical effort when normal oxygenation is maintained. This can be done quite easily by way of an oximeter through thin areas of the skin, for instance, an earlobe (Tiep, Burns, Kao, Madison, & Herrera, 1986). By contrast, hyperventilation occurs when ventilation is too great for the metabolic requirements of the moment. As a result, CO_2 pressure drops, resulting in hypocapnia, and the blood becomes less acid. Although the pO_2 may rise in acute hyperventilation, after some time, the pH increases, which causes oxygen to be bound more tightly to hemoglobin. This, paradoxically, results in less tissue oxygenation. Hypocapnia can be measured by capnography of the expired air, either exhaled through a mouthpiece or sampled from a tube in the nostrils. It may also be measured transcutaneously, but that is less accurate. Hypocapnia can lead to many complaints (see Table 12.2 later in the chapter). When there is no physical cause for hyperventilation, the excess ventilation is thought to be a problem of tension, anxiety, or faulty breathing (van Dixhoorn & Folgering, 2015).

The same amount of ventilation can be achieved through many combinations of CPM and V_t: Many small breaths per minute move the same amount of air in and out of the lungs as well as a few large breaths. Although mechanical constraints limit the number of possible combinations without extra work in breathing, the remaining range is large. So far, no optimal breathing pattern has been established. The actual choice of V_t and CPM, controlled by the breathing regulatory centers in the brain, depends much on the state of the organism as a whole and reflects its condition.

The effect of ventilation is that outside air, which is high in oxygen and low in CO_2, comes into contact with capillary venous blood, which is low in oxygen and high in CO_2, so that diffusion occurs. An important factor in respiratory regulation is the space between the opening for air (mouth and nose) and the lung alveoli. It is called "dead air space" because the air passes through it without actual diffusion. It consists of the throat, trachea, and bronchi. The size depends on the structure of the body, but it averages about 0.15 liters (L). Dead space can be enlarged by breathing through a tube, which decreases effective ventilation. When the tube is so large that the volume of the dead-air space equals tidal volume, there is no effective ventilation: No outside fresh air comes into the lung alveoli. Similarly, high-frequency breathing with very small breaths leads to very little effective ventilation, because the size of V_t approaches dead-air space. Its main use is to cool the body by the flow of air: inhaling cool outside air, which is heated inside the body and flows outside by exhalation (for instance, dogs or sheep use it for cooling). By contrast, when the body becomes more active and metabolism increases, ventilation increases first by an increase of V_t (Wientjes, 1993). The deeper breaths lead directly to more effective ventilation because the dead-air space becomes a smaller part of tidal volume.

The combination of time and volume results in flow: The flow rate of air during inhalation represents force of inhalation or respiratory drive. When more air is inhaled in a shorter time, the inhalation drive is high. This may occur when the person is dyspneic, for instance, if COPD is present, or when a novice diver breathes through a gas mask for the first time under water. It may lead to a breathing pattern of "gasping," that is, making great effort to inhale air. It also can happen in response to anxiety or emotional stress.

The contraction and expansion of the body is performed by the respiratory muscles, which change the volume of the trunk. When the volume of the trunk increases, the interior pressure decreases relative to the atmospheric pressure. When the airways are open, the air flows inside the lungs. However, the movement of expansion is made in the trunk as a whole, reaching from the first rib to the pelvic floor. Under resting conditions, about two-thirds of the volume changes are produced by the diaphragm. This is a double dome-like muscle that separates the chest from the abdominal cavity. When it contracts, it increases the size of the chest cavity in three directions. It moves downward, pressing on the abdominal cavity, and it lifts the lower ribs, which elevate sideways and in anterior direction (Kapandji, 1974). Other respiratory muscles include the intercostal muscles, some of which elevate the ribs for inhalation (specifically the upper and more frontal external intercostals; Campbell, 1957), whereas others bring them down for exhalation (especially the lateral and lower internal intercostals). The scalene muscles elevate the upper ribs toward the neck. The abdominal muscles compress the abdominal cavity and help the diaphragm to move upward for exhalation. The pelvic floor muscles resist the downward pressure of the diaphragm and help with exhalation.

A Systems Perspective

Breathing is dependent on many factors, both physical and mental, that influence its rate, depth, and shape. Within psychophysiology, respiratory measures function mainly as dependent variables, reflecting the state of the individual. Within *applied* psychophysiology, however, respiration also functions as an independent variable, a potential influence on one's state. Breathing is one of the vital functions that is open to conscious awareness and modification (Ley, 1994). The individual may voluntary modify breathing patterns in order to change mental or physical tension states. In a study by Umezawa (2001), such modification was found to be the most popular maneuver for managing stress. Thus there is a double relationship between breathing and the state of the system, represented in Figure 12.2, which makes matters rather complicated. The arrows that lead from respiration toward physical or mental tension state represent the regulatory role of breathing; the arrows that lead toward respiration represent its role as indicator. The distinction is important to avoid confusion, but it tends to be overlooked. For instance, breathing may respond to relaxation of the system, indicated by longer exhalation pauses or participation of the abdomen in breathing movement. These characteristics are then taken as a guide for regulation by practicing exhalation pauses or abdominal breathing. It does not follow, however, that the system automatically relaxes when these characteristics are imitated. Voluntary breath modification in a high-tension state may give a sense of control but may also disturb respiration even more. This explains why some individuals do not

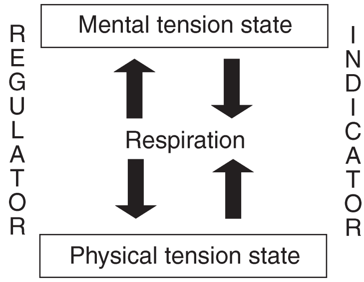


FIGURE 12.2. Model of double relationship between respiration and individual system.

respond favorably to simple breathing advice or instruction. The model further shows that measuring breathing to estimate the individual's state (indicator role) can be complicated by voluntary changes in breathing or control of breathing when the individual is aware that breathing is being measured. Focusing attention on breathing may modify it. It is, therefore, not easy to measure "spontaneous" breathing. Conversely, any breathing maneuver involves both respiratory changes and changes in mental and physical state. Thus, although its effects may be attributed to respiration, they also may be caused by concomitant changes in the entire body system that affect both respiration and the outcome parameter.

Paced breathing, for example, consists of modification of respiration rate but also involves focusing of attention and often a stabilization of posture, which have widespread effects as well. It is therefore important in studies of breathing therapies that respiratory measures be included to see whether breathing actually changes, although even in such cases the effects may also be attributed to other factors. For instance, Meuret, Rosenfield, Hofmann, Suvak, and Roth (2009) studied the effect of six sessions of breathing regulation, assisted by capnography feedback to raise CO_2, for patients with panic disorder. In addition, they applied mediation analysis to see whether the rise of pCO_2 caused the panic attacks to decrease or, vice versa, whether the cognitive changes were the cause of pCO_2 rise (see Meuret and Ritz, Chapter 11, this volume). The CO_2 changes clearly were primary. However, in a later study, it was found that lowering CO_2 was equally as effective as teaching clients to raise it (Kim, Wollburg, & Roth, 2012). The authors theorized that the participants in the study may have concluded from the sessions that their anxiety attacks were not due to an impending catastrophic event but simply related to breathing. In another study, it was found that respiratory muscle tension was an important factor (Ritz, Meuret, Bhaskara, & Petersen, 2013). Other studies examined the role of thoracic breathing as a concomitant of breathing discomfort and negative mood states. In one study, we confirmed that the quality of breathing movement (degree to which there was thoracic dominance) was an important mediator for the sense of dyspnea (Courtney, Van Dixhoorn, Anthonissen, & Greenwood, 2011). In another study, it was found that negative mood was an important mediator for respiratory variables, and especially for thoracic dominance (Lehrer et al., 2013). We conclude that physiological, as well as cognitive, factors, mood, and breathing movement are all important when explaining the effect of breathing interventions.

This model represents a systems view of respiration. It underlines the complexity of breathing instruction, which always includes both mental and physical components and effects. One consequence of the model is that breathing instruction consists of two parts—one in which breathing is consciously modified or regulated and one in which this regulation is consciously stopped. This is comparable to Jacobson's procedure of consciously tensing a muscle in order to learn to consciously stop muscle tension. One cannot ask the participant to stop breathing, but it is possible to stop a conscious regulatory practice. The purpose is to observe how the system responds to the regulation and whether there is a durable and stable effect on breathing after regulation has stopped. The instruction that regulates breathing is more like an invitation to the system to respond favorably than a dominant influence. To underline this, it is important to teach a specific skill to practice, but it is equally important to have the participant stop practicing.

Another consequence of the model is that breathing instruction may consist of instructions for posture, body movement, or attention, not even mentioning breathing explicitly, but influencing it indirectly. The list of practical strategies shows this clearly (see the "Instructions" subsection of the "Treatment Manual" section). A good example is an intervention that helps patients with lung cancer deal with dyspnea (Bredin et al., 1999), of which direct respiratory regulation is only a part. Once breathing has changed, this can spontaneously draw the attention of the patient, or the patient may be asked to pay attention to it. In scientific studies, the complexity is often overlooked or ignored to reduce the treatment to a reproducible protocol. However, the model specifies that such a reduction may be costly when the context of an instruction is as important as the instruction itself. For instance, many participants, particularly novice ones, have trouble performing a breathing instruction and initially use too much effort. This may lead to an overshoot and production of opposite effects. Such effects probably occur less often when sufficient attention is paid to the physical and mental tension states. For instance, Choliz (1995) reported on a highly effective breathing instruction for insomnia, in which underventilation gradually led to a state of drowsiness. He simply described the respiratory protocol without mentioning any strategy to make the system accept the instruction and facilitate a favorable response. The protocol was replicated (Hout & Kroeze, 1995) and led, in many participants, to *hyper*ventilation! This opposite effect is remarkable but can be ascribed in all probability to subtle differences between the original protocol and the replication. To repeat, physiological, as well as cognitive, factors, mood, and breathing movement are all important factors when explaining the effect of breathing interventions. A further consequence of the systems view is that it is very difficult to define "good" and "bad" or "functional" and "dysfunctional" breathing. A particular breathing pattern may look irregular or effortful but actually result from a specific factor within the system to which breathing responds. A good example is the breathing pattern of patients with COPD whose lungs are hyperinflated and whose diaphragms are maximally active. Their breathing is clearly upper thoracic and effortful, involving auxiliary respiratory muscles, and they are often told or taught to breathe more abdominally. In that condition, however, upper thoracic breathing might be a functional way of elevating the chest to inhale air, and "abdominal" breathing might not be functional at all and could even worsen their already insufficient ventilation (Cahalin, Braga, Matsuo, & Hernandez, 2002; Gosselink, Wagenaar, Rijswijk, Sargeant, & Decramer, 1995). However, upper thoracic breathing might also be a dysfunctional exaggeration of a functional adaptation. The sense of dyspnea, for instance, leads to a quick inhalation (Noseda, Carpiaux, Schmerber, Valente, & Yernault, 1994), which might result in insufficient time for adequate distribution of inhalation and thus to excess ventilatory effort. It is difficult, therefore, to differentiate between functional and dysfunctional breathing. In our method we do it by careful observation of whether instructions that aim to reduce unnecessary effort in breathing are successful in changing the breathing pattern and whether the sense of dyspnea responds to that (Van Dixhoorn, 1997; Giardino, Chan, & Borson, 2004; Thomas et al., 2003). It is important to include time for the response to occur after instruction. When a particular breathing pattern changes and respiration remains visibly less effortful after instruction, the original pattern was probably dysfunctional to some extent. This formulation refers to the first consequence of the model: that breathing instruction entails that conscious regulation is stopped. If not, the observed change in breathing pattern may be simply the result of conscious practice while being monitored or observed.

Definitions of Breathing

Breathing instruction and the sense of labored breathing, or dyspnea, can be viewed from various perspectives, depending on the definition of breathing (van Dixhoorn & Folgering, 2015). The most common definition of breathing refers to the *passage of air* for *lung function and ventilation*. Breathing is measured by way of lung function parameters such as rate, inhalation time, exhalation time, pauses, tidal volume, minute volume, flow (duty cycle), O_2 saturation, and end-tidal CO_2. The mechanics by which the air is moved in and out of the lungs are of secondary importance, because they hardly influence lung function. Dyspnea is a common complaint in lung disease, and the medical point of view is to objectify its basis in lung function. However, other factors than lung function can affect both air flow and dyspnea. For example, lung function does not account for the function of air passage in communication. Without air movement, there is no voice or sound, and the person cannot smell. The regulation of air passage to ensure speech and communication is highly complex and represents a different process from ventilation (Conrad, Thalacker, & Schonle, 1983). The behavioral demands contingent on communication mostly overrule the ventilatory requirements (Phillipson, McClean, Sullivan, & Zamel, 1978). Thus air passage has an important expressive function that is often neglected. The implication is that breathing difficulties may signify difficulties in social interaction and experience rather than ventilatory problems (Petersen, Orth, & Ritz, 2008).

A second definition refers to the *rhythmic expansion and contraction* of the body. This breathing motion serves, of course, to bring the air in and out of the lungs, but it has other functions as well. Breathing is a central pump, or oscillator, that moves various organs and the fluids throughout the body. For example, it acts to move venous blood, lymph fluid, and the cerebrospinal fluid. In addition to these hydraulic effects throughout the body, there is a clear oscillatory relationship with heart rate and heart rate variability that affects the autonomic nervous system (see Lehrer, Chapter 10, this volume). Next, the mechanical properties of volume changes have a dynamic of their own. The coordination of breathing movement determines, to a large extent, the effort of the pump and the sense of dyspnea but also affects movement and posture. Various components of the breathing apparatus play a role in posture, weight bearing, walking, and lifting objects, as well as in moving air in and out of the lungs. The qualities that apply here have to do with smoothness of movement, fluency, effortlessness, and coordination throughout the whole system, from head to feet.

A third definition of breathing refers to its role in *self-perception*. Like any movement, breathing is a sensory motor activity that serves to provide important feedback to the conscious individual about his or her state. The sense of freedom of movement or restriction in space, the sense of tension or relaxation within oneself, the sense of safety or danger within the environment, all have much to do with the quality of internal feedback (Petersen, Van Staeyen, Vogele, von Leupoldt, & Van den Bergh, 2015). Thus a person may feel free and at ease or restricted and even dyspneic because of changes in self-perception. An important aspect of breathing is, therefore, to what degree its sensation is accessible to conscious awareness without this awareness leading to disruption of the natural rhythm. For instance, patients with lung disease can be "nonperceivers," which means that they do not notice changes in lung function (Noseda, Schmerber, Prigogine, & Yernault, 1993). This is a risk because it prevents them from taking adequate measures in time, but it also appears that the response to medication is greater in "perceivers." Poor perceivers of respiratory sensations, for example, have more "near death" experiences from asthma (Kikuchi et al., 1994). In clinical practice, it appears that many individuals have little awareness of the quality of breathing and lack this sort of feedback. The purpose of breathing therapy is to enhance this awareness by inducing a marked improvement in perceived quality. At the same time, overconsciousness needs to be avoided. Breathing is a natural and automatic function that does better without constant conscious attention. For that reason, the indirect strategies are extremely useful.

These three perspectives are complementary; they represent three ways of looking at dyspnea and breathing difficulties, and they lead to different measurements and treatment strategies. Ideally, breathing therapy should take all three into account. It is interesting what the result of a dysfunction is in the latter two viewpoints—when the quality of breathing movement is low and effortful but at the same time self-perception is also limited. What will a person with these characteristics report? Clearly, nothing special. Although internal tension may be high, it does not enter conscious awareness. This is a quite common situation and may explain why breathing instructions can be met with mixed feelings. An increased ease in breathing may feel pleasant, but the increased awareness of the tension is unpleasant. It all depends on which one dominates.

Strategies for Breathing Regulation

This section describes strategies to modify breathing that are either direct or indirect, that often consist of combinations of breathing instruction with attentional and/or movement instruction, and that aim at breath or tension regulation or both.

Timing

Counting breathing is a common procedure that consists of coupling attention to breathing. In Benson's Respiration One Method (Benson, 1993), breathing is counted '1, 1, 1,' and so on, so that the person doesn't lose count, which would give rise to unrest. Breathing may also be counted from 1 to 10, or the inhalation and exhalation may be mentally followed with such words as *in, out, in, out* (Wollburg, Meuret, Conrad, Roth, & Kim, 2007). Attention can also be coupled to breathing without counting. The instruction may be to follow inhalation and exhalation mentally. *Pacing breathing* is used mostly to slow down breathing and may consist of the instruction to breathe at fixed rates: "in, 2, 3, out, 2, 3," or "1, 2, 3, 4" during inhalation and "5, 6, 7, 8, 9" during exhalation. There may be protocols for this, gradually increasing length, and it may be prescribed in a directive fashion or may be more open and free. The rationale is mostly that slower breathing leads to relaxation or that it increases CO_2 and reduces hyperventilation or both. Sometimes a device is constructed that indicates by a tone of varying pitch how long to inhale and how long to exhale. Again, this may be a fixed preset rate, or the instruction may depend on the actual breathing rate of the individual, in which case the instrument contains a sensor to measure that rate (Schein et al., 2005). Sometimes the rate is subsequently adapted to breathing measured during sessions (Grossman, Swart, & Defares, 1985) to achieve a feasible lengthening.

Focusing on *exhalation pauses* is a good way to lengthen breathing, because pauses naturally appear under relaxation conditions (Umezawa, 1992). Instructions that can help achieve this state are "in, out, stop" or "pause" or "in, 2, 3, out, 2, 3, pause 2, 3." In the Buteyko method, these pauses are gradually lengthened to approach almost 1 minute (Bowler, Green, & Mitchell, 1998). During transcendental meditation, they are observed to occur spontaneously for about 30 seconds, particularly during electroencephalogram (EEG) changes (Badawi, Wallace, Orme-Johnson, & Rouzere, 1984). In hyperventilation treatment, the focus on exhalation pause serves to increase CO_2. By contrast, pacing may also be used to increase respiration rate and decrease depth. An example is to count "in, out, stop" at such a pace that it results in a ratio of respiration to heart rate of approximately 1:4. Such short and shallow breathing helps to break an overly conscious pattern of slow breathing or to induce shallow breathing, which is useful when there is a persistent unproductive cough. The therapist should warn the client not to breathe deeply. This pattern fits a situation of high tension or challenge, for instance, during delivery of a child, but it is also useful to show that, during rest, reduced ventilation is sufficient.

Interestingly, short and shallow breathing during rest may lead to effortless breathing, whose movement is perceptible throughout the whole trunk. The reason is that a low volume requires little effort and leads to relaxation of respiratory muscles. Another variation is to pay attention to the *transitions* between inhalation and exhalation and exhalation and inhalation. The breathing cycle is divided into four parts: in, pause, out, pause. Attention is brought to the period when breathing reverses direction and stops for a brief moment. This is a natural control strategy that helps to focus and calm the mind and make breathing less hurried. It is useful when someone is dyspneic from lung disease, because it provides a small margin of control. It may be a starting point for gradually increasing the time period of the transitions.

Coupling to Movement

An indirect and natural way of pacing breathing is through *coupling to movement*. Any movement that has a periodicity similar to breathing tends to synchronize with it. Walking, cycling, or running tend to go easier when the rate of repetition has a whole-number ratio to the rate of breathing. When coupling has occurred, slowing down the movement tends to slow down breathing. For example, walking slowly for some time may gradually lead to slower and deeper breathing. At the same time, inhibition of habitual speed leads to increased mental focus and attention, which, in turn, favors slower breathing. When the goal is too slow, however, the effort to do it creates unrest and distraction and thus quickens respiration.

Small, repetitive movements are easy to couple to breathing: rolling the hands or arms in and out, moving the head up and down, pressing the fingers together and relaxing them, flexing and extending the feet. Single tense–release cycles can also be coupled to breathing, as is done in an abbreviated version of progressive relaxation, in which tensing a muscle is coupled to inhaling. This is a natural combination, but in the present method it is reversed: Tensing is coupled to exhaling (see the "Instructions" subsection). This combination requires attention and, therefore, acts as a focus of attention. Movements that flex and extend the spine play a special role, because this tends to couple mechanically with inhaling and exhaling. When running, animals such as dogs and horses tend to inhale when the four legs are spread out and breathe out when the legs are together. Similarly, the yoga exercise series "sun greeting" consists of an alternation of flexing the whole body (exhaling) and extending it (inhaling). On a smaller scale, in the sitting position, bending forward or sitting upright tends to extend the spine and couples with inhaling, whereas sitting backward in a slump tends to flex the spine and couples with exhaling (see "Instructions"). Using these combinations facilitates breathing instruction, but it may also be used with reverse coupling. The reason for *reverse coupling* is to break the habitual combination and thereby increase the flexibility and the area of breathing movement. For instance, sitting slumped or with head down helps the body to breathe in while the spine is flexed. Once this is possible, the movement to sit back and round the spine can be combined with inhalation and sitting upright with exhalation. This facilitates "width breathing," which may feel unfamiliar and strange. When someone gets the knack of it, however, the range of breathing movement increases, the diaphragmatic motion is stimulated (Cahalin et al., 2002), and dyspnea may decrease. Another option is to reverse the habitual combination of raising the shoulders while inhaling (see "Instructions"). These kinds of instructions may extend to ones in which movement and breathing are *uncoupled*. This increases flexibility of breathing. Also, breathing serves as an indicator of the effort involved in the movement. Thus, in Feldenkrais's method, a fully functional movement implies that it is carried out with undisturbed breathing. A simple example is rolling the head in the supine position, which tends to interfere with breathing until breathing has become more flexible and/or the rolling movement has become more effortless.

Air Passage

The passage of air is a good way to modify respiration, which is done naturally by patients with COPD who use pursed-lips breathing to lengthen exhalation when dyspnea occurs. The added resistance to the air by the lips helps to keep the airways open and postpones airway collapse, thus enhancing ventilation. Similarly, audible exhalation through the lips (see "Instructions") lengthens the outbreath. In this method, however, it is done with less force than in pursed-lips breathing and is combined with slow inhalation. After a longer exhalation, one tends to inhale hurriedly, and a fast inhalation tends to be an upper-thoracic movement with auxiliary breathing muscles, particularly when one has gotten out of breath. The resulting "gasping" inhalation confirms the sense of dyspnea. Gasping is prevented by the instruction to exhale gently and slowly. It results in generally larger tidal volumes and should be done a few times (5–6), after which normal nose breathing is resumed. This is to prevent hyperventilation.

Slow inhalation tends to improve the distribution of breathing movement, because all the components are allowed more time to become involved. It results in a larger volume, more involvement of the whole body, and less risk of hyperventilation, particularly when breathing though the nose. A good example is to think of smelling a nice fragrance, such as a flower. The image of enjoying the inhalation of the air slowly into the body adds to this effect. It may be combined with imagery of the airways and of the air passing from the tip of the nose through the inside of the nose and throat, down into the lungs and chest, and even further down the body. By contrast, mouth breathing tends to result in shorter inhalation times.

Resistance training is a technique for strengthening the power of inhalation muscles, such as the diaphragm. It can be done by breathing through a mouthpiece with a varying opening width, thus increasing the resistance and providing a training impulse to the muscles (Dekhuijzen, 1989). This is useful for patients with lung disease, but a recent study showed a good effect on exercise capacity and dyspnea in patients with heart failure (Laoutaris et al., 2004). A similar method of resistance training comes from voice training and is used by singers to open the upper airways. They breathe in through the lips, making a sound like "fff" from the lips, in order to increase resistance (Ulrich, 1928; Balfoort & Van Dixhoorn, 1979). This trains rapid and full inhalation, which is important for performance. Generally, inhaling through a mouthpiece or a tube tends to increase ventilation (Han et al., 1997). This effect compensates for the effect of breathing through a tube, which is used for hyperventilation complaints to increase dead-air space.

Distribution of Breathing

The shape or form of the volume changes with breathing can vary considerably, because the potential volume change in the trunk is much larger than is possible for the lungs. Thus the same ventilation can be achieved by different parts of the trunk, which ensures that ventilation can be maintained in very different postures. This leaves a large margin of flexibility and also allows conscious control and modification. Before practicing voluntary control of the location and form of breathing movement, however, it is important to realize that the areas of the body that are actively involved in breathing movement largely depend on posture, on mental state (focused or passive), on emotional or expressive state, and on physical tension state (energy and ventilation requirement, nervous tension). When a person is resting, mentally and physically, tidal volume is relatively low, primarily achieved by the diaphragmatic pump, and the muscles of the trunk are relatively relaxed. In this situation, visible breathing is mainly costo-abdominal: the lower ribs widen, and the abdomen expands with inhalation. The upper ribs and so-called auxiliary muscles, such as the scalenes, nevertheless also contract with each inhalation. This is necessary to prevent a slight collapse of the rib cage under the increased negative pressure inside that leads to the inflow of air (Decramer & Macklem, 1985). It is hardly visible, but its absence is not functional, and maintaining upper chest immobility should not be taken as a sign of relaxed breathing. When the activity and tension levels rise, volume increases and involves more movement of the rib cage, and the muscles around the abdomen may tighten a little. As a result, breathing becomes "higher." From this natural response, many strategies advocate that breathing remain "low" in the body while under stress or during greater activity. See also the instruction in Table 12.3 (later in the chapter).

Thus abdominal breathing, diaphragmatic breathing, and slow deep breathing are common practices, probably the most common (Gevirtz & Schwartz, 2005). This strategy is quite effective for remaining or becoming quiet and calm and reducing stress (Czapszys, McBride, Ozawa, Gibney, & Peper, 2000; Peper & Tibbets, 1994). The person is taught to put the hands on the abdomen (or the therapist does so) or to put a weight, such as a book (Lum, 1977), on the abdomen in the reclining position and make it move up and down with breathing. Lum (1981) reported 75% success among more than 1,000 patients with anxiety and hyperventilation using this method. It is particularly useful in individuals who demonstrate an exaggerated response to the rise of tension, which is dysfunctional and dysponetic (Whatmore & Kohli, 1974) and which can be reduced in this way. This makes breathing a quick and easy tool for handling stress.

However, two points need to be considered. First, the effect of this strategy may not be due to breathing itself but to the concomitant shift in attention, which is directed to the center of the body. This is the area of the center of gravity, and, as such, it represents a neutral ground for attention, less threatening or challenging than the visual perspective in front or in one's mind. This reduces the mental tension state. Attention in the center also implies that body movement tends to become more functional and less effortful. This reduces the physical tension state. Thus breathing may simply be the tool to induce these shifts. A second point is that the emphasis on abdominal movement may lead to the mistaken notion that the (upper) chest should be immobile. As stated earlier, reducing exaggerated upper thoracic breathing does not imply that the upper ribs should not move at all. Functional inhalation requires the rib cage to change its shape as a whole (Balfoort & Van Dixhoorn, 1979; Bergsmann & Eder, 1977; Parow, 1980).

Functional upper chest movement is important for breathing and, in particular, for emotional freedom of expression, as well as for voice production. It is intimately linked to an adequate use of the upper back. In the best singers, the upper chest rises simultaneously, with a slight lengthening of the upper spine, thereby increasing the length of the scalene muscles and making their contraction more effective. The head is tilted slightly forward, relative to the neck, which moves slightly backward. Thus the head remains still. This pattern is evoked by the beginning of a yawn (Xu, Ikeda, & Komiyama, 1991), whereby the throat and vocal cords descend. This favors voice production. It is the basis for the instruction “looking up and down” (see “Instructions”). The opposite pattern is seen in a dyspneic person, whose head is tilted backward relative to the neck, increasing lordosis of the neck during inhalation. This moves the head frontally and up, which appears as a movement of gasping for air. Instructions that promote functional upper chest breathing are also important for neck problems.

Another aspect of location of breathing movement is the *pelvic floor.* This lower diaphragm is a natural antagonist of the middle respiratory diaphragm, and their functions support each other. When the respiratory diaphragm contracts during inhalation, the pelvic diaphragm relaxes, and vice versa. Adequate contraction of the pelvic floor is necessary to carry the weight of the internal organs and to counteract the force of breath holding when lifting or carrying a weight. Thus pelvic floor dysfunction tends to compromise breathing, as well as posture. The instruction “sitting, standing” aims to facilitate pelvic floor contraction when getting up because of its coupling to exhalation. This helps to prevent urine leakage in women who suffer from this problem. Another option is the Muslim prayer posture, in which relaxation of the pelvic floor during inhalation can be observed.

Focus of Attention

Providing a *single focus of attention* is the most common way to relax and reduce tension (Benson, 1993), and it also tends to quiet and regulate breathing. Its effect on breathing does not require a focus on respiration. For some, it is best not to focus on breathing directly, as that tends to disturb it and to cause overconsciousness and dysregulation. The object of attention may be breathing movement anywhere in the body, sound or sensation of air passage, or respiratory feedback signals, but also the sense of body weight in sitting, standing, or lying quietly or during movement, the sense of touch by the therapist or oneself, words that are repeated to oneself, or any visual or auditory focus. Another dimension of attention is active versus passive concentration. *Passive attention,* or *receptivity,* is a hallmark of relaxation (Smith, 1988) and can be seen as a prerequisite for self-regulation of tension (Peper, 1979). In particular, it is present when the indicator role of breathing is emphasized. In an older study, Burrow (1941) found that during “cotention,” which is like an unfocused gaze in the distance, respiration rate drops greatly. He associated this with a state of mind in which the individual is in more direct contact with the whole organic system of the body. In a more recent study, passive attention, or mindfulness, was found to be associated with a different pattern of EEG activity than was focused attention (Dunn, Hartigan, & Mikulas, 1999). Thus the very attitude of passivity and not being goal directed may induce a change, including a respiratory response.

An intriguing aspect is a relationship between the *object of attention* and *distribution of breathing*. Respiratory movement follows the direction and content of attention. For instance, calling attention to the supporting ground for the body on the backside and also emphasizing the width of the body may help distribute an evenness of breathing in the body. In the instruction "circling knees," the repetitive movement of rolling over the sitting bones draws attention to the supporting ground in a passive way. Awareness of the width of the eyes and the distance between the outer corners of the eyes or of the corners of the mouth and slightly increasing their distance induces the beginning of a smile, as well as a sense of breathing very easily (Van Dixhoorn, 1998b). This relationship is also present in the influence of attention on the *direction of inhalation*. Although the diaphragm moves downward as it contracts, a common image of inhalation is a movement upward, as if drawing in the air from above. This is strengthened when one is experiencing dyspnea. By contrast, the image of inhalation as a downward movement helps to let the air flow in easily and reduces dyspnea.

Feedback Devices

Various measurements of respiration can be used as a source of biological or instrumental feedback. A detailed description is given by Gevirtz and Schwartz (2005). An obvious parameter is CO_2 feedback, using capnographic measurements (van Doorn, Folgering, & Colla, 1982; Meuret et al., 2009; Terai & Umezawa, 2004). The patient may or may not receive instructions for breathing. The main purpose is for CO_2 to reach normal levels. CO_2 biofeedback is particularly useful when hypocapnia is present. Similarly, feedback of oxygen tension is useful when PO_2 is low (Tiep et al., 1986). Another parameter is feedback for respiration rate. In contrast to paced respiration, in which a specific frequency is given, respiration rate feedback does not impose a frequency but only provides feedback of actual respiration rate. It appears to have a soothing influence, and respiration rate gradually slows down (Leuner, 1984; Schein et al., 2005; Zeier, 1984). A more direct approach (MacHose & Peper, 1991) is to use measurement of muscle tension and to teach a specific way of breathing that does or does not involve these muscles (Johnston & Lee, 1976; Kotses et al., 1991; Reybrouck, Wertelaers, Bertrand, & Demedts, 1987; Peper & Tibbets, 1992; Tiep, 1995). In these approaches, the breathing strategy is the prime intervention, and the feedback device serves as an aid for teaching it. Another intervention is to give feedback for tidal volume in order to make patients aware that they tend to hold their breath when under stress. This may or may not be accompanied by teaching strategies for inhaling more effectively (MacHose & Peper, 1991; Peper, Smith, & Waddell, 1987; Roland & Peper, 1987) An overview of biofeedback techniques for lung patients is given by Tiep (1995). Finally, a more recent form of biofeedback is resonance feedback, in which the parameter consists of heart rate variability that increases with slow breathing (Del Pozo, Gevirtz, Scher, & Guarneri, 2004; Lehrer et al., 2004).

Whole-Body and Spinal Column Involvement

Given the many interdependencies of respiration that follow from the system's perspective, the method on the one hand seeks orientation in specifying attention and posture and on the other hand follows the skeletal structure of the body. A model was developed during the 1980s that specified the relationship of the spinal column, the core structure of the skeleton, to respiratory movement (Van Dixhoorn, 1997). The spinal column connects the rib cage with the head and the pelvis. When it is extended, standing upright or in the supine position, respiration involves a minute wave-like motion in the spine, which is more like a preference for motion than an actual visible movement. It originates from the rib cage, which makes a rolling movement during respiration. Inhalation involves an upward rolling movement of the chest, which is accompanied by a preference for slight lumbar lordosis and flattening of the cervico–thoracic junction. The opposite preference is present during exhalation. Therefore, small movements, initiated from the legs, arms, or head, are able to influence respiration indirectly. This is the first pattern of interaction between spinal column and respiration. The coupling of inhalation to extension is called "length" breathing. It is complemented by the opposite pattern of "width" breathing. When the spinal column is flexed—for instance, in a slightly slumped sitting posture—the connections of the first pattern are blocked. This is also the case when a person lies prone. In that situation, the rib cage cannot roll upward, and the cervico–thoracic junction cannot flatten during inhalation. Instead, the costo-abdominal circumference expands and lumbar lordosis flattens during inhalation. Because of the emphasis on sideways expansion, it is called *width breathing.* Breathing in both directions, horizontally and vertically, allows the body to respond flexibly to various postures. Therefore, the degree to which both patterns can be utilized is an important indicator of functional breathing and serves as a parameter of the success of breathing therapy.

These connections are the background for many instructions of the method. They also help to deal with the issue of attention and of conscious control of breathing in several ways. First, the perception of "whole body involvement" invites a more passive concentration than attention that is actively focused on one particular area or movement. The instruction starts with one area or movement and then invites the individual to notice connected movements all over the body. Second, the periphery (arms and legs) and the back are, for most individuals, not consciously related to breathing. Thus facilitation of breathing through the instructions happens unwittingly and does not elicit conscious control. Third, the skeletal connections promote a functional use of the muscles, which tends to lead to a greater ease of movement and of breathing. The patterns may not be fully habitual, and the instructions may feel strange at first, but they tend to remain present after the period of conscious practice. Thus the instructions tend to generalize and become part of automatic movement.

Flexibility and Variability

In the 1990s the Dutch branch of what later became the International Society for the Advancement of Respiratory Psychophysiology (ISARP) set up a series of meetings to discuss the criteria for what constitutes a proper breathing pattern. It was also a theme at the international meetings (Ley, Timmons, Kotses, Harver, & Wientjes, 1996). In the end, not a single characteristic could be validated to serve as such a criterion. This conclusion supported the assumption of this method that a key characteristic of functional breathing is its variability. The manual (Van Dixhoorn, 1998) stated that, rather than working toward a particular pattern of breathing, the main goal is that breathing should be flexible and should adapt to changing demands without causing a sense of effort. This goal has several implications.

1. Functional breathing responds to changes in the environment. For example, a respiratory response to imagery cues, touch, speech, posture, or movement is likely to be noticeable. Many instructions from the method have as their purpose to test and promote this responsiveness.
2. Variability in breathing is a sign of healthy function. Donaldson (1992) found irregularity in all respiratory parameters: lowest for CO_2, highest for expiratory

time. He concluded “that the chaotic nature of resting human respiration allows for fast and flexible responses to sudden changes” (p. 313), which strengthens the stability of the system as a whole (Vlemincx et al., 2013). In some studies, variability was associated with a positive emotional state (Boiten, 1998). The power spectrum of variations is probably complex, because many determinants exist.

3. High irregularity is not always healthy—for instance, when it is the result of two conflicting determinants. Highly anxious people tend to have a high standard deviation around the mean respiration rate because of an alternation between relatively fast regular breathing and deep sighs (Wilhelm, Trabert, & Roth, 2001). The power spectrum is probably less complex.
4. Healthy variability includes adequate recovery from a stimulus, which includes the response to hyperventilation provocation. A delayed recovery of CO_2 after fast, deep breathing or a single deep breath can be seen, therefore, as a sign of reduced flexibility (Wilhelm et al., 2001)
5. The criterion of flexibility also includes the possibility of slow, deep breathing. Although there is increasing evidence that periodic slow, deep breathing is healthy (see Lehrer, Chapter 10, this volume), it cannot be stated that one should always breathe that way. Flexibility entails that slow, deep breathing should occur or be possible under proper conditions and without giving rise to a sense of dyspnea.

To summarize, an individual with an erratic, highly irregular breathing pattern may benefit from measures to introduce rhythm and stabilize breathing. However, a too-regular pattern is not optimal, and the person may benefit from measures that require variable respiratory responses. Vlemincx et al. (2013) found that a certain amount of sighing, which produces a certain amount of irregularity in breathing, is necessary for respiratory regulation.

ASSESSMENT OF EFFECTS

Respiratory and Physiological Effects

The method has been tested so far in one clinical trial in which 156 patients with myocardial infarction were randomly assigned to exercise rehabilitation only or to exercise plus six individual sessions for breathing and relaxation therapy (Van Dixhoorn, 1990). Although the breathing method did not focus on teaching a particular form of breathing and utilized mainly an indirect, “whole body” approach to respiration (some of the instructions are described here), respiratory changes did occur and figured prominently among the physiological effects. Respiration was measured in a protocol of 20 minutes’ duration, consisting of standing, lying down, slow breathing, lying still for 6 minutes, and slow breathing. Care was taken that the patients did not practice any technique during testing. Average respiration rate at pretest was about 15 cycles per minute (CPM) in both groups. It remained at this frequency in the control group but dropped in the treatment group to 12 CPM at posttreatment and at follow-up (Van Dixhoorn & Duivenvoorden, 1989). The difference was small but statistically highly significant. Figure 12.3 shows that a small but consistent reduction occurred across all measurements of spontaneous respiration (time periods 1, 2, 4, 5) but not during slow, controlled breathing (time periods 3 and 6). Thus the responsiveness of respiration to standing, lying down, and quietness remained intact. The small increase during quietness (time periods 4 and 5) indicates that patients were not practicing or controlling breathing. During the 20-minute testing

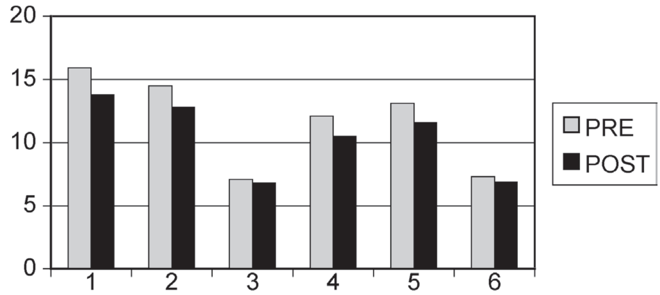


FIGURE 12.3. Respiration rate before and after breathing therapy in patients with myocardial infarction. 1, standing; 2, after lying down; 3, 6, slow, deep breathing through mouthpiece; 4, 5, beginning and end of 6 minutes of lying quietly.

period, estimated minute volume decreased in both groups, before and after rehabilitation, indicating lowered arousal from lying down and quietness. In the experimental group, significantly more patients reported pleasant body sensations during testing after rehabilitation.

The effect on respiration rate was evident even at 2-year follow-up, when respiration was measured in 38 patients. Two patients from the treatment group appeared to practice the breathing technique during measurement. Their respiration rate was about 7 CPM, and they were excluded from analysis. The control group was still breathing at a rate of 15 CPM, and the treatment group continued to show a reduction to 11 CPM. Further analysis showed that all of the time components lengthened somewhat but that the reduction was mainly due to an increase in exhalation pause time (Van Dixhoorn, 1994). This indicates that the patients were breathing in a more relaxed way. Another aspect was the distribution of inhalation movement, assessed in the sitting position (see the subsection "Assessment of Breathing" in the "Treatment Manual" section later in this chapter). The distribution of breathing in the treatment group was lower in the body and remained lower during deep breathing. This indicates that the participants in the treatment group were sitting in a more relaxed way, less upright, and, as a result, were breathing a little more slowly and abdominally. This supports the conclusion that the reduction of respiration rate was not the result of training aimed at this particular goal but more likely represented an increased restfulness in breathing, which probably coincided with, and at least did not conflict with, physical and mental tension state.

Heart rate and heart rate variability (HRV) data were available for 76 patients before, after, and at 3-month follow-up (Van Dixhoorn, 1998a). Heart rate was reduced in both groups, which is to be expected, as both underwent physical training. However, it was more pronounced in the relaxation group, particularly at 3-month follow-up, when heart rate rose a little in the control group but continued to decline in the treatment group. This indicates a stable reduction in physical tension state. However, this reduction was associated with the lower respiration rate, and the effect disappeared when respiration was factored out. Thus a lower respiration rate may have contributed to a state of lower arousal (regulator role), or it may have reflected a state of lower arousal (indicator role), or both may have been the case. The same held true for HRV. It remained unchanged in the control group but steadily increased with time in the treatment group. The difference was statistically highly significant but, again, was associated with respiration rate. When respiration rate was controlled for, statistical significance disappeared, except when comparing HRV at the end of the 20-minute testing period. The latter fact may mean that spending 20 minutes in the supine position was more restful and resulted in greater relaxation for the treatment group. Mental relaxation is known to result in increased HRV (Sakakibara, Takeuchi, & Hayano, 1994). Two years after treatment, there no longer were effects on heart rate or HRV. Thus the effect on breathing pattern appears to be a rather specific result of treatment, not only a reflection of lowered arousal.

Evidence of Clinical Effects

The effects of the trial with patients with myocardial infarction on clinical parameters are summarized here and discussed in the light of other studies, which were reviewed (Van Dixhoorn & White, 2005) . It appeared that breathing therapy had multiple effects on psychological, social, and physical states and on prognosis. It clearly increased a sense of well-being. Patients felt more at ease and more relaxed (Van Dixhoorn, Duivenvoorden, Pool, & Verhage, 1990). Interestingly, patients who improved in well-being in the treatment group were breathing more slowly than at baseline, whereas patients who did not improve or patients in the control group did not change in respiration rate (van Dixhoorn & Duivenvoorden, 1989). Data on exercise testing showed that breathing therapy improved the effect of exercise training. It significantly reduced, by half, the occurrence of myocardial ischemia (ST-depression; Van Dixhoorn et al., 1989). This unexpected effect could not be accounted for by heart rate reduction. It was confirmed in three other studies (Van Dixhoorn & White, 2005). Also, the outcome of exercise training was assessed by a composite criterion of all parameters of exercise testing, which divided patients into those groups with a clear benefit, those with no change, and those with clear deterioration. It appeared that the occurrence of training failure was reduced by half when relaxation was added to exercise training. The outcome was not associated with respiration rate. There was no effect on blood pressure.

In the longer term, breathing therapy improved return to work to a moderate degree, an effect that was confirmed in two other studies (Van Dixhoorn & White, 2005). The occurrence of cardiac events (cardiac death, reinfarction, coronary artery bypass graft) was reduced by half over a 5-year period (Van Dixhoorn & Duivenvoorden, 1999), particularly the need for open-heart surgery after infarction (Bondi-Zoccai et al., 2016). This long-term clinical effect was confirmed in several other studies over varying time periods (Van Dixhoorn & White, 2005).

From these outcomes, it may be concluded that therapies involving relaxation and breathing are effective, but also that stress and tension play an important role in the condition of cardiac patients and that its management is beneficial to them in many respects.

The same composite criterion for training outcome was later assessed in 138 patients with myocardial infarction or coronary surgery, all of whom attended one group session of relaxation therapy alongside regular exercise training. In addition, 54 of them were referred for individual relaxed breathing therapy. The percentage of patients with clear benefit was larger in those who had participated in individual therapy (67%) than in those who had not (48%). This confirmed the previous finding that relaxed breathing therapy improves the effect of exercise training.

A national Dutch Web-based registry was set up to assess the clinical effects and responsiveness of clients who were referred to a certified therapist. The protocol was approved by a regional medical ethics committee in 2010. The idea was to find out what the indications for the method were and which were clinically successful (Van Dixhoorn, 2017), that is, those in which habitual tension was not complicated by blocking stressors.

We took the experienced complaints as a starting point and distinguished four categories: (1) with a specified medical condition, (2) with a psychological diagnosis, (3) with functional problems of musculoskeletal nature, and (4) without specific cause. All certified therapists could participate and upload treatment data, which were anonymous. More than 5,000 records up to 2015 were uploaded. This database served as a stock from which several students could draw for their doctoral theses. It appeared, as expected, that more than half were referrals for "tension related" problems, of which hyperventilation and functional breathing problems were the most common. Problems with movement were also common (17%), including neck and back problems and chronic pain. Another large group consisted of clients with problems with specific somatic diseases, of which heart, lung, and neurological diseases were the most common. Finally, 14% had psychological problems, mainly anxiety and depression. The responsiveness was, on the whole, positive. In about 40%, blocking stressors complicated treatment. These patients showed less effect on the Nijmegen Questionnaire (NQ), a measure of functional respiratory complaints with overlapping hyperventilation symptoms (Van Dixhoorn & Hoefman, 1987), but they still could benefit from treatment—that is, their NQ scores remained above normal, but they did improve in a statistically significant manner. In responsive clients, NQ scores dropped to near normal values. This confirmed the idea that the problem was largely due to habitual tension and that this had been alleviated. Table 12.1 (later in the chapter) lists conditions for which breathing and relaxation therapy has been applied successfully.

In one study, the outcome was assessed in routine practice, using Jacobson's analysis of clinical significance (Jacobson & Truax, 1991). The purpose was to establish the proportion of patients who reliably improved and those who improved enough to be classified as "recovered" (Wakker, Bosscher, & Van Dixhoorn, 2009). A total of 146 patients who completed the NQ were included. Most frequently occurring complaints were stress, hyperventilation, inadequate sleeping, and anxiety-related problems. The analysis of clinical significance showed that 70% of the patients reliably improved, and 60% improved enough to be considered as having recovered. Patients with hyperventilation complaints had higher improvement and recovery rates compared with patients with sleeping and anxiety-related problems.

In another observational study, we investigated whether patients with mainly anxiety complaints but without a DSM-IV diagnosis responded differently from patients with a DSM-IV diagnosis (Mataheru, Overbeek, Scheffers, & Dixhoorn, 2014; Van Dixhoorn et al., 2017). Of 255 participants, 205 did not have a DSM-IV diagnosis, and 50 (19%) did. Complicating factors appeared to be present in 47% of the patients. Factors associated with complications were: the presence of a DSM-IV diagnosis; not being employed full time; having anxiety but no breathing complaints at baseline; the persistence of anxiety complaints; having had treatment aborted within the screening phase of 3–4 sessions; and persistent elevated NQ after treatment. We concluded that our screening procedure selected more clients with complex situations deserving attention than indicated by presence of a DSM-IV diagnosis alone.

In an observational study of 145 patients with burnout, adequate tension regulation was assessed (Van Dixhoorn, 2013). Tension-related complaints were measured before and after treatment using a validated questionnaire for general distress state (GDS). Treatment of 145 participants took, on average, seven sessions. GDS improved significantly ($p < .001$). Most (73%) patients did not exhibit stressors (they were absent, not relevant, or were initially present but had changed and were no longer blocking or prohibiting effect). In the absence of stressors, the percentage of study participants working full time increased from 20% to 57%, and GDS normalized in two-thirds of them. Among them, the number referred initially for the treatment by the occupational medical doctor and the number with trouble sleeping were higher; moreover, the patients in whom fatigue disappeared was high (89%). When stressors were present, the percentage working full time remained low (13%), and GDS normalized in only 20%. It was concluded that a process-oriented approach is a valid way to differentiate patients and that tension regulation is a realistic treatment option, particularly in the first phase of treatment of patients with burnout.

A student of osteopathy was curious about the occurrence of dysfunctional breathing (DB) among clients with chronic nonspecific back and neck pain (Jansen, Dixhoorn, & Eupen, 2015). He used three parameters to assess breathing dysfunction: the NQ, a manual assessment method known as the Manual Assessment of Respiratory Motion (MARM; Courtney, Van Dix0hoorn, & Cohen, 2008) and capnography. There were 37 (54%) patients with DB and 31 (46%) without. The three measures of DB were significantly correlated: between exhaled CO_2 and MARM (r = .67), between MARM and NQ (r = .59) and between CO_2 and NQ (r = .40). Complaints were measured before, after, and at 1-month follow-up, with a Visual Analogue Scale (VAS) of pain intensity and a measure of functional disability. At the start, the group with DB had many more complaints. After treatment, DB was reversed in half of the cases, whereas VAS and functional ability were normalized. It was concluded that breathing was of importance in osteopathy.

A number of older studies focused on hyperventilation. In one study, 12 patients diagnosed as having hyperventilation syndrome were compared with 13 patients with nonspecific pain, mainly in head, neck, and back, and also with elevated scores (> 20) on the Nijmegen hyperventilation questionnaire (Van Dixhoorn & Hoefman, 1987). It appeared that NQ scores were reduced in both groups and that the main complaint improved in both groups equally. Thus breathing therapy is useful when hyperventilation complaints are present, irrespective of the diagnosis of hyperventilation syndrome.

In a second, unpublished, study, 55 hyperventilation patients were followed up for 3 years after breathing therapy. The average decrease in the NQ, from 29 to 24, was highly significant. This agrees with two other long-term follow-up studies of breathing intervention (DeGuire, Gevirtz, Hawkinson, & Dixon, 1996; Peper & Tibbets, 1992). However, only 33 (61%) reported major and stable improvement of their main complaints at the time they were treated. These treatment responders showed high reductions in the NQ. Interestingly, nonresponders had high initial scores on the NQ (about 30), which did not change much. An important finding was that very few patients (8%) needed a second or third round of treatment sessions with breathing therapy. It seems, therefore, that patients with hyperventilation who respond to breathing therapy receive lasting and sufficient benefit but that almost half of them do not respond or need additional psychological treatment.

Before beginning treatment, it is important to verify whether a given problem is suitable for this method, that is, that the problem is at least partly caused by dysfunctional tension and/or dysfunctional breathing. The list of diseases, complaints, or problems that are, at least in part, due to dysfunctional tension is obviously quite long, and there is much evidence for positive effect on a wide variety of conditions. These conditions are listed in Table 12.1, divided into four categories. They resemble the list of conditions for breathing therapy in Germany (Buchholz, 1994; Mehling, 2001). Treatment of patients with specific somatic causes often occurs in specialized settings such as a rehabilitation clinic, but all conditions are also mentioned by therapists in private practice.

TABLE 12.1. List of Conditions for Which Breathing Therapy Has Been Applied Successfully

Tension-related problems without specific cause • Feelings of tension • Hyperventilation complaints • Burnout • Headache • Chronic fatigue • Sleeping problems • Concentration problems	*Functional problems of musculoskeletal nature and breathing* • Lower back, shoulder, and neck complaints • Shortness of breath • Chronic pain (repetitive strain injury, whiplash, fibromyalgia) • Functional voice disorders, dysphonia, stuttering
Psychological problems • Anxiety and phobia • Panic disorder • Depression	*Tension problems with a specific, somatic cause* • Lung disease (asthma, COPD) • Heart disease (myocardial infarction, arrhythmia, CABG) • Neurological disease (hemiplegia, Parkinson disease)

SIDE EFFECTS AND CONTRAINDICATIONS

Although very few side effects have been reported in the literature, clinical experience affirms that they do occur and are reason for caution. Some of them are nonspecific; others are specific for breathing.

Hyperventilation or Functional Respiratory Complaints

The most common and specific side effect of breathing interventions is hyperventilation. The word *hyperventilation* is also used to characterize complaints associated with breathing, in a broader sense. Nowadays these problems tend to be categorized under "dysfunctional breathing" (Boulding, Stacey, Niven, & Fowler, 2016; Van Dixhoorn & Folgering, 2015).

Overventilation occurs when individuals use too much effort, breathe too deeply, or perform regulated breathing exercises for too long a time. Tidal volume increases, and respiration rate decreases as well, because larger inhalation volumes take more time. These two effects counteract each other. However, when volume increases more than frequency drops, minute ventilation increases, and, after some time, hypocapnia occurs. The inverse relationship between volume and rate may not be sufficient to prevent hyperventilation. For instance, when V_t increases from 0.3 L to 0.6 L and frequency drops from 16 to 8 CPM, the resulting minute volume remains the same: 4.8 L. However, effective ventilation is ventilation minus the dead-air space. Thus, effective ventilation actually increases from $(0.3–0.15 \text{ L}) \times 16 = 2.4$ L to $(0.6–0.15 \text{ L}) \times 8 = 3.6$ L. When one maintains such slow, controlled breathing for some time, hypocapnia is the result. Although it is quite common, few authors have reported it (Hout & Kroeze, 1995; Terai & Umezawa, 2004).

The experience of hypocapnia seems fairly accurately represented by the NQ (see Table 12.2). However, the correlation between end-tidal CO_2 and the NQ is variable and usually lower than might be expected (Courtney, Greenwood, & Cohen, 2011; Grammatopoulou et al., 2014; Hornsveld & Garssen, 1997; Wientjes & Grossman, 1994). Therefore, NQ is not a reliable tool for diagnosing hypocapnia. In a recent editorial, Folgering and I specified that the NQ reflects "a subjective aspect of dysfunctional breathing" (Van Dixhoorn & Folgering, 2015). High NQ scores only mean that the individual has more

TABLE 12.2. Symptoms of True Hypocapnic Hyperventilation Compared with Isocapnic Hyperventilation

General symptoms	
• Dizziness (NQ)	• Hot flashes
• Paresthesias (NQ)	• Headache
• Faintness	• Muscle weakness
• Muscle stiffness (NQ)	• Stiffness around the mouth (NQ)
• Cold hands or feet (NQ)	• Warm feeling in the head
• Shivering	• Sweating
• Muscle cramps	• Blurred vision (NQ)
• Fatigue	• Rapid heartbeat (NQ)
Respiratory symptoms	*Psychological symptoms*
• Tightness in the chest (NQ)	• Unrest/tension (NQ)
	• Anxiety/panic (NQ)
	• Feelings of unreality (NQ)

Note. (NQ), items from the Nijmegen Questionnaire.

complaints about breathing, and more negative awareness of it, than is normal. We prefer to describe the NQ as a measure of "functional respiratory complaints." Normal NQ values range from 12 ± 7 (7 = 1 standard deviation), and abnormal values are > 19. There are many reasons for NQ elevation, including psychological, medical, and behavioral. I have for many years tried to pinpoint a specific pattern of complaints to elevated NQ scores (Van Dixhoorn & Anthonissen, 2015). A scientist does not like to leave an interpretation open, but, in reality, an elevated NQ can be interpreted in many ways: It can be sign of a lung disease (Grammatopoulou et al., 2014; Thomas et al., 2009), but also of burnout (Ristiniemi, Perski, Lyskov, & Emtner, 2014), neck pain (Jansen et al., 2015; McLaughlin, Goldsmith, & Coleman, 2010), upper thoracic breathing (Courtney et al., 2011), nasal congestion (Hanna, Woodman, & Adair, 2012), and, of course, stress and anxiety (Han, Stegen, Schepers, Vandenbergh, & Van de Woestijne, 1998). It "detects [a] transdiagnostic and probably nonmedical abnormality" (van Dixhoorn & Folgering, 2015). Moreover, in a process-directed approach, it is recommended to leave matters open and to wait and see how the person responds to treatment (Van Dixhoorn et al., 2017). In fact, the multi-interpretability makes the NQ a good measure of the subjective experience of dysfunctional breathing. However, if you want to make a statement about the actual breathing pattern, you should add at least one somatic measurement (Barker & Everard, 2015; Courtney et al., 2011; Hagman, Janson, & Emtner, 2008, 2011; Hagman, Janson, Malinovschi, Hedenstrom, & Emtner, 2016) in order to make a multicomponent assessment.

Increased Unpleasant Awareness

A second common side effect of breathing therapy is the unpleasant confrontation with bodily functions that hitherto have remained unconscious. Breathing is largely automatic, but breathing regulation leads necessarily to an awareness that helps to deal with tension but that may also be disturbing. Even in my first study on cardiac patients, it became clear that we should assess which patients welcomed the awareness and found it helpful and which did not. Some clearly found the procedures very strange, and some found that it disturbed their habitual use of themselves. We divided the patients into three groups: those who clearly practiced, those who clearly did not or did not practice correctly, and those in between. The first group did much better in the psychological evaluation, except for the degree of well-being; all patients were doing better in that respect (Van Dixhoorn, 1985). Clearly, here was a factor of social desirability at work. All patients wanted to relax more, and they reported as such, but they did not all have the same ability.

This experience confirmed that awareness is important in relaxation and that it can be both pleasant and unpleasant. In fact, when both are the case, it is often a sign of increased awareness. Nummenmaa and colleagues (Nummenmaa, Glerean, Hari, & Hietanen, 2014) asked a sample of healthy people to draw where in the body they felt certain emotions. The responses indicated that only happiness was felt in the whole body. Therefore, I think that the emphasis we put on the whole body has a special meaning. We have instructions that involve the feet, the lower back, putting weight on the feet sitting and standing, and so forth. A general instruction to pay more attention to the feet or "feel the feet" is too vague. More specific instructions are given in our method.

The unpleasant awareness may be characterized by an increasing sense of dyspnea or may extend to feelings and sensations in general (Craig, 2010; Petersen et al., 2015; Rosenkranz & Davidson, 2009; Stoeckel, Esser, Gamer, & von Leupoldt, 2018; von Leupoldt et al., 2006). Such unpleasant experiences were assessed by a checklist that contained six pleasant and six unpleasant experiences of breath regulation; for instance, more quiet versus less quiet, more tired versus less tired (Van Dixhoorn, 1992). It was completed by 181 students in a professional course in breathing therapy and by 144 patients with hyperventilation complaints. It appeared that the two scales were unrelated; that is, unpleasant sensations occurred independently of pleasant experiences. The students had six times more pleasant than unpleasant experiences. Only in 4% of responses were the unpleasant experiences as large as the pleasant experiences. However, students with relatively high scores on the Dyspnea subscale of the NQ had more unpleasant and fewer pleasant experiences. The experiences were not related to anxiety. This confirms that negative awareness of breathing (dyspnea) is a specific reason for unpleasant sensations in general after breathing therapy.

In the patients with hyperventilation complaints, the unpleasant experiences were, on average, more frequent and the pleasant experiences less frequent than in the students (Van Dixhoorn, 2002). Still, pleasant experiences outweighed the unpleasant ones, and they were three times as frequent. However, the experience greatly differed between those who achieved a good or moderate clinical success and those who experienced no clinical effect on their main complaints. The latter group tended to stop treatment early and not to complete the checklist. Nevertheless, 14 checklists were available. This group listed as many unpleasant experiences as pleasant ones. By contrast, patients with good clinical success reported the same number of positive experiences as the students and just as few unpleasant experiences.

It is important, therefore, in evaluating the experiences with breathing instructions to include unpleasant experiences and name them specifically. This needs to be done in addition to evaluating clinical effect on the main complaints. Moreover, evaluation needs to be done individually. The group that shows no benefit is probably rather small, and their presence will be masked when only average outcome scores are reported. The presence of unpleasant awareness has been a major reason to use instructions with an indirect approach to breathing (van Dixhoorn, 2014)—for instance, asking the client to pull up his or her feet will indirectly influence breathing. The procedure is to ask the client "How does it feel?" without indication of what you expect. After some repetition, most clients have found that this tends to block an easy and full abdominal inhalation. Then one can proceed with the next step.

Relaxation Overdose

Like any relaxation instruction, breathing therapy may lead to a decrease in sympathetic tone. The normal relaxed state consists of a decrease in activity level and lessened sympathetic activity. However, the resulting effect may, on occasion, turn out to be too big. This may happen when getting up from a reclining position or when practicing in the upright position, sitting or standing. The person may feel dizzy or lightheaded or may want to lie or sit down again. When blood pressure drops too much, fainting may occur, and the person's face becomes pale. It is important, therefore, always to get up slowly and not to practice too long in the standing position at first. Usually, faintness is temporary and passes in a few minutes, but sometimes it takes a while for the person to recover. It may happen more frequently after an illness or when one is tired. Usually the individual does not feel bad, only tired, or sometimes even refreshed. Faintness or fainting is promoted by concentrating too long or too intensely on the supporting surface when sitting or standing upright, without moving the body.

Relaxation-Induced Anxiety

The fear of losing control may result in a higher anxiety state after relaxation instruction than before. This is most frequently described when the instructions are suggestive, as in autogenic training (Heide & Borkovec, 1983). It rarely seems to occur during instructions that focus on movement and muscle tension. This is a reason primarily to choose movement instructions to influence breathing and not to have the patient sit or lie still for too long a time. When signs of unrest appear, one option is to modify the instruction and introduce some kind of movement. When that rule is observed, panic or anxiety attacks rarely occur during breathing therapy. However, during any kind of relaxation instruction, panic may be caused by unintentional hyperventilation (Ley, 1988).

Cathartic Responses

An intriguing possibility is that, during relaxation, spontaneous movements or emotions occur that are like a discharge of pent-up emotional energy. They are described in autogenic training as the result of homeostatic processes in the brain (Linden, 1993; Luthe, 1965; see Linden, Chapter 18, this volume). Someone may start shivering or yawning, legs or arms may move, or sudden movements may occur along the whole body. It is an involuntary bodily response, like sneezing or sobbing heavily. When this happens, first ask what the person notices and how it feels. Do not try to stop the reaction right away, because it may feel natural or good. When that is the case, the impulses may simply run their course and leave the person refreshed and relieved afterward. If they are too disturbing for the individual or in a group session, ask the person to start moving voluntarily or to inhale deeply and hold the breath for a few seconds. This usually stops it.

Sometimes the responses are emotionally charged and include experiencing past traumatic events, with associated sadness, shock, or anger. This is not a bad thing, either, but the situation should be appropriate for it. If that is the case, it may feel beneficial.

Another form of spontaneous self-regulation is a positive side effect on memory. During the passive attentional state ideas, images, memories, and mental pictures may arise, which may have specific meaning for a problem at hand. An unexpected solution or a new perspective on a problem may be helpful. Somehow, hidden resources may come to the front, a development that can only be welcomed.

Cardiac Arrhythmia

In over 30 years of working with breathing therapy with cardiac patients, it has struck me that cardiac arrhythmias sometimes are accompanied by specific breathing patterns that may provoke them. Arrhythmias often increase under the influence of sympathetic activity, and relaxation may be helpful (Benson, Alexander, & Feldman, 1975). Similarly, breathing slowly and more abdominally may be helpful. However, this may also be counterproductive. It seems that sometimes breathing is too slow and exhalation pauses too long in comparison with the state of agitation of the whole system. Also, exhalation constricts the intrathoracic space and may stimulate the heart mechanically. The heart responds with extra beats, mostly supraventricular, but also premature ventricular contractions may occur.

The association with breathing is confirmed when the frequency of abnormal beats decreases or disappears on altering breathing and a more shallow and functional upper thoracic breathing pattern is elicited. Inhaling to the upper chest and not exhaling too strongly or for too long helps to create more space in the chest, and this respiratory pattern matches the state of the organism as a whole. Similarly, sitting more upright and elevating the chest may be helpful in that situation. When the heart has calmed down, respiration may be changed to a slower and more abdominal pattern that does not evoke arrhythmia.

TREATMENT MANUAL

The method utilizes a relatively large number of instructions, as well as manual techniques, 50 of which are described in detail in a Dutch handbook (Van Dixhoorn, 1998b). A few instructions are available in English through the Internet (*www.methodevandixhoorn.com*). In this chapter, seven techniques are described for use with the client mainly in the sitting position. A chair with a flat, horizontal surface is required, preferably a stool without back support. The techniques are described in a sequence that may actually take place during several sessions, although you should evaluate each step and may decide to change the sequence. First, decide whether you want to start with manual assessment of breathing pattern or with an instruction. For manual assessment, you take a position behind the client and place both hands on his or her lower back. If either you or the client does not feel comfortable doing this, it can be skipped or postponed. Always obtain permission from the client to touch the body before starting this procedure. Patient instructions for whole body breathing are summarized in Table 12.3.

Assessment of Breathing

Distribution of Breathing Movement

Have the client sit comfortably on a stool and sit behind it on a lower stool. Pass your hand along the spinal column, feel the curvature of the back and whether posture is erect or slumped. Put the palms of both hands alongside the lumbar spinal column, with the thumbs in a vertical direction, parallel to each other, with the top at about the level of the lower thoracic vertebra and the fingers spread out. The second and third fingers should touch the lower ribs, and the fourth and fifth fingers should touch the area below the ribs. The full surface of the hands should touch the body. Do not squeeze the ribs together but lightly touch the middle of the body. When the client is sitting erect, this area is tight.

TABLE 12.3. Patient Instructions for Whole-Body Breathing

Lie on the back with your arms at ample distance next to you.	Take care that there is sufficient space between the arms and the ribcage.
Bend the arms in order that you can bring the hands on the chest.	Notice where the hands come to lie on top of the chest.
Let the hands lie loosely, do not press hard.	The more restless (dyspneic, anxious) you are, the higher the hands lie down.
Do you feel the breathing movement under your hands?	The chest rises and sinks.
Pay attention to the movement for some time and accept how it moves.	Do not try to correct it; do not try to have a regular and even movement.
It is what it is.	If you feel stressed or anxious, it is only natural that your breathing movement is not even.
Stop doing this.	Feel how you are lying; is your head comfortable?
Now pay attention to the air flow, in and out.	Follow the flow of air attentively, notice if you inhale through the nose or through the mouth.
Maintain your attention to the flow of air.	Listen if you can hear the flow.
Stop doing this.	Feel how you are lying; is your head comfortable?
Feel again the chest movement under your hands, and at the same time the flow of air.	Do you feel that they are simultaneous?
When the air goes in your chest rises and when the air goes out the chest sinks again.	Pay special attention to the timing.
Maintain this focus for some time.	How is the flow of air now? Through the nose or the mouth?
Stop doing this.	Feel how you are lying; is your head comfortable?
Let the hands lie loosely on the ribs.	Your hands move downward.
Let the elbows rest a bit more away from the ribs and the arms bend more.	Shift the hands downward until they lie against the sides of the chest.
Do you feel the breathing movement under your hands?	Take your time for this, and let the hands lie loosely, do not press them.
Follow the movement for some time.	Note the movement on top of the chest; it is rising and sinking.
At the same time you can also feel that the ribs move sideways, becoming broader and narrower.	Take your time and feel the sideways movement.
Maintain this focus for some time.	How is the flow of air now? Through the nose or the mouth?
Feel how you are lying, is your head comfortable?	
Again pay attention to the movement under your hands.	How do your hands feel to you now?
Do you notice both movements? The ribs elevate, they rise and sink again.	At the same time you can feel the sideways movement, the ribs become broader and narrower.
Take your time to really perceive both movements.	
When you breathe out the chest becomes more narrow.	Emphasize this a little by pressing lightly with the hands to the sides of the chest.

(continued)

TABLE 12.3. *(continued)*

Stop pressing when the exhalation is finished.	Let go of the pressure completely and feel that the chest widens with the inhalation.
Do this 45 times and stop.	
Feel how you are lying now and how you are breathing, take time for this.	
When you are sufficiently rested, repeat the previous step.	Press softly to the chest with exhalation and let it completely go with inhalation.
Repeat the whole sequence once more and then take a rest.	Feel how you are lying now and how your body is.
What do you notice in your body?	(You can stop here.)
Option 1. (for those who have become interested in what there is to feel in the body). Pay close attention to the breathing movement.	Feel the chest and ribcage moving with breathing.
Now watch carefully: which movement comes first and with which movement it starts.	Is it the lifting of the ribs or the broadening of the chest, that initiates the breathing?
Try both possibilities.	
Let the chest first rise and then the lower ribs widen, a few times.	Feel how this is and how the breathing then is.
Then let the widening of the ribs be the first and then the rise of the chest, a few times.	Feel how this is and how the breathing then is.
What is the difference between the two?	
Then compare the two again and start with the most pleasant sequence.	
Compare again.	Try to feel which is the easiest and feels most natural.
Stop doing this and feel how you are.	
Option 2. (for those who became tired of the concentrated focus on the ribcage).	Imagine that you are inhaling towards the lower legs and feet.
Feel the legs lying down, they are stretched out, and notice the calf's relaxing.	
Pull the feet towards you and relax them.	Where do you feel the tension, increasing when you pull and decreasing when you relax.
Continue pulling up and relaxing the feet.	
Stop doing this and feel how you are.	What do you notice?
Do it again, for some time.	
Stop doing it.	Feel how you are and how the legs are lying.

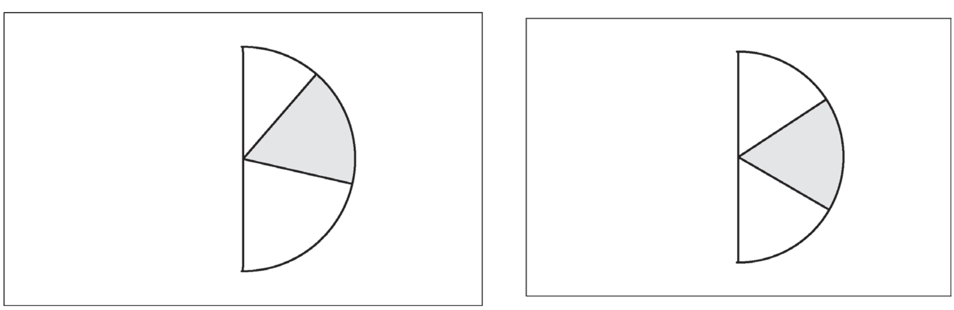
Note. Instructions are meant to be read to a person who is lying, more or less comfortably, on his or her back, with hands on ribs (costoabdominal breathing). The instructor sits next to the person, at a distance, and reads the text out loud in a normal conversation tone. Meanwhile, the instructor observes the person carefully in order to see how the words affect him or her. The person should be able to do as he or she is told, without having to think about what exactly is meant. If the instructor sees confusion or a negative feeling in the person, the instructor should stop and ask what is the matter, and take care not to correct the subject. There is no right or wrong; it is all a subjective experience. The questions are not meant to be answered; it is what you can ask yourself. It can be discussed afterward.

In that case, ask the client to sit "at ease"; support the body with your hands. When the client is slumping too much, this part is round. In that case, ask the client to sit up a bit more and push the body a bit forward. Ask the client to move forward and backward a few times and find the middle position, sitting fully on the sitting bones. A complete description can be found in Courtney and Van Dixhoorn (2014).

You might notice the breathing movement under your hands. Do not try to change, guide, improve, or amplify it. Simply let your hands notice the direction of movement at inhalation and mentally try to form a picture of the area in which respiratory movement is present. The fingers on the ribs might notice a sideways movement, a lateral expansion. This is the result of both the elevation of the rib cage and the diaphragmatic pull on the ribs (Kapandji, 1974). Try to feel to what degree there is upward movement and to imagine how much the upward movement extends to the top of the rib cage. When the elevation of the rib cage dominates, there is largely an upward movement and little lateral expansion. When the diaphragmatic down-pull dominates, there is a large sideways movement that spreads upward only a little and is particularly present at the fourth and fifth fingers below the ribs and in the palm of the hands. Try to form an image of the degree to which there is an outside push. This originates from the diaphragm, which descends with inhalation and pushes the abdominal content outward. Then release your touch, get up, and graphically describe the area by drawing two lines to form a slice of a pie chart (see Figures 12.4 and 12.5). The center of the pie corresponds with the thoraco–lumbar junction. The size of the slice, the distance between the upper and lower lines, corresponds with the *area* in which inhalation movement is present, in your estimation. The place of the slice in the pie corresponds with the location of breathing: upper half corresponds to chest, lower half to abdomen. They represent *distribution of breathing movement*. Although this assessment is based on subjective estimates, this seems to adequately represent reality. Interrater reliability and correspondence to somatic measurements of the procedure have shown satisfactory values (Courtney et al., 2008). See the report by Jansen and colleagues (2015) for correlations between MARM, NQ and CO_2.

Further Observations

During this assessment, you have an opportunity to observe other qualities, which might be noted on an observation sheet (Figure 12.6). You get an impression of respiratory speed, and you might count the exact frequency per minute. Although it might be expected that a small area of distribution coincides with a small and rapid respiration,



FIGURES 12.4. AND 12.5. Graphic description of distribution of breathing movement. Figure 12.4: sitting upright; Figure 12.5: sitting easy. Data from 6 participants.

Pace and respiratory frequency:	Rapid, slow, cycles per minute
Pauses:	Natural, marked, long, inhalatory or exhalatory, or absent
Irregularity:	Normal, absent, marked, frequent
Response to breathing deeply:	Normal return, slow return, area, location
Smoothness:	Normal, absent
Symmetry:	Present, absent, degree and location of curve
Scoliosis (lateral curves):	Absent, present, degree and location of curve
Spinal (sagittal) curves:	Normal, marked, diminished, lordosis/kyphosis
Sounds:	Absent, marked, origin, inhalatory or exhalatory
Awareness of "width" breathing:	Normal, pleasant or absent, disturbs breathing

FIGURE 12.6. Breathing observation sheet for sitting.

the correlation between size of the area and respiration rate is relatively low. Next, you might notice other time-domain qualities, including the transitions between inhaling and exhaling and irregularities. Transitions may be clearly present as a momentary "pause" at the end of a movement, which is a natural phenomenon and represents proper coordination of the breathing movement. They can be marked or take longer time, in particular after exhalation, and can be noted as such (e.g., marked, long). They might also be clearly absent, in which case breathing is hurried, coordination is not smooth, and the process of reversing breathing from inhalation to exhalation movement and vice versa is abrupt and choppy.

Irregularity of rhythm is to some extent a natural phenomenon, reflecting a relaxed state of the individual and being prepared for environmental changes. It indicates that breathing is responsive, and it should occur when the person is asked to move, for instance, turning the head left or right or shifting the body. Marked irregularities, however, consist mostly of deep sighs, which are a sign of dyspnea, shortage of breath, or anxiety. They should be noted, particularly when they are frequent.

Tidal volume is impossible to estimate manually. Size of trunk expansion cannot be taken as a proxy measure of tidal volume. It also correlates poorly with measurements of circumference changes of the abdominal and thoracic compartments, which do provide a good estimate of tidal volume once they are properly calibrated. Thus it is quite possible to breathe a large volume with little sideways expansion or to have a wide distribution along a large area with relatively small volume. A wide distribution reflects the involvement of the ribs, their ease of movement, and coordination with the diaphragm. However, within one individual, changes in area probably correlate highly with changes in volume. Having someone take a deep breath results in a marked increase in expansion of the trunk. From this, the tendency to hyperventilate can be estimated from the time it takes to return to normal breathing after a deep breath. Also, the degree that breathing shifts upward can be assessed and noted graphically.

An important aspect of functionality is smoothness of movement and ease of breathing. Manual assessment is the best way to judge this, because the hands are sensitive to the amount of effort or strength employed by the intercostal muscles to breathe. Also, notice the occurrence of extra "pushes" during inhalation or exhalation, which diminish smoothness and indicate some form of voluntary or habitual controlled breathing pattern. Manual assessment is the best way to find asymmetry between the left and right sides of the chest. In addition to visual inspection, the hands might find that one side is moving more than the other. This can be represented graphically by drawing both sides in the pie chart. Asymmetry might be a cause of dyspnea or breathing difficulty of which the person is often unaware. It may coincide with scoliosis of the spinal column, which is also assessed at the beginning, but which requires more attention when the rib cage shows asymmetry. Asymmetry of the thorax can lead to thoracic strain and eventually to backache. The degree of the convexity should be described, its direction (left/right), and its location along the spine. In general, the shape of the spinal column deserves attention. Pronounced kyphosis or pronounced lordosis of the lumbar or cervical parts is important as a marker of thoracic strain, as well as a marked absence of the natural curves, for instance, in the lumbar or upper thoracic area.

Finally, the observer may pay attention to the sounds of air passage. During quiet breathing, clearly audible sounds reflect turbulence in the airways, which deserves attention because they reflect marked effort in passing the air and great pressure gradients. Sounds may occur during inhalation or exhalation and may originate from the chest, throat, mouth, or nose. Individuals who habitually breathe through their mouths should be asked to close them and try breathing through their noses. Sounds that originate from the chest usually reflect lung problems.

Observation of Breathing Awareness

An essential quality of functional breathing is that it is open and accessible to conscious attention. Although awareness always influences function somewhat, it should not disturb breathing nor lead to marked changes or to a particular voluntary breathing pattern. This happens frequently when attention is drawn to breathing in the chest or abdomen. Because few people are conscious of the back or of breathing into the back, this procedure is well suited to assess openness of awareness of breathing.

Ask the client to pay attention to your hands on the back and ask how it feels. Then ask whether the client is aware of any respiratory movement in the back at the location of your hands. If the client notices respiratory movement and breathing does not change much, then ask in what direction movement is felt during inhalation: upward, forward, backward, or sideways. Next, ask the client to focus on the movement sideways and to describe it: The ribs or back broaden during inhalation and become smaller during exhalation. Then ask how it feels to mentally follow this breathing pattern. The normal response is a feeling of pleasant, natural breathing, which does not require much effort or control. However, some people do not notice any movement in the back, although it is obvious to the observer; or the movement may be absent or may disappear when the person pays attention, to be replaced by inhalations upward and extending the back to lift the chest. Others are unable to notice respiration passively and might try to actively control it; this attempt is often accompanied by a decrease in smoothness and an increase in effort. These responses constitute disturbances in breathing awareness.

Instructions

Seven instructions are described. The texts of the instructions themselves are numbered and can be read verbatim to the patient. (In these enumerations, *you* refers to the patient, whereas in the rest of the text, *you* refers to the reader). Each row consists of several sentences. Do not read all the sentences one after another, but pace the reading to the client. Pause and observe after each sentence and determine whether continuing is appropriate.

Each instruction consists of several steps in which the instruction evolves. You must observe the response of the patient before deciding to proceed. You could also stay at a step and repeat it. *Note: Please do exactly as is written.*

A good way to start is in the sitting position, because it easily transfers to daily life. After each sitting instruction, it is a good idea for the client to stand up in order to notice any changes in habitual posture. It is also a good idea for the client to lie down for a few minutes, when the occasion allows it, in order to let the spine relax. Instructions in the supine position are indicated when the client seems a little tired or in need of a rest, or when an emphasis on passive relaxation is needed.

The first instruction in the sitting position can be "circle knees" or "forward, backward." "Circle knees" takes more time and requires an undisturbed situation for practice. "Forward, backward" can be done in between activities and is less conspicuous. "Sitting, standing" teaches a specific way of standing up, which the client can apply each time he or she gets up without doing the exercise itself. "Shoulders up" improves coordination of the breathing movement of the chest. "Exhale audibly" is a direct instruction to change breathing and can be done in any posture. In the supine position, "pulling up the feet" is a good way to start, because it is an indirect way to influence breathing, easy to do, and gradually involves breathing. "Looking up and down" is more difficult. When the client has trouble doing it, it is better to stop the exercise. Generally, it is recommended to first do the easy instructions, which prepare the way for direct breathing regulation. Then proceed with the more difficult instructions.

Start with . . .	Circle knees . . .	Forward, backward . . .	Pulling up the feet
Next . . .	Audible exhaling		
End . . .	Shoulders up . . .	Sitting, standing . . .	Looking up and down???

Sit, Circle Knees

Position yourself on a chair at right angles to the client, so that you see each other obliquely and do the instruction yourself while giving it in order to model it.

1. Place the feet a little beyond the knees. Put the hands on the knees, palm downward, and notice how you sit.
2. Move the hands around the knees, circling the knees, downward at the outside, upward at the inside, about twenty or twenty-five times. Do it unhurriedly, as easily as you can, somewhat carelessly, without counting.

   Notice that the body moves forward when the hands go down and backward when the hands go upward. You can feel this movement in the sitting bones.

   Stop the movement, look straight ahead, and notice how the body feels.
3. Repeat the movement, about twenty to twenty-five times. This time pay attention to the shifting of weight between the sitting bones and feet. When you move back, the weight on the sitting bones increases until you sit fully on top of them. When you move forward, there is more weight or pressure on the feet.

   Stop the exercise, look straight ahead, and notice how the body feels.
4. Do the same movement for the third time, again twenty to twenty-five times. Is there a difference in the way your body moves? This time notice that, each time when you come back, with the weight fully on the sitting bones, you look straight ahead.

   Stop the exercise, look straight ahead, and notice how the body feels.

   Stand up. Notice what you feel.

Check whether the body really moves back and forth and that the client does not limit the movement to the arms and hands circling the knees. The continuous shifting or rolling of weight on the sitting bones tends to draw attention in a passive way. Afterward, most clients are more aware of the fact that they are sitting; they feel their weight more clearly when asked. This attentional shift tends to facilitate a less erect sitting posture, a sense of relaxation, both physical and mental, and thus a quieter respiration. Also, the movement tends to become slower during subsequent steps. The awareness of the supporting surface, including the feet, is emphasized in steps 3 and 4. When leaning more forward, one notices the weight on the feet more. This helps to enlarge the movement in a natural way, which also occurs as a result of repetition and increasing ease of movement.

You might manually assess breathing movement after each step or at the end, or simply ask or observe. An optimal response is that breathing becomes easy and almost automatic, undisturbed by one's perception. Emphasis on the position of the head, in step 4, facilitates involvement of the spine: straightening a little when going forward, flexing when going backward. Observe to what degree this actually occurs. When it does, the sensation of standing on both feet at the end is much clearer and the body is more erect, but without effort.

Sitting Forward, Backward

You can join this instruction yourself, but you might also visually observe the movement from the side or from the back, with both hands on the body of the client, to check whether the spine really changes shape and to encourage it, when necessary.

1. Lean the body forward and backward a few times. Then go backward, stay there, and notice the back. Go forward, stay there, and feel the back. Repeat this a few times and notice the change in the form of the back: becoming a bit rounder when leaning backward and straightening up when leaning forward. Do not tilt the pelvis intentionally.

   Stop the movement, sit in the middle, and notice how you are sitting.
2. Move back and forth slowly and pay attention to the change in the shape of the back. Go backward, stay there, breathe in and out a few times, and notice the place of breathing movement. Go forward, stay there, breathe, and notice the place of breathing. Repeat this until you notice that respiration responds to the position. Be aware of this response and follow the way breathing changes with posture.

   Stop the movement, sit in the middle, notice how you sit, and breathe.
3. Move back and forth, and do it in a rhythm similar to your breathing. Continue and notice whether a connection appears. Are you breathing in when moving forward or when moving backward? Continue this awhile until the connection becomes easy.

   Then try and change the connection. If you inhaled when moving forward, now move forward during exhalation, and vice versa. Continue this and notice how it feels. Both options are equally possible.

   Stop the movement, sit in the middle, notice how you sit, and breathe.

   Stand up and notice what you feel.

This instruction is essential for awareness of the relationship between posture and breathing. When the spinal column really changes in shape, breathing responds. When the spine is extended, respiration follows the pattern of "length" breathing. When the spine is flexed, respiration follows the pattern of "width" breathing. So, moving forward and backward can both be connected to inhalation. When both patterns are present, flexibility of both posture and breathing is increased. It demonstrates that there might be more than one pattern of "good" breathing. This might require explanation and discussion in order to restructure the client's cognitions.

Sitting, Standing

This instruction facilitates standing up from a chair with less effort by using the strength of the feet and legs economically. You may join the instruction yourself or you may observe from the side.

1. While sitting in a chair, place the feet a little behind the knees, flat on the floor. Move the body slowly forward and backward and notice the pressure changes in the feet. . . . Continue, and, when going forward, press the feet more toward the floor a few times. . . . Continue, and, when going forward, look straight ahead. Notice the effect on your back. . . . Stop and feel how you are sitting.
2. Lean backward, inhale, move forward and exhale, again backward and inhale. Repeat this a few times until it is easy. . . . Then go forward, press the feet, exhale, look ahead, and go further until the buttocks lift off the chair a little. Return, lean backward, and inhale. Do this a few times. . . . Stop and feel how you are sitting.
3. Finally, move forward, exhale, and stand up, remain standing, and inhale, exhale, and sit down. Do this a few times, then remain standing and notice how you stand. Sit down and notice how you sit.

When a person leans forward, presses the feet on the floor, and looks ahead, the spine tends to extend. Continue with the instruction only when spinal extension occurs in the first step. Observe that the hands are lying loosely on the upper legs and slide forward and backward with the body. They should not press on the legs or be held stiff.

In the second step, respiration is coupled to the movement in a way that is contrary to what is done habitually by most people. Inhaling when leaning backward promotes "width" breathing and sideways expansion of the ribs. As a result, the lower ribs in the back are in an optimal position to contract with exhalation while moving forward, and this facilitates extension of the spine. Moreover, exhaling when starting to lift off the chair helps to contract the pelvic floor muscles and to withstand the rise in abdominal pressure. This prevents urine leakage, particularly for people who have urinary incontinence problems. Also, it prevents the tendency to "brace" when making effort, which is coupled to inhalation and excessive muscular effort. The hands might move forward and extend beyond the knees to help the shifting of weight. At the end of step 2, the client may sit differently, often more active and erect but without much effort.

In step 3, the client stands up and sits down again, both with exhalation. When successful, standing up requires less effort, although the procedure may still feel somewhat strange. It is important to have the client remain standing for a while and to observe any differences from habitual posture. Usually, the person stands more firmly on both feet and is aware of the weight in the feet. The client is encouraged to notice this many times during the day.

Exhale Audibly

This instruction can be done in any position, preferably when respiration is not disturbed by conscious awareness. Therefore, preparatory instructions are appropriate. In the supine position, the client may be asked to let a hand rest on the abdomen and notice the respiratory movement. In the sitting position, it is sufficient to ask the client to notice respiration, or he or she might be asked to observe the sides of the body.

1. Take some time to notice your breathing, pay attention to the breathing movement without changing it. Do not deliberately breathe more deeply or more abdominally. Form a mental picture of the breathing curve, with inhalation going up and exhalation going down. Notice the steepness of the rise and fall and the pauses in between.

   Inhale slowly through the nose and exhale through slightly pursed lips, making the sound of "fff" at the lips. Inhale again, slowly, through the nose and exhale audibly, softly. Do it five to six times, then stop and take time to notice your breathing until it has resumed its natural rhythm.

   Compare the breathing pattern with the beginning of this step: depth, speed, volume, location, pauses.
2. Repeat this two to three times.

   What changes in respiration do you notice? Is there any other change that you observe? In mental state, in the head, or in the way the body feels, sits, lies, stands?

The instruction results in a temporarily larger tidal volume that involves more parts of the respiratory apparatus, particularly the ribs. It is essential that respiration resume its natural rhythm after exhaling audibly and that deeper breathing stop. Some clients have trouble in stopping the controlled breathing, and they continue breathing steadily deeper, often at a fixed pace. This may result in hyperventilation. It is important to have the client understand the necessity of stopping the direct control of breathing and then to observe any changes in spontaneous breathing. These are usually rather small changes in breathing pattern: slightly longer and/or deeper but, more important, easier and less hurried, with smoother transitions from inhaling to exhaling and from exhaling to inhaling, and with an increased sense of "space" for breathing in the body. Breathing involves more parts of the body, is more evenly distributed, and can be perceived all over the trunk. When asked, clients might notice that their mental state is changed, as if the head were clearer, with fewer thoughts. Also, because the instruction involves modest training of the respiratory muscles, the coordination of respiratory muscles may have improved, as well as their role in posture. This results in a more balanced and stable erect posture or in sitting more firmly (Czapszys et al., 2000)

The instruction differs from pursed-lips breathing, which is a familiar technique for emphysema patients, in some respects: Inhalation is slower, and exhalation is not as strong, but with a moderate, even sound. Although this maneuver might reduce dyspnea, it is not meant to be done continuously, but only a few times, then stopped, and the changes observed.

Sit, Shoulders Up

This instruction helps to improve movement of the ribs and chest with breathing and is feasible when respiration is not disturbed by conscious awareness. It is helpful to assist by having manual contact with the lower ribs in the back during instruction, but the therapist might also do the movement while teaching it. This exercise can be done standing as well.

1. Notice the breathing movement in the chest. The lower ribs expand sideways when you inhale, and the chest bone lifts a little upward. Continue until breathing is clearly perceptible, but no longer disturbed by your attention. Stop if breathing becomes disturbed. Raise the shoulders toward the ears, then keep them there and continue breathing. Notice the ribs and chest. Let the shoulders sink down. Compare the breathing with the beginning of this step. . . . Repeat this until the breathing movement in ribs and chest is hardly influenced anymore by raising the shoulders. . . . Stop the movement, feel how you are sitting (standing), how your body is breathing, and how you are feeling.
2. When you succeed easily with the first step, raise the shoulders about halfway to the ears and keep them there. Then try raising the shoulders a little during exhalation and let them sink down a little during inhalation. Do this several times, then let the shoulders sink fully during inhalation and leave them there. . . . Stop the movement, feel how you are sitting (standing), how your body is breathing, and how you are feeling. Repeat this twice.

Although the bony connection between shoulders and rib cage is limited to the clavicle, the muscular interconnection is so tight and the habitual association so strong that raising the shoulders is usually closely coupled to inhalation and elevation of the ribs. This instruction helps to disengage the tight association, thereby improving mobility of both the shoulders and the rib cage.

First, respiratory movement in the chest must be felt and allowed to happen. Sometimes the mistaken idea that respiration should happen only in the abdomen needs to be discussed. When the movement of the ribs, particularly the lateral expansion of the lower ribs and the elevation of the chest bone, is perceived clearly, then the instruction is to raise the shoulders to the ears and to continue this movement as much as possible. Say "Let the position of the shoulders not bother you or interfere with breathing, just continue breathing in and out." This requires some repetition. Each time ask whether any differences occurred after the instruction, in comparison with before the instruction. Although clients differ in the degree to which they tolerate new or strange actions, most feel afterward that breathing is easier and better distributed and includes the chest but is equally present in the abdomen. The body feels more relaxed and more open to breathing. It is important to explain that improved mobility of the ribs facilitates diaphragmatic action and improves its downward motion. Also, when the chest bone quietly elevates, this helps diaphragmatic descent and thus promotes lower abdominal breathing. Breathing is a natural "whole body" movement, and this instruction can make the point clear. It may give a sense of relief, both somatic, with respect to breathing sensation in the chest and relief of dyspnea, and cognitive, with respect to the mental picture that breathing can simply be allowed to happen along the whole of the trunk.

When the first step has been successful, the second step increases the difficulty. Its aim is to reverse the association between raising the shoulders and inhalation by asking the client to do the opposite: Raise the shoulders a little during exhalation. This creates two opposing movements. The shoulders go up, the ribs go down. As a result, when during inhalation the shoulders sink a little, there is a very clear sensation of lateral expansion and elevation of the ribs. This creates an unusually large space for inhalation. Also, the shoulders relax even more. As a side effect, this instruction inhibits the habit of bracing or forceful, upper thoracic inhalation during stress.

Supine Position

For instruction in the supine position, a flat, horizontal surface is required, such as a treatment couch or a mat on the floor. A reclining chair or "relaxation" chair will not work, particularly for "looking up and down."

Be sure that the client's head is supported well. Instruction may include having the client try several layers of support under the head, for instance, in the form of a large towel that you could fold several times to increase its thickness. Offer various layers of support and ask the client how each feels. When the client reports feeling all right, offer even more variations, and observe the position of the head until it is clear that one level of support is optimal. Usually the face is horizontal and parallel to the surface. It may not be the habitual position of the head, but it actually feels comfortable. It is worthwhile to take some time with this procedure, because it contains several messages. First, the client may answer "I am okay" when asked how he or she feels, but this may be a social answer, and it does not mean that the position of the head is optimal. However, the awareness of differences in tension and the sensation of optimal comfort is essential for success with these breathing instructions. Second, the care taken to find an optimal position for the client demonstrates your intention that the client's comfort is the prime concern. You should not challenge or put the client to a test to find out about his or her level of tension. Finally, the procedure clarifies that support for the head should be just that: an elevated surface for the head to rest on, while maintaining its mobility. It is not a cushion stuffed behind the neck that blocks motion.

Pulling Up the Feet

This instruction is very easy and can be done by almost everybody, particularly the first part. It is a good way to start in the supine position. It attracts attention to the feet, away from the location of most tension problems.

1. Pull your toes toward you while the legs remain straight and notice where the tension increases in the legs. Stop the exercise and take time to notice the tension slowly disappearing. Repeat this two or three times.

   Then stop the movement and notice how your legs and body are lying.
2. Do it again, and this time pay attention to the difference in tension in the legs and to the difference in breathing with the legs relaxed and tense. When is inhaling easier? Then stop and notice how the legs and body are lying.
3. Do it a third time, now coupling it to breathing. When you exhale, and while exhaling, slowly and gradually pull up the feet. When exhalation is complete, release the feet and relax the legs completely. Then inhale. Continue this until it feels natural and easy.

   Then stop and notice how your legs and body are lying. How do you feel?
4. Get up slowly. First, sit up and notice how you feel. Take your time. Then get to your feet, stand, and notice how you stand.

This instruction starts as a tense–release instruction, as in Jacobson's method, but it does not focus on the source of tension, the tibialis muscle. Instead, all signals are accepted—the stretch sensation in the calf, the joint movement in the ankle, the tensing of the quadriceps muscle. During instruction you may discuss the client's observations. With repetition, tension signals are felt in the whole of the leg and even in the lower half of the body. That is, the abdomen tightens a little, making inhalation more difficult. Inhalation becomes easier when the legs are relaxed. This is the experience to emphasize in the second step. Some clients fail to notice this, and any attention to breathing disturbs it. In that case, continue with tense–release cycles until the legs are lying more relaxed, and stop there. For some clients, the experience of easy inhalation when the legs are relaxed is sufficiently new and impressive. In that case, stop there and ask the client to repeat the exercise at home.

When the second step is clear and easy, the third step of the instruction introduces an unusual coupling of tension increase with exhalation. Our bodies are programmed for the association of inhalation with tension increase. This is easy to do and is utilized in progressive relaxation (see McGuigan & Lehrer, Chapter 7, this volume). In this instruction, the reverse is done. This requires conscious attention and therefore stimulates distraction of attention away from the tension; that is, when the tension problem is not in the legs. Coupling breathing to a small movement also tends to slow down respiration. Also, making an effort during exhalation implies that inhalation becomes associated with less or no effort. This favors natural breathing, that is, letting the body inhale and not adding extra effort. Pulling up the feet is mechanically coupled to exhalation, as the abdomen tightens and the pelvis tilts backward. Thus relaxing the legs helps to relax the abdomen, which allows the diaphragm to descend. Also, the pelvic tilt pushes the spinal column upward, which facilitates exhalation according to the pattern of "length" breathing: The ribs flatten, the head tilts upward, and the chest bone descends. This reverse coupling may take a while to get used to, but once the client gets the knack of it, it feels very easy. Altogether it makes the instruction a good way to introduce very slow breathing, as in resonant breathing. Afterward, most clients stand surprisingly stable and with full awareness on both feet.

Looking Up and Down with Breathing

This instruction is somewhat difficult, because it influences rather directly the position of the head, as well as respiration. Clients with severe tension problems in the head, neck, or jaw or clients with severe dyspnea may benefit from it, but it may also be unsuccessful and even cause problems. It is important, therefore, to observe carefully and to inquire of the client how the response feels during each step. This exercise can be done in the supine, sitting, or standing position, but it is best to start with the client lying down.

1. Look comfortably straight ahead. Focus on a point in front of you. Then slowly look up, stay there, breathe in and out a few times, and look back again. Repeat this a few times and notice that there is a tendency for the head to tilt a little upward when you look up. Allow this to happen. Also, the jaw may tend to open a little. Allow this.

   Stop the movement and notice what you feel.
2. Feel your breathing going in and out a few times, and then, while exhaling, look slowly up. When inhaling, look ahead again. Do it in such a way that the exhalation and the shift of your gaze coincide. As long as you exhale, continue looking upward. Repeat a few times. Stop, and notice how you are breathing. Do it again, until the coupling feels easy and the result feels comfortable.

The instruction directly appeals to the pattern of length-breathing in the upper body. Exhalation implies a slight sinking of the chest bone. This downward motion of the upper ribs in the front results in and is facilitated by a small bending forward movement of the upper back. When this is accompanied by a small increase of cervical lordosis, the position of the head remains stable, and breathing movement is functional. Thus breathing in and out more deeply is accompanied by and facilitated by a small motion at the cervico–thoracic junction: going forward with exhaling, and backward with inhaling. The eye movement tends to enhance and emphasize this pattern of coordination. When looking up with the eyes, the head tends to tilt backward a little, and the reverse occurs when looking down.

Another aspect of the instruction is that respiration responds synchronously to any stimulus that occurs. Thus the very fact of coupling a movement to inhaling or exhaling tends to lengthen and consequently deepen respiration. Because, in this instruction, respiration is coupled to a movement that is matched mechanically, the effect tends to be longer lasting.

CASE EXAMPLE: REPETITIVE STRAIN INJURY

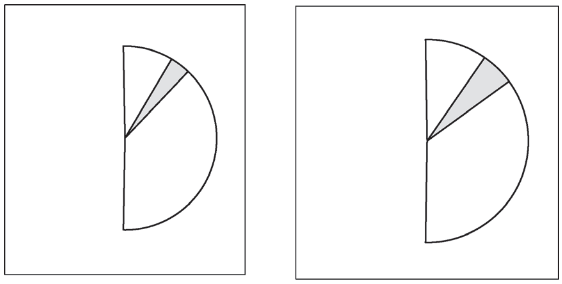
A 40-year-old woman, married, without children, had a hectic job in an international company that involved much computer work. Four years previously, she developed pains in the right shoulder, hand, and fingers. The arm felt stiff, tired, and painful. Physiotherapy and yoga had no success. After 1 year she eventually changed her job for a less demanding one with less computer work, but the pain remained, even when she took days off. After 3 years she finally found a treatment, manipulation therapy, that greatly diminished the complaints. However, the pains returned fully when she was under stress. It became clear that she had difficulty relaxing. She was referred for relaxation therapy.

First Session

The client's pain in her right arm was present daily. She had trouble relaxing. Her score on the NQ was high: 35.

Assessment of breathing in the sitting position showed a small area of movement, located high in the chest, with almost no lateral expansion (Figure 12.7). The lower back was rather tight, the right shoulder was a bit lower than the left, the head was bending forward a bit, and she was looking downward. There were no marked irregularities, no air sounds, and no asymmetry in breathing.

The instruction "circle knees" was done and went well. The client was sitting more stably, her lower back was a bit rounder, and she felt more relaxed. Respiration responded only slightly (Figure 12.8); the distribution shifted a little downward, it increased a little in area, and long postexhalation pauses appeared. She was standing more firmly on her feet and found it easier to look straight ahead. In the supine position, her legs were lying rather close together and turned inward. The instruction was given to roll the legs inward a bit more, to notice the tension and stop it. After a few times, her legs were lying more open, and this felt more comfortable for her. In the standing position, her feet were also rather close together and pointing straight ahead. The instruction to try to put a bit more distance between the feet and to point them more toward the outside resulted in a pleasant and more stable posture, with knees slightly bent. She was asked to practice all three instructions.



FIGURES 12.7. AND 12.8. Distribution of breathing at the beginning and end of the first session.

Second Session

Two weeks later, the client had practiced "sit, circle knees" regularly and felt that it was relaxing, but she could not specify concrete changes. The instruction to stand with feet further apart was very helpful; she practiced it often during the day and stood more stably. She also felt weight more in the feet, which decreased the tension in her shoulders. The instruction in the supine position did not work; she felt no effect. Generally, she had become more aware of a high level of tension. I explained that treatment is not aimed at reduction of complaints but at increasing relaxation and that focusing on tension areas usually increases tension, whereas focusing on relaxed states does not. This helped her to understand and accept the purpose of the treatment. The instruction "sit, circle knees" was repeated and went quite well. Next, the instruction "sitting, standing" was given, and she became aware that she usually used her arms too much to stand up. After she rested awhile in the supine position, her legs were lying more open and relaxed. In the standing position, she felt a variety of changes but could not specify them.

Third Session

Two weeks later, the client had continued to practice "sit, circle knees" and had become more aware of changes in the whole body afterward. She also noticed that the effects were greater when she moved more from the trunk than from the arms. The explanation that the primary aim was to increase moments of relaxation rather than fighting the tension was a very new perspective that helped her to find such moments. She had also become aware that her breathing was often short and erratic. The pain was less; pain episodes occurred now every other day instead of daily and were of shorter duration. The NQ showed a reduction to a score of 28. This time only techniques in the supine position were selected to emphasize passive rest. First, the therapist employed manual techniques (not described here) by holding both feet and influencing breathing from there. There was a good response: Respiration became deeper and felt freer. Also, long postexhalation pauses became apparent. Next, the instruction "pulling up the feet" was done, which helped to deepen breathing. Standing straight afterward, her shoulders felt more relaxed and her arms were hanging more loosely.

Fourth Session

Three weeks later, the client had practiced all instructions occasionally and had noticed that, when awareness of the lower body increased, this helped to decrease tension in the upper body. The instruction "pulling feet up" helped to relax her legs. The instruction "sitting, standing" was quite difficult. The pain had continued improving. The pain episodes occurred only 2 days per week, particularly after she worked too long or watched a thrilling movie. Her NQ score had dropped further, to 19, which is just within the normal range. The instruction "sitting, standing" was repeated, and her attention was brought to the fact that she tended to close the knees when trying to get up. The movement became much easier when she opened her knees a little in getting up. In the sitting position, breathing was assessed and used to bring her attention to the movement of lateral expansion during inhalation. She was asked to pay attention to this movement regularly. Finally, the instruction "sit, shoulders up" was done to enhance the whole-body involvement of the respiratory movement. The first step went well, but the second step did not. Raising the shoulders during exhaling was too difficult for her and too contrary to her habit.

Fifth Session

One month later the client occasionally practiced the sitting instructions "circle knees" and "sitting, standing." "Shoulders up" was too difficult, and the combination with breathing was contrary to what she was instructed to do in fitness classes. However, she had become increasingly attentive to moments of rest during the working day, and she noticed quickly when tension started to rise too high. She liked this a lot, and this awareness helped her to gain a more relaxed style of working. The sensation of "width breathing" was not very clear to her. The pain continued to improve; it occurred now once a week, was less intense, and involved fewer body parts. She had no more pain in her arms.

The instruction "sit forward, backward" was chosen in order to clarify the concept of "width breathing," to demonstrate that proper sitting can be done in more than one way and that it involves alternation of posture. The movement went well, and breathing felt quiet and easy for her, as if the body were breathing by itself. This corresponded with the assessment (Figure 12.9). Because her conscious attention did not disturb respiration, the instruction "exhaling audibly" was added. This went well, and afterward breathing became even fuller and slower. Then, standing straight, she felt very heavy in the lower body and light in the upper body. It was emphasized to her that the purpose of the exercises was to learn to enjoy and recognize moments of relaxation rather than to think "I have to relax in order to combat the tension signs."

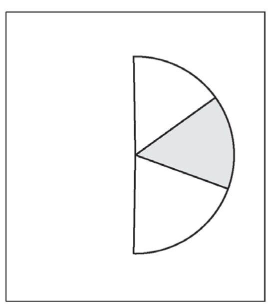


FIGURE 12.9. Distribution of breathing at the end of therapy.

Sixth Session

After 6 weeks, the client came for the final session. The pain was better; she had only one episode per 3 weeks. Her NQ score had dropped to a low level of 7. She felt the rise of tension much earlier; signs included shoulders and arms tensing up, breathing becoming shorter and less free. She frequently practiced the breathing instruction "slow inhaling, exhaling audibly," as well as the instruction "sitting, standing," and she attended to opening her knees when getting up. She also made many alterations in posture and movement during the day. When watching a thrilling movie, she was less involved and could distance herself more frequently, which resulted in smaller increases in tension.

Because her right shoulder remained a bit lower than her left, an instruction from the Feldenkrais method was done to regain better balance in the rib movement on the left and right side. We stopped the treatment with the agreement that she would phone if the complaints returned.

Discussion

This is clearly a case of high and dysfunctional muscle tension, which remained present but without awareness during rest. Thus resting was not helpful, and tension flared up immediately under challenge. This pattern is a good indication for relaxation therapy, but it does not explain the effects of treatment. One mediator may have been the client's increasing awareness of her own tension state (a discussion of the different intermediating processes is given in van Dixhoorn, 2008). This was the direct result of the success of the instructions to lower her tension, which she reported. As relaxation grew stronger, she became increasingly aware of early warning signals of rising tension. Thus tension reduction and body awareness went hand in hand. This was supported by an important cognitive change: her understanding that increasing relaxation is more helpful than fighting tension. She gradually developed a more positive perspective on her complaints. However, these discussions followed her experiences and clarified them rather than preceding them, as would be the case in cognitive therapy, in which the cognitive intervention is primary. There was little indication of a mental shift, or mental relaxation. She did not feel more mentally calm or quiet, but she had no trouble in sleeping and did not worry much. Also, restorative processes, as were indicated by feeling more energetic or "refreshed," were not clearly present. She was not tired, and thus she did not feel more refreshed or more energetic. Interestingly, the complaints in her upper body decreased to the degree that she became more aware of her legs and lower body and of receiving support for posture from the ground rather than from her shoulders and arms. Instructions for the position of her feet were particularly helpful and practical in daily life, as was the idea of slightly opening the knees rather than closing them when getting up. The instruction "sit, circle knees" was helpful from the beginning to the end. Initially, it helped to shift her focus on the sitting bones, which made her sit in a more relaxed and less effortful way. Gradually she noticed that the effect was greater and involved the whole body, as the quality of movement improved and became more functional. Thus awareness of and making a better use of the bony structure seems to have been an important mediator.

This case was chosen because it clearly illustrates the indicator role of breathing and the utility of indirect breathing instructions. The breathing pattern seemed obviously dysfunctional: small tidal volume, high frequency, and a predominance of upper thoracic breathing movement. I could have confronted this breathing pattern and tried to correct it directly. Instead, I observed it and waited for the response of the client. Distribution of breathing movement responded slightly in the first session, and long postexhalation pauses appeared, without her awareness. This indicated a functional respiratory response to a change in the tension state to which breathing should respond without drawing attention to itself. The postexhalation pauses, which also appeared in the third session, indicated a functional compensation to increased tidal volume, thus preventing hyperventilation that may occur when trying to improve breathing. Instead, the dominant subjective response was a sense of increased weight on the feet and ease in standing up. This process of functional movement guided the choice for movement instructions instead of direct breathing instructions in subsequent sessions. In the third session, the client had noted that her breathing was often short and erratic. This guided the choice for the instruction "pulling feet up," which helps to relax the legs and connects relaxation to inhaling. In the fourth session, she reported only the effect of relaxation of the legs and did not mention any effect on breathing. Thus the indirect approach to breathing was confirmed. I followed up by bringing her attention to the pattern of breathing that naturally occurs when sitting comfortably, that is, "width" breathing with sideways expansion. This did not have great effect. The instruction to combine exhaling with raising the shoulders was too difficult and too different from her habitual pattern. Only in the fifth session, when breathing had clearly responded to her increasingly relaxed state and showed a normal pattern and distribution, was a direct breathing instruction given: exhaling audibly. She was able to perform the instruction; it enhanced both breathing and the relaxed state. She felt very heavy in the lower body and very light in the upper body. In the final session, she reported having used the breathing instruction quite a lot. Thus respiration could finally play its role as an instrument for self-regulation of tension effectively.

To conclude, although no capnograph measurements were done, the client's seemingly dysfunctional breathing pattern, as well as her high score on the NQ, indicated an important role of respiration in the etiology of her complaints. Nevertheless, following the process model, instructions were chosen that matched the response of the individual. These did not include direct breathing regulation but focused on reduction and awareness of physical tension. It appeared that when the physical tension state had decreased, the breathing pattern and the complaints score on the NQ became normal. From that moment, direct breathing instructions could be used effectively for self-regulation of tension.

Four years later, the client's condition still remained stable, and she was able to self-regulate tension. She had become aware at an early stage when tension started to increase. The complaints may return a little, but when she takes appropriate action to find and change their cause, the complaints remain absent. She is satisfied and functions normally.

REFERENCES

Badawi, K., Wallace, R. K., Orme-Johnson, D., & Rouzere, A. M. (1984). Electrophysiologic characteristics of respiratory suspension periods occurring during the practice of the transcendental meditation program. *Psychosomatic Medicine, 46*(3), 267–276.

Balfoort, B., & Van Dixhoorn, J. (1979). *Ademen wij vanzelf? [Are we breathing naturally?]* Baarn, the Netherlands: Bosch en Keuning.

Barker, N., & Everard, M. L. (2015). Getting to grips with "dysfunctional breathing." *Paediatric Respiratory Reviews, 16*(1), 53–61.

Benson, H. (1993). The relaxation response. In D. Goleman & J. Gurin (Eds.), *Mind body medicine* (pp. 233–257). Yonkers, NY: Consumer Reports.

Benson, H., Alexander, S., & Feldman, C. L. (1975). Decreased premature ventricular contractions through the use of the relaxation response in patients with stable ischaemic heart disease. *Lancet, 2*(7931), 380–382.

Bergsmann, O., & Eder, M. (1977). *Thorakale Funktionsstorungen [Thoracic functional disorders]*. Heidelberg, Germany: Haug Verlag.

Boiten, F. A. (1998). The effects of emotional behaviour on components of the respiratory cycle. *Biological Psychology, 49,* 29–51.

Bondi-Zoccai, G., Mazza, M., Roever, L., van Dixhoorn, J., Frati, G., & Abbate, A. (2016). Evidence-based psychotherapy in ischaemic heart disease: Umbrella review and updated meta-analysis. In A. Roncella & C. Pristipino (Eds.), *Psychotherapy for ischemic heart disease* (pp. 131–158). Rome: Springer Nature.

Boulding, R., Stacey, R., Niven, R., & Fowler, S. J. (2016). Dysfunctional breathing: A review of the literature and proposal for classification. *European Respiratory Review, 25*(141), 287–294.

Bowler, S. D., Green, A., & Mitchell, C. A. (1998). Buteyko breathing techniques in asthma: A blinded randomised controlled trial. *Medical Journal of Australia, 169, 575–578.*

Bredin, M., Corner, J., Krishnasamy, M., Plant, H., Bailey, C., & A'Hern, R. (1999). Multicentre randomised controlled trial of nursing intervention for breathlessness in patients with lung cancer. *British Medical Journal, 318,* 901–904.

Buchholz, I. (1994). Breathing, voice and movement therapy: Applications to breathing disorders. *Biofeedback and Self-Regulation, 19*(2), 141–153.

Burrow, T. (1941). Kymograph records of neuromuscular (respiratory) patterns in relation to behavior disorders. *Psychosomatic Medicine, 3*(2), 174–186.

Cahalin, L. P., Braga, M., Matsuo, Y., & Hernandez, E. D. (2002). Efficacy of diaphragmatic breathing in persons with chronic obstructive pulmonary disease: A review of the literature. *Journal of Cardiopulmonary Rehabilitation, 22,* 7–21.

Campbell, E. J. M. (1957). *The respiratory muscles and the mechanics of breathing.* London: Lloyd-Luke.

Choliz, M. (1995). A breathing-retraining procedure in treatment of sleep-onset insomnia: Theoretical basis and experimental findings. *Perceptual and Motor Skills, 80,* 507–513.

Committee for the Rehabilitation of Cardiac Patients. (1996). *Richtlijnen Hartrevalidatie [Guidelines for cardiac rehabilitation].* Den Haag, the Netherlands: Nederlandse Hartstichting [Netherlands Heart Foundation]/ Ned Ver Cardiologie [Netherlands Association of Cardiology].

Conrad, B., Thalacker, S., & Schonle, P. W. (1983). Speech respiration as an indicator of integrative contextual processing. *Folia Phoniatrica, 35,* 220–225.

Courtney, R., Greenwood, K. M., & Cohen, M. (2011). Relationships between measures of dysfunctional breathing in a population with concerns about their breathing. *Journal of Bodywork and Movement Therapies, 15*(1), 24–34. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/21147415.*

Courtney, R., & Van Dixhoorn, J. (2014). Questionnaires and manual methods for assessing breathing dysfunction. In L. Chaitow, D. Bradley, & C. Gilbert (Eds.), *Recognizing and treating breathing disorders* (pp. 137–146). London: Churchill Livingstone.

Courtney, R., Van Dixhoorn, J., Anthonissen, E., & Greenwood, K. (2011). Medically unexplained dyspnea: Partly moderated by dysfunctional (thoracic dominant) breathing pattern. *Journal of Asthma, 48*(3), 259–265.

Courtney, R., Van Dixhoorn, J., & Cohen, M. (2008). Evaluation of breathing pattern: Comparison of a Manual Assessment of Respiratory Motion (MARM) and respiratory induction plethysmography. *Appied Psychophysiology and Biofeedback, 33*(2), 91–100. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/18320303.*

Craig, A. D. (2010). The sentient self. *Brain Structure and Function, 214*(5–6), 563–577.

Czapszys, R., McBride, N., Ozawa, H., Gibney, K. H., & Peper, E. (2000). Effect of breath patterns on balance: Breathe diaphragmatically to prevent falling. *Proceedings of the 31st annual meeting of the Association for Applied Psychophysiology and Biofeedback* (pp. 18–19). Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

de Vries, H., Kemps, H. M., van Engen-Verheul, M. M., Kraaijenhagen, R. A., & Peek, N. (2015). Cardiac rehabilitation and survival in a large representative community cohort of Dutch patients. *European Heart Journal, 36*(24), 1519–1528.

Decramer, M., & Macklem, P. T. (1985). Action of inspiratory muscles and its modification with hyperinflation. *Airways, 4,* 19–21.

DeGuire, S., Gevirtz, R., Hawkinson, D., & Dixon, K. (1996). Breathing retraining: A three-year follow-up study of treatment for hyperventilation syndrome and associated functional cardiac symptoms. *Biofeedback and Self-Regulation, 21,* 191–198.

Dekhuijzen, P. N. R. (1989). *Target-flow inspiratory muscle training and pulmonary rehabilitation in patients with chronic obstructive pulmonary disease.* Nijmegen, the Netherlands: Academisch proefschrift Nijmegen University.

Del Pozo, J. M., Gevirtz, R. N., Scher, B., & Guarneri, E. (2004). Biofeedback treatment increases heart rate variability in patients with known coronary artery disease. *American Heart Journal, 147*(3), E11. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/14999213.*

Donaldson, G. C. (1992). The chaotic behaviour of resting human respiration. *Respiratory Physiology, 88,* 313–321.

Dunn, B. R., Hartigan, J. A., & Mikulas, W. L. (1999). Concentration and mindfulness meditations: Unique forms of consciousness? *Applied Psychophysiology and Biofeedback, 24,* 147–165.

Feldenkrais, M. (1972). *Awareness through movement.* New York: Harper & Row.

Gevirtz, R., & Schwartz, M. S. (2005). The respiratory system in applied psychophysiology. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback. A practitioner's guide* (pp. 212–244). New York: Guilford Press.

Giardino, N. D., Chan, L., & Borson, S. (2004). Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: Preliminary findings. *Applied Psychophysiology and Biofeedback, 29*(2), 121–133. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/15208975.*

Gosselink, H. A. A. M., Wagenaar, R. C., Rijswijk, H., Sargeant, A. J., & Decramer, M. L. A. (1995). Diaphragmatic breathing reduces efficiency of breathing in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine, 151,* 1136–1142.

Grammatopoulou, E. P., Skordilis, E. K., Georgoudis, G., Haniotou, A., Evangelodimou, A., Fildissis, G., et al. (2014). Hyperventilation in asthma: A validation study of the Nijmegen Questionnaire—NQ. *Journal of Asthma, 51*(8), 839–846.

Grossman, P., de Swart, J. C. G., & Defares, P. B. (1985). A controlled study of a breathing therapy for treatment of hyperventilation syndrome. *Journal of Psychosomatic Research, 29*(1), 49–58.

Hagman, C., Janson, C., & Emtner, M. (2008). A comparison between patients with dysfunctional breathing and patients with asthma. *Clinical Respiratory Journal, 2*(2), 86–91. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/20298312.*

Hagman, C., Janson, C., & Emtner, M. (2011). Breathing retraining: A five-year follow-up of patients with dysfunctional breathing. *Respiratory Medicine, 105*(8), 1153–1159. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/21454062.*

Hagman, C., Janson, C., Malinovschi, A., Hedenstrom, H., & Emtner, M. (2016). Measuring breathing patterns and respiratory movements with the respiratory movement measuring instrument. *Clinical Physiology and Functional Imaging, 36*(5), 414–420.

Han, J. N., Stegen, K., Schepers, R., Vandenbergh, O., & Van de Woestijne, K. P. (1998). Subjective symptoms and breathing pattern at rest and following hyperventilation in anxiety and somatoform disorders. *Journal of Psychosomatic Research, 45*(6), 519–532. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/9859854.*

Han, J. N., Stegen, K., Simkens, K., Cauberghs, M., Schepers, R., de Bergh, O. V., et al. (1997). Unsteadiness of breathing in patients with hyperventilation syndrome and anxiety disorders. *European Respiratory Journal, 10,* 167–176.

Hanna, B. C., Woodman, P., & Adair, R. (2012). Assessing the role of chronic hyperventilation in patients with nasal congestion: Our experience in 118 patients. *Clinical Otolaryngology, 37*(2), 155–158. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/22515714.*

Heide, F. J., & Borkovec, T. D. (1983). Relaxation-induced anxiety: Paradoxical anxiety enhancement due to relaxation training. *Journal of Consulting and Clinical Psychology, 51,* 171–182.

Hornsveld, H. K., & Garssen, B. (1997). Hyperventilation syndrome: An elegant but scientifically untenable concept. *Netherlands Journal of Medicine, 50,* 13–20.

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59*(1), 12–19. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/2002127.*

Jansen, E., van Dixhoorn, J., & Eupen, L. (2015). *Chronic non-specific back and neck pain: Dysfunctional breathing and osteopathy.* Doctoral dissertation, University of Antwerp, Antwerp, Belgium.

Johnston, R., & Lee, K.-H. (1976). Myofeedback: A new method of teaching breathing exercises in emphysematous patients. *Physical Therapy, 56*(7), 826–829.

Kapandji, I. A. (1974). *The physiology of the joints: III*. London: Churchill Livingstone.

Kikuchi, Y., Okabe, S., Tamura, G., Hida, W., Homma, M., Shirato, K., et al. (1994). Chemosensitivity and perception of dyspnea in patients with a history of near-fatal asthma. *New England Journal of Medicine, 330*(19), 1329–1334. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/8152444*.

Kim, S., Wollburg, E., & Roth, W. T. (2012). Opposing breathing therapies for panic disorder: A randomized controlled trial of lowering vs raising end-tidal PCO_2. *Journal of Clinical Psychiatry, 73*(7), 931–939. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/22901344*.

Kotses, H., Harver, A., Segreto, J., Glaus, K. D., Creer, T. L., & Young, G. A. (1991). Long-term effects of biofeedback-induced facial relaxation on measures of asthma severity in children. *Biofeedback and Self-Regulation, 16,* 1–21.

Kruyssen, H. A. C. M., Leenders, C. M., Hellemans, I. M., Senten, M. C. M., Cluytmans, J. M., Craen, I. D., et al. (1996). Guidelines for cardiac rehabilitation 1995. *Cardiologie, 3,* 47–63.

Laoutaris, I., Dritsas, A., Brown, M. D., Manginas, A., Alivizatos, P. A., & Cokkinos, D. (2004). Inspiratory muscle training using an incremental endurance test alleviates dyspnea and improves functional status in patients with chronic heart failure. *European Journal of Cardiovascular Preventative Rehabilitation, 11*(6), 489–496. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/15580060*.

Lehrer, P., Buckman, J. F., Mun, E. Y., Vaschillo, E. G., Vaschillo, B., Udo, T., et al. (2013). Negative mood and alcohol problems are related to respiratory dynamics in young adults. *Applied Psychophysiology and Biofeedback, 38*(4), 273–283.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Shouen, L., Scardella, A., Siddique, M., et al. (2004). Biofeedback treatment for asthma. *Chest, 126,* 352–361.

Leuner, H. (1984). Indications and scientific basis of respiratory feedback. *General Practitioner, 6,* 344–354.

Ley, R. (1988). Panic attacks during relaxation and relaxation-induced anxiety: A hyperventilation interpretation. *Journal of Behavior Research and Experimental Psychiatry, 19,* 253–259.

Ley, R. (1994). An introduction to the psychophysiology of breathing. *Biofeedback and Self-Regulation, 19,* 95–96.

Ley, R., Timmons, B. H., Kotses, H., Harver, A., & Wientjes, C. J. E. (1996). Highlights of the annual meeting of the International Society for the Advancement of Respiratory Psychophysiology and the 14th International Symposium on Respiratory Psychophysiology. *Biofeedback and Self-Regulation, 21,* 241–260.

Linden, W. (1993). The autogenic training method of J. H. Schultz. In P. M. Lehrer & R. L. Woolfolk (Eds.), *Principles and practice of stress management* (2nd ed., pp. 205–229). New York: Guilford Press.

Lum, L. C. (1976). The syndrome of chronic habitual hyperventilation. In O. Hill (Ed.), *Modern trends in psychosomatic medicine* (pp. 196–230). London: Butterworth.

Lum, L. C. (1977). Breathing exercises in the treatment of hyperventilation and chronic anxiety states. *Chest, Heart and Stroke Journal, 2,* 6–11.

Lum, L. C. (1981). Hyperventilation and anxiety state. *Journal of the Royal Society of Medicine, 74,* 1–4.

Lumb, A. B. (2000). *Nunn's applied respiratory physiology.* Oxford, UK: Butterworth, Heineman.

Luthe, W. (1965). Autogene entladungen wahrend der Unterstufenubungen [Autogenic discharges during basic exercises]. In W. Luthe (Ed.), *Autogenes training correlationes psychosomaticae* (pp. 22–52). Stuttgart, Germany: Thieme.

MacHose, M., & Peper, E. (1991). The effect of clothing on inhalation volume. *Biofeedback and Self-Regulation, 16*(3), 261–265.

Mataheru, A., Overbeek, R., Scheffers, M., & van Dixhoorn, J. (2014). *Het effect van Adem—en Ontspanningstherapie op mensen met angstklachten* [*The effect of breathing—a relaxation therapy on people with anxiety symptoms*]. Unpublished master's thesis, Windesheim University for Applied Sciences, Zwolle, the Netherlands.

McLaughlin, L., Goldsmith, C. H., & Coleman, K. (2010). Breathing evaluation and retraining as an adjunct to manual therapy. *Manual Therapy, 16*(1), 51–52. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/20933458*.

Mehling, W.-E. (2001). The experience of breath as a therapeutic intervention: Psychosomatic forms of breath therapy. *Forschende KomplemtarMedizin und Klassische Naturheilkunde, 8,* 359–367.

Meuret, A. E., Rosenfield, D., Hofmann, S. G., Suvak, M. K., & Roth, W. T. (2009). Changes in respiration mediate changes in fear of bodily sensations in panic disorder. *Journal of Psychiatric Research, 43*(6), 634–641. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/18835608*.

Noseda, A., Carpiaux, J. P., Schmerber, J., Valente, F., & Yernault, J. C. (1994). Dyspnoea and flow-volume curve during exercise in COPD patients. *European Respiratory Journal, 7,* 279–285.

Noseda, A., Schmerber, J., Prigogine, T., & Yernault, J. C. (1993). How do patients with either asthma or COPD perceive acute bronchodilation? *European Respiratory Journal, 6,* 636–644.

Nummenmaa, L., Glerean, E., Hari, R., & Hietanen, J. K. (2014). Bodily maps of emotions. *Proceedings of the National Academy of Sciences of the USA, 111*(2), 646–651.

Parow, J. (1980). *Funktionelle Atmungstherapie* [Functional breathing therapy]. Heidelberg, Germany: Haug Verlag.

Peper, E. (1979). Passive attention: The gateway to consciousness and autonomic control. In E. Peper, S. Ancoli, & M. Quinn (Eds.), *Mind–body integration: Essential readings in biofeedback* (pp. 119–124). New York: Plenum Press.

Peper, E., Smith, K., & Waddell, D. (1987). Voluntary wheezing versus diaphragmatic breathing with inhalation (Voldyne) feedback: A clinical intervention in the treatment of asthma. *Clinical Biofeedback and Health, 10*(2), 83–88.

Peper, E., & Tibbets, V. (1992). Fifteen-month follow-up with asthmatics utilizing EMG/incentive inspirometer feedback. *Biofeedback and Self-Regulation, 17,* 143–151.

Peper, E., & Tibbets, V. (1994). Effortless diaphragmatic breathing. *Physical Therapy Products, 6,* 67–71.

Petersen, S., Orth, B., & Ritz, T. (2008). Awareness of breathing: The structure of language descriptors of respiratory sensations. *Health Psychology, 27*(1), 122–127. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/18230023*.

Petersen, S., Van Staeyen, K., Vogele, C., von Leupoldt, A., & Van den Bergh, O. (2015). Interoception and symptom reporting: Disentangling accuracy and bias. *Frontiers in Psychology, 6,* 732.

Phillipson, E. A., McClean, P. A., Sullivan, C. E., & Zamel, N. (1978). Interaction of metabolic and behavioral respiratory control during hypercapnia and speech. *American Review of Respiratory Disease, 117,* 903–909.

Revalidatiecommissie Nederlandse Vereniging Voor Cardiologie. (2011). *Multidisciplinaire Richtlijn Hartrevalidatie 2011.* Utrecht: NVVC.

Reybrouck, T., Wertelaers, A., Bertrand, P., & Demedts, M. (1987). Myofeedback training of the respiratory muscles in patients with chronic obstructive pulmonary disease. *Journal of Cardiopulmonary Rehabilitation, 7,* 18–22.

Ristiniemi, H., Perski, A., Lyskov, E., & Emtner, M. (2014). Hyperventilation and exhaustion syndrome. *Scandinavian Journal of Caring Sciences, 28*(4), 657–664.

Ritz, T., Meuret, A. E., Bhaskara, L., & Petersen, S. (2013). Respiratory muscle tension as symptom generator in individuals with high anxiety sensitivity. *Psychosomatic Medicine, 75*(2), 187–195. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/23324873*.

Roland, M., & Peper, E. (1987). Inhalation volume changes with inspirometer feedback and diaphragmatic breathing coaching. *Clinical Biofeedback and Health, 10*(2), 89–97.

Rosenkranz, M. A., & Davidson, R. J. (2009). Affective neural circuitry and mind–body influences in asthma. *NeuroImage, 47*(3), 972–980. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/19465136*.

Sakakibara, M., Takeuchi, S., & Hayano, J. (1994). Effect of relaxation training on cardiac parasympathetic tone. *Psychophysiology, 31,* 223–228.

Schein, M. H., Gavish, B., Herz, M., Rosner-Kahana, D., Naveh, P., Knishkowsky, B., et al. (2005). Treating hypertension with a device that slows and regularises breathing: A randomised, double-blind controlled study. *Journal of Human Hypertension, 15,* 271–278.

Smith, J. C. (1988). Steps toward a cognitive-behavioral model of relaxation. *Biofeedback and Self-Regulation, 13*(4), 307–329.

Stoeckel, M. C., Esser, R. W., Gamer, M., & von Leupoldt, A. (2018). Breathlessness amplifies amygdala responses during affective processing. *Psychophysiology, 55*(9), e13092.

Stough, C. (1970). *Dr. Breath: The story of breathing coordination*. New York: Morrow.

Terai, K., & Umezawa, A. (2004). Effects of respiratory self-control on psychophysiological relaxation using biofeedback involving the partial pressure of end-tidal carbon dioxide. *Japanese Society of Biofeedback Research, 30,* 31–37.

Thomas, M., McKinley, R. K., Freeman, E., Prodger, P., Foy, C., & Price, D. (2003). Breathing retraining for dysfunctional breathing in asthma: A randomised controlled trial. *Thorax, 58,* 110–115.

Thomas, M., McKinley, R. K., Mellor, S., Watkin, G., Holloway, E., Scullion, J., et al. (2009). Breathing exercises for asthma: A randomised controlled trial. *Thorax, 64*(1), 55–61. Retrieved from *www.ncbi.nlm.nih.gov/pubmed/19052047.*

Tiep, B. L. (1995). Biofeedback and ventilatory muscle training. In T. Kikuchi, H. Sakuma, I. Saito, & K. Tsuboi (Eds.), *Biobehavioral self-regulation* (pp. 398–406). Tokyo: Springer.

Tiep, B. L., Burns, M., Kao, D., Madison, R., & Herrera, J. (1986). Pursed lips breathing training using ear oximetry. *Chest, 90,* 218–221.

Ulrich, B. (1928). *Die Sängeratmung.* Leipzig, Germany: Dörffling & Franke Verlag.

Umezawa, A. (1992). Effects of stress on post expiration pause time and minute ventilation volume. In K. Shirakura, I. Saito, & S. Tsutsui (Eds.), *Current biofeedback research in Japan* (pp. 125–132). Tokyo: Shinkoh Igaku Shuppan.

Umezawa, A. (2001). Facilitation and inhibition of breathing during changes of emotion. In Y. Haruki, I. Homma, A. Umezawa, & Y. Masaoka (Eds.), *Respiration and emotion* (pp. 139–148). Tokyo: Springer Verlag.

van den Hout, M. A., & Kroeze, S. (1995). An untenable rationale for treating insomnia. *Perceptual and Motor Skills, 81,* 316–318.

Van Dixhoorn, J. (1985). Zur Effektivitat eines Entspannungstrainings in der Fruhrehabilitation von Myokardinfarktpatienten [Efficacy of relaxation training in the early rehabilitation of myocardial infarction patients]. In W. Langosch (Ed.), *Psychische Bewaltigung der chronischen Herzerkrankung* (pp. 308–327). Berlin: Springer.

Van Dixhoorn, J. (1990). *Relaxation therapy for cardiac rehabilitation: A randomized controlled clinical trial of breathing awareness as a relaxation method in the rehabilitation after myocardial infarction.* Doctoral dissertation, Rotterdam Erasmus University, Rotterdam, the Netherlands.

Van Dixhoorn, J. (1992). Voordelen en nadelen van adembewustwording bij therapeuten [Advantages and disadvantages of breathing awareness in therapists]. *Bewegen en Hulpverlening, 9,* 40–51.

Van Dixhoorn, J. (1994). Breath relaxation: Two-year follow-up of breathing pattern in cardiac patients. In *Proceedings of the 25th annual meeting of the Association for Applied Psychophysiology and Biofeedback* (pp. 176–179). Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Van Dixhoorn, J. (1997). Functional breathing is "whole-body" breathing. *Biological Psychology, 46,* 89–90.

Van Dixhoorn, J. (1998a). Cardiorespiratory effects of breathing and relaxation instruction in myocardial infarction patients. *Biological Psychology, 49,* 123–135.

Van Dixhoorn, J. (1998b). *Ontspanningsinstructie: Principes en oefeningen [Relaxation instruction: Principles and practice].* Maarssen, the Netherlands: Elsevier/Bunge.

Van Dixhoorn, J. (2002). Experiences of breathing therapy for patients with "hyperventilation" complaints and healthy students. *Biological Psychology, 59,* 234–235.

Van Dixhoorn, J. (2008). Whole body breathing. *Biofeedback, 36*(2), 54–58.

Van Dixhoorn, J. (2013). Spanningsregulatie in de behandeling van burn-out: Een exploratief onderzoek [Tension regulation in the treatment of burn-out: An exploratory study]. *Tijdschr Bedrijfs Verzekeringsgeneeskd, 21*(9), 412–418.

van Dixhoorn, J. (2014). Indirect approaches to breathing regulation. In L. Chaitow, D. Bradley, & C. Gilbert (Eds.), *Recognizing and treating breathing disorders* (pp. 155–161). London: Churchill Livingstone.

Van Dixhoorn, J. (2017). *Overspanning is een probleem in de hele gezondheidszorg [Excess tension is a problem in healthcare in general].* De Adem-en Ontspanningstherapie Stichting (AOS) (Bulletin no. 1).

Van Dixhoorn, J. J., & Anthonissen, E. (2015). Upper-thoracic (tense) breathing pattern: Relationship to respiration, tension/anxiety and general distress. *Biological Psychology, 104*(1), 160.

Van Dixhoorn, J. J., & Duivenvoorden, H. J. (1989). Breathing awareness as a relaxation method in cardiac rehabilitation. In F. J. McGuigan, W. E. Sime, & W. J. Macdonald (Eds.), *Stress and tension control* (Vol. 3, pp. 19–36). New York: Plenum Press.

Van Dixhoorn, J. J., & Duivenvoorden, H. J. (1999). Effect of relaxation therapy on cardiac events after myocardial infarction: A 5-year follow-up study. *Journal of Cardiopulmonary Rehabilitation, 19,* 178–185.

Van Dixhoorn, J. J., Duivenvoorden, H. J., Pool, J., & Verhage, F. (1990). Psychic effects of physical training and relaxation therapy after myocardial infarction. *Journal of Psychosomatic Research, 34*(3), 327–337.

Van Dixhoorn, J. J., Duivenvoorden, H. J., Staal, J. A., & Pool, J. (1989). Physical training and relaxation therapy in cardiac rehabilitation assessed through a composite criterion for training outcome. *American Heart Journal, 118*(3), 545–552.

Van Dixhoorn, J., & Folgering, H. (2015). The Nijmegen Questionnaire and dysfunctional breathing. *European Respiratory Journal Open Research, 1*(1).

Van Dixhoorn, J., & Hoefman, J. D. (1987). Registratie van behandelings resultaat van ademtherapie [Treatment outcome after breathing therapy]. *Nederlands Tijdschrift Fysiotherapie, 96*(1), 10–15.

Van Dixhoorn, J., Scheffers, M., & Bussbach, J. (2017). Relaxation therapy for anxiety: An observational study of a process-oriented approach. *International Journal of Stress Prevention and Wellbeing, 1*(art. 8), 1–6.

Van Dixhoorn, J., & White, A. R. (2005). Relaxation therapy for rehabilitation and prevention in ischaemic heart disease: A systematic review and meta-analysis. *European Journal of Cardiovascular Prevention and Rehabilitation, 12,* 193–202.

van Doorn, P., Folgering, H., & Colla, P. (1982). Control of the end-tidal PCO_2 in the hyperventilation syndrome: Effects of biofeedback and breathing instructions compared. *Bulletin Europeen de Physiopathologie Respiratoire, 18*(6), 829–836.

Vlemincx, E., Abelson, J. L., Lehrer, P. M., Davenport, P. W., Van Diest, I., & Van den Bergh, O. (2013). Respiratory variability and sighing: A psychophysiological reset model. *Biological Psychology, 93*(1), 24–32.

von Leupoldt, A., Petersen, S., Scheuchl, S., Fullekrug, K., Gerwin, C., Reer, R., et al. (2006). Reliability of verbal descriptors of dyspnea and their relationship with perceived intensity and unpleasantness. *Giornale italiano di medicina del lavoro ed ergonomia, 28*(3, Suppl. 2), 83–88.

Wakker, J., Bosscher, R., & Van Dixhoorn, J. (2009). Clinical effectiveness of breathing and relaxation therapy: Results in routine practice. Amsterdam: Vrije Universiteit, Faculty of Human Movement Sciences. Retrieved from *https://methodevandixhoorn.com/bibliotheek/onderzoek-vanuit-aot/107-clinical-effectiveness-of-breathing-and-relaxation-therapy/file.*

Whatmore, G. B., & Kohli, D. R. (1974). *The physiopathology and treatment of functional disorders.* New York: Grune & Stratton.

Wientjes, C. J. E. (1993). *Psychological influences upon breathing: Situational and dispositional aspects.* Doctoral dissertation, Tilburg University, Tilburg, the Netherlands.

Wientjes, C. J. E., & Grossman, P. (1994). Overreactivity of the psyche or the soma?: Interindividual associations between psychosomatic symptoms, anxiety, heart rate and end-tidal partial carbon dioxide pressure. *Psychosomatic Medicine, 56,* 533–540.

Wilhelm, F. H., Trabert, W., & Roth, W. T. (2001). Physiological instability in panic disorder and generalized anxiety disorder. *Biological Psychiatry, 49,* 596–605.

Wollburg, E., Meuret, A. E., Conrad, A., Roth, W. T., & Kim, S. (2007). Psychophysiological reactions to two levels of voluntary hyperventilation in panic disorder. *Journal of Anxiety Disorders, 22*(5), 886–898.

Xu, J. H., Ikeda, Y., & Komiyama, S. (1991). Biofeedback and the yawning breath pattern in voice therapy: A clinical trial. *Auris Nasus Larynx, 18,* 67–77.

Zeier, H. (1984). Arousal reduction with biofeedback-supported respiratory meditation. *Biofeedback and Self-Regulation, 9,* 497–508.

SECTION B

MEDITATIVE METHODS

CHAPTER 13

Modern Forms of Mantra Meditation for Stress Management

Patricia Carrington
Paul M. Lehrer

HISTORY OF THE METHOD

To the surprise of researchers investigating behavioral methods of dealing with stress, an ancient and revered method of ritual concentration known as "mantra meditation" turned out to be an unexpectedly promising candidate when it was reviewed in the latter part of the 1900s. This method seemed to fill a place that had long been neglected by modern methods of psychology. It was discovered that some modern forms of mantra meditation, simplified and divested of esoteric trappings and religious overtones, can possess outstanding therapeutic properties. This chapter presents ways in which these new, noncultic techniques can be applied to clinical practice.

Mantra meditation is distinguished by the use of a repeated sound (mantra) as the focus of meditation. In this respect, it differs from those meditation methods that direct the person's attention to the breath or the contents of the mind or to contemplation of some other meditational object. Mantra meditation differs from these other forms in the protocol followed, in the type of client it appeals to, and in its clinical applications. It is therefore discussed separately from mindfulness meditation, which is discussed by Kristeller in Chapter 14 of this volume.

Technically, meditation can be classified as "concentrative" or "nonconcentrative" in nature. A concentrative technique limits stimulus input by directing attention to a single unchanging or repetitive stimulus (e.g., a mantra, a candle flame). The directives of mantra meditation are concentrative in nature. A nonconcentrative technique expands the meditator's field of attention to include as much of his or her conscious mental activity as possible (e.g., the directives of mindfulness meditation as here defined are nonconcentrative in nature).

The modern forms of mantra meditation discussed in this chapter are simple to learn. These techniques are typically practiced while the person is seated in a quiet environment, with the object of the meditator's attention being a mentally repeated sound. When the meditator's attention wanders, he or she is directed to bring it back to this attentional object in an easy, unforced manner.

Mantra meditation is a simple procedure, and various forms of it have been used by numerous societies throughout recorded history to alter consciousness in a way that has been perceived as deeply beneficial. Traditionally, its benefits have been defined as spiritual in nature, and meditation has constituted a part of many religious practices. Simple forms of mantra meditation have been used for stress management with excellent results. Contributing to the rising interest in these meditative techniques is the fact that they are related to the biofeedback techniques (which also emphasize a delicately attuned awareness of inner processes) and to muscle relaxation and visualization techniques used in the behavior therapies.

In addition to providing deep relaxation, the majority of the meditative disciplines appear to assist the client in an area peripheral to many other therapeutic interventions: the fostering of a new kind of communication between the client and his or her own self, apart from his or her interpersonal environment. In a world in which inner enrichment from any source is a rare commodity, many people hunger for a more profound sense of self than is implicit in merely "getting along with others." Such people seek an awareness of their identity as *being* (as distinct from their identity as *doing*). The inner communion of meditation offers a means of fulfilling this need, thus promising to heal an aspect of the psyche that may be as needful as any other presently identified. The use of meditation, along with other forms of therapy, may therefore be an inevitable accompaniment to the trend currently seen in the behavioral sciences toward encompassing more and varied aspects of life.

Noncultic Methods

Of all the Westernized forms of mantra meditation, transcendental meditation (TM) has been, up until now, the most widely known and extensively studied—aside from mindfulness meditation in its various forms, which has also received a major portion of research attention. TM is more accurately described as "transitional" rather than modern, because it retains certain cultic features such as the *puja* (Hindu religious ceremony). TM teachers do not permit mental health practitioners to assume an active role in its clinical management unless they are also TM teachers. Despite its popularity with segments of the general public, therefore, the TM method has been less widely used in clinical settings than might be expected, considering the extensive research available on the effectiveness of this technique.

Among the clinically oriented mantra meditation techniques, *clinically standardized meditation* (CSM; Carrington, 1978) and the *respiratory one method* (ROM, using the word "one" as the mantra), most commonly known as the *relaxation response* technique (Benson, 1975), have been the most widely studied to date. Benson discontinued the use of ROM some years ago and instead uses a mantra-like prayer or the repetition of secular phrases, coupled with attention to the breath, to induce relaxation. He does not advocate any standardized approach (Benson & Stark, 1996), and, as with most health practitioners using mantra meditation to reduce stress, his standard protocol includes other therapeutic approaches including social support, cognitive skills training, and positive psychology (Park et al., 2013; Stahl et al., 2015). However, his original method is still studied experimentally and is therefore considered in this chapter.

These techniques were devised with clinical objectives in mind and are strictly noncultic (i.e., are divested of religious or esoteric trappings). The two methods differ from each other in several important respects, however. A trainee learning CSM selects a sound from a standard list of sounds (or creates one according to directions) and then repeats this sound mentally, without intentionally linking the sound to the person's breathing pattern or pacing it in any structured manner. CSM is thus a permissive meditation technique and may be subjectively experienced as almost "effortless." By contrast, when practicing ROM, the trainee repeats the word *one* (or another word or phrase) to himself or herself mentally, while at the same time intentionally linking this word with each exhalation. ROM is thus a relatively disciplined form of meditation, with the attention placed on two meditational objects—the chosen word and the breath. Accordingly, ROM requires more mental effort than CSM and may appeal to a different type of person.

Some nonconcentrative methods of meditation have also been used clinically. Mindfulness meditation is probably the most commonly used of the nonconcentrative methods for this purpose (see Kristeller, Chapter 14, this volume). It is somewhat more difficult to learn than the modern forms of mantra meditation, however, because it requires handling one's spontaneous train of thought in a manner that is foreign to most people. By contrast, repeating a sound mentally over and over again, as in mantra meditation, is a relatively simple act that requires little preparation to be executed successfully. Thus the student's success in learning a simple form of mantra meditation often depends more heavily on the individual expertise and personality of the instructor. Other nonstandardized forms of meditation are not discussed here, although their usefulness in the proper hands is not to be negated.

The Physiology of Mantra Meditation

All of the simplified mantra meditation techniques, including TM, have in common the fact that they can rapidly bring about a deeply restful state that possesses certain well-defined characteristics. Although mantra meditation is not the only intervention that can create such a state, it is clearly one of the most effective for this purpose. An extensive series of psychophysiological studies in this area established the nature of the meditative state in the 1970s and 1980s. Because its basic characteristics were established in that period, only a handful of studies have been conducted in this area following that time. For the most part, these newer studies simply confirm the earlier ones and add some refinements.

Much research has shown that during mantra meditation, body and mind typically enter a state of profound rest. Oxygen consumption can be lowered during 20–30 minutes of meditation to a degree ordinarily reached only after 6–7 hours of sleep (Wallace, Benson, & Wilson, 1971), and heart and respiration rates typically decrease during meditation (Allison, 1970; Wallace, 1970). However, heart rate can also speed up during meditation in response to the introduction of stimuli perceived as stressful (Goleman & Schwartz, 1976), a finding that these researchers interpret as showing heightened alertness. The latter researchers' findings also indicated faster physiological recovery—a stress-protective effect that the authors suggest may reflect the fact that an adaptive organism should be prepared to respond to threatening stimuli but also should recover quickly so that there are no ill effects of long-term stress.

In addition, electrical resistance of the skin tends to increase during meditation (Wallace, 1970), suggesting a lowering of sympathetic arousal, and a sharp decline in the concentration of blood lactate may occur (Wallace et al., 1971). Although some studies have failed to show such clear-cut indications of decreased physiological arousal or heightened recoverability during meditation as the preceding studies do, subjective reports of meditators typically describe marked anxiety reduction during this state, and clinical reports generally confirm the anxiety-reducing properties of meditation (Delmonte, 1987).

During the meditative state, the electroencephalogram (EEG) shows an alert–drowsy pattern with high alpha- and occasional theta-wave patterns (Harne & Hiwale 2018) as well as an unusual pattern of swift shifts from alpha to slower (more sleep-like) frequencies and then back again (Das & Gastaut, 1957; Wallace et al., 1971). These findings suggest that meditation may be an unusually fluid state of consciousness, partaking of qualities of both sleep and wakefulness, and possibly resembling the hypnogogic or "falling asleep" state more than any other state of consciousness. Hippocampal activation has also been reported after meditation (Engstrom, Pihlsgard, Lundberg, & Soderfeldt, 2010), suggesting strengthened modulation of emotion. A number of studies have also shown that the physiology of meditation differs from that of ordinary rest with eyes closed and from that of most hypnotic states (Brown, Stewart, & Blodgett, 1971; Travis & Wallace, 1999; Wallace, 1970; Wallace et al., 1971). Other studies, however, have shown that true uninterrupted "rest," as induced in the laboratory, shares many of the same features.

In general, the research suggests that during meditation deep physiological relaxation, somewhat similar to that occurring in the "deepest" non–rapid-eye-movement (NREM) sleep phase, occurs in a context of wakefulness. Wallace and colleagues (1971) have thus characterized meditation as being a "wakeful, hypometabolic physiologic state" (p. 79), and Gellhorn and Kiely (1972) consider it a state of trophotropic dominance compatible with full awareness. When practiced regularly, meditation also appears to alter behavior occurring outside of the meditative state itself, with both clinical and research evidence suggesting that a number of beneficial changes may take place in people who meditate. These changes are described later in this chapter, when clinical indications for meditation are discussed.

THEORETICAL FOUNDATIONS

Several theories have been proposed concerning the way mantra meditation operates to effect its reported changes. Four of the most widely accepted are presented next.

Global Desensitization

There is an interesting similarity between the situation that occurs during a meditation session and that which occurs during the technique of systematic desensitization used in behavior therapy (Carrington & Ephron, 1975; Goleman, 1971). In the latter process, increasingly greater increments of anxiety (prepared in a graded hierarchy) are systematically "counterconditioned" by being paired with an induced state of deep relaxation. If the treatment is successful, presentation of the originally disturbing stimulus ceases to produce anxiety. In mantra meditation, awareness of the meditative "focus" (the mantra) becomes a signal for turning inward and experiencing a state of deep relaxation. Simultaneously, the meditator maintains a permissive attitude with respect to thoughts, images, or sensations experienced during meditation. Without rejecting or unduly holding onto these thoughts, he or she lets them "flow through the mind" while continuing to direct attention to the focal point of the meditation—the mantra.

This dual process—free-flowing thoughts occurring simultaneously with a repetitive stimulus that induces a state of calm—sets up a subjective state in which deep relaxation is paired with a rapid, self-initiated review of an exceedingly wide variety of mental contents and areas of tension, both verbal and nonverbal. As thoughts, images, sensations, and amorphous impressions drift through the mind during meditation, the soothing effect of the mantra appears to neutralize the disturbing thoughts. No matter how unsettling a meditation session may feel, a frequent response of meditators is that they discover that, on emerging from meditation, the "charge" has been taken off their current concerns or problems.

Do the modern forms of mantra meditation work, then, merely because they are a form of systematic desensitization? This theory would seem to overlook certain important differences between the two approaches. In systematic desensitization, the therapist and patient work together to identify specific areas of anxiety and then proceed to deal with a series of single isolated problems in a sequential, highly organized fashion. During meditation, the areas of anxiety to be "desensitized" are selected by the meditator in an entirely automatic and nonconscious manner. At this time, the brain of the meditator might be said to act like a computer programmed to run certain material through "demagnetizing" circuits capable of handling large amounts of data at one time. We might conceptualize subsystems within the brain scanning vast memory stores at lightning speed during the meditative state, particularly those most salient at the time. For these reasons, meditation would seem to operate with a considerably wider scope than systematic desensitization, although, for exactly this reason, it may lack the clinical precision of the latter.

Blank-Out

Ornstein (1972) has proposed that mantra meditation (or other forms of concentrative meditation in which stimulus input is intentionally limited) may create a situation such as occurs when the eye is prevented from continuously moving over the surface of the visual field but is instead forced to view a constant fixed image without recourse to its natural scanning. When an image is projected onto a contact lens placed over the retina, the lens can follow the movement of the eye, so that the image becomes stabilized in the center of the visual field. Under such conditions, the image soon becomes invisible; without constantly shifting his or her eyes to different parts of the perceived image, the person apparently cannot register the object mentally. At this point, which Ornstein refers to as *blank-out,* prolonged bursts of alpha waves may be recorded in the occipital cortex.

It may be, therefore, that the central nervous system is so constructed that if awareness of any sort is restricted to one unchanging source of stimulation, then consciousness of the external world may be turned off or greatly attenuated, and the individual may achieve a form of mental blank-out. Because mantra meditation involves continuously recycling the same input over and over, it could possibly result in a blank-out effect, which in turn has the effect of temporarily clearing the mind of all thoughts. The aftereffect of blank-out may be an opening up of awareness and a renewed sensitivity to stimuli. After meditation, some meditators seem to experience an innocence of perception similar to that of the young child who is maximally receptive to all stimuli.

Although Ornstein (1972) does not address the therapeutic implications of the blank-out effect, it is evident that, at the least, such a phenomenon may interrupt an unproductive mental set, thus giving the meditator the opportunity to restructure his or her thoughts along more productive lines. This could result in a fresh point of view on emotional problems, as well as on other aspects of life. Also, becoming more open to direct sensory experience may in itself be valuable in a world beset by problems that derive from overemphasis on cognitive activity. The enlivened experiencing that follows meditation (often described by meditators as “seeing colors more clearly,” “hearing sounds more sharply,” or “sensing the world more vividly”) may in fact be a prime reason for the antidepressive effects of meditation.

Effects of Rhythm

In mantra meditation, in which a lilting sound is continuously repeated, rhythm is an obvious component. But rhythm also plays a role in all other forms of meditation, as the inner stillness involved allows the meditator to become profoundly aware of his or her own bodily rhythms. In the unaccustomed quiet of the meditative state, one’s own breathing may be intimately sensed, the pulse rate may be perceived, and even such subtle sensations as the flow of blood through the veins are sometimes described as emerging into awareness. Some meditative techniques even use bodily rhythms as their object of focus, as when the Zen meditator is instructed to concentrate on his or her own natural, uninfluenced breathing.

This rhythmic component of mantra meditation may be a major factor in inducing calm. Rhythm has universally been used as a natural tranquilizer; virtually all known societies use repeated sounds or rhythmic movements to quiet agitated infants, for example. The world over, parents have rocked children, gently hummed lullabies to them, recited nursery rhymes, repeated affectionate sounds in a lilting fashion, or bounced the children rhythmically on their laps, with an intuitive awareness of the soothing effects of such rhythmic activities on the children’s moods. Similarly, in the psychological laboratory, Salk (1973) demonstrated that neonates responded to a recorded normal heartbeat sound (played to them without interruption day and night) by greatly lessening their crying, as compared with a control group of infants who were not exposed to the sounds, and also by gaining more weight than the controls. If contacting deep biological rhythms in oneself is a prominent component of meditation, then regular meditation might be expected to exert a deeply soothing effect. One might, so to speak, gain considerable stabilization from returning periodically to a source of well-being (in meditation) from which one could draw strength in order to deal more effectively with an outer environment whose rhythms are, more often than not, out of phase with one’s own. Some internal body rhythms correspond to resonant oscillations caused by homeostatic processes. See Lehrer, Chapter 10, this volume, for one example: the rhythm of the baroreflex at about 0.1 Hz (six cycles per minute, or 10 seconds per cycle), which can be stimulated by breathing at this frequency. In a study of zen monks doing zen meditative exercises, the meditation altered breathing to this frequency (Lehrer, Sasaki & Saito, 1999), which itself appears to stimulate and strengthen processes in the cingulate cortex and hippocampus that modulate emotion (Mather, 2019; Vaschillo, Vaschillo, Buckman, & Bates, 2019). It is notable that many chants and hymns, as well as soothing music, often also have phrase lengths of about 10 seconds.

Balance between Cerebral Hemispheres

Research suggests that during mantra meditation a greater equalization in the workload of the two cerebral hemispheres may occur (Banquet, 1973). Verbal, linear, time-linked thinking (processed through the left hemisphere in the right-handed person) seems to be lessened during meditation as compared with the role it plays in everyday life, whereas holistic, intuitive, wordless thinking (usually processed through the right hemisphere) comes more to the fore. The therapeutic effects derived from meditation may reflect this relative shift in balance between the two hemispheres. Consistent with these findings, Thomas, Jamieson, and Cohen (2014) reported that advanced Yoga practitioners who used mantra meditation showed greater activity in right-hemisphere areas, although this pattern may also reflect greater emotionality (Gainotti, 2019). Intermediate practitioners showed increased right hemisphere slow wave activity, suggesting decreased imagery but also decreased emotionality. Fox et al. (2016) found that mantra meditation produced increased activity in brain areas involved in verbal behavior and a sharp focus of attention and well-practiced motor activity. These areas also mediate a blockade of incoming sensory stimuli and awareness of body sensations. These findings would be consistent with the common clinical finding that mantra meditation can be specifically helpful in blocking intrusive or obsessional thoughts as well as sensations of pain and stress-related body symptoms. In this way saying a mantra or similar repetitive verbalizations may block worry or obsessional activity through effortful attention to the mantra (Cooney Roxbury, 2018; Lawrence et al., 2020). Worry and mantra meditation may be incompatible activities, for which the mantra may win out.

During the early stages of meditation practice, when the technique is relatively new to the meditator, the left-hemispheric activity of the brain—which predominates during waking life in our modern world, often almost to the exclusion of right-hemispheric activity—has been shown to take a lesser role during meditation, with a shift toward right-hemisphere dominance occurring (Davidson, Goleman, & Schwartz, 1976). During the more advanced types of meditation, however, EEG records of experienced meditators frequently display an unusual balancing of the activity of the two cerebral hemispheres during meditation (Earle, 1981). In terms of the clinical applications of mantra meditation considered here, this distinction is relatively unimportant, because the "early" stages of a meditation practice constitute the entire meditative experience for the vast majority of those who take up modern forms of mantra meditation. An occasional client in psychotherapy does advance beyond these beginning stages, but such a person is likely to be using meditation to explore altered states of consciousness or to further spiritual development rather than for therapeutic purposes. More advanced practices of meditation are, of course, valid in their own right, but a discussion of them is beyond the scope of this chapter.

Because restrictive moral systems are for the most part transmitted verbally, with much role modeling dependent on verbal imitation, ameliorative effects of meditation on self-blame—a clinically relevant benefit of this technique—might be explained by this basic shift away from the verbal left-hemispheric mode during meditation. Minimizing verbal–conceptual experience (yet still remaining awake) may afford the individual temporary relief from self-derogatory thoughts, as well as from excessive demands on the self that have been formulated through internal verbalizations. Having obtained a degree of relief from these verbal injunctions during the meditative state, the meditator may find him- or herself less self-critical when returning to active life. The reduction in the strength of self-criticism may have generalized from the meditative state to the life of action.

There are, therefore, a number of theoretical reasons why a simple form of meditation may be of benefit in clinical practice. We now turn to the identification of those clinical conditions that have been shown to respond to the mantra meditation techniques.

ASSESSMENT

Clinical Conditions Responding to Mantra Meditation

Based on research and clinical reports, a substantial body of knowledge has accumulated on the usefulness of meditation in clinical practice. Clinical effects of mantra meditation are summarized in Table 13.1, from a search for "mantra meditation" in the PsycInfo and Medline databases. As can be seen, there is evidence that mantra meditation can improve various symptoms of stress and psychological distress, including anxiety, daily stress, depression, anger, substance craving, and PTSD symptoms. Physical symptoms affected by stress also are helped, including various cardiorespiratory symptoms, immune function, pain, and fatigue.

TABLE 13.1. Clinical Mantra Meditation Effects: Controlled Trials and Evidence Reviews

Condition/outcome	Studies
Cardiac/vascular conditions	Barnes & Orme-Johnson (2012); Bernardi, Bordino, Bianchi, & Bernardi (2017); Goldstein, Josephson, Xie, & Hughes (2012); Ooi, Giovino, & Pak (2017); Park & Han (2017); Shi et al. (2017)
Respiratory function and conditions	Bernardi et al. (2017)
Stress signs and symptoms	Brat (2018); Carrington et al. (1980); Janowiak & Hackman (1994); Maclean et al. (1997); Shulte (2018); Waelde, Meyer, Thompson, Thompson, & Gallagher-Thompson (2017)
Anxiety	Cooney Roxbury (2018); Domar, Noe, & Benson (1987); Lehrer, Schoicket, Carrington, & Woolfolk (1980); Leserman, Stuart, Mamish, & Benson (1989); Glueck (1973); Pearl & Carlozzi (1994); Travis et al. (2018); Zuroff & Schwartz (1978)
Depression, negative mood and affect	Carrington et al. (1980); Innes, Selfe, Kandati, Wen, & Huysmans (2018); Kirsten (2001); Oman & Bormann (2015); Torkamani, Aghayousefi, Alipour, & Nami (2018); Travis et al. (2018)
Sleep disturbance	Beck et al. (2017); Cooney Roxbury (2018); Innes et al. (2018)
Substance abuse	Benson & Wallace (1971); Haaga et al. (2011); Hawkins (2003); Shafii, Lavely, & Jaffe (1974, 1975); Parks & Marlatt (2004)
Immune function	Infante et al. (2014); Torkamani et al. (2018)
Pain	Innes et al. (2018); Yogitha, Nagarathna, John, & Nagendra (2010)
Fibromyalgia symptoms	Kozasa et al. (2012)
PTSD	Kang et al. (2018); Lang et al. (2012); Nidich et al. (2018); Oman & Bormann (2015)
Feelings of well-being	Oman & Bormann (2015)
Anger	Carrington et al. (1980); Travis et al. (2018); Walton & Levitsky (2003)
Fatigue	Travis et al. (2018)

As in most areas of research, however, not everyone agrees in interpretation of the findings. Holmes (1984), for example, considers meditation to be no more effective in lowering arousal or providing therapeutic benefit than is resting with eyes closed, whereas other researchers (Benson & Friedman, 1985; Delmonte, 1984; Shapiro, 1985; Suler, 1985; Travis et al., 2010; Travis & Wallace, 1999; West, 1985) cite compelling evidence to support the concept that meditation possesses some special therapeutic properties distinct from those of rest. Because many of the more clinically relevant effects of meditation are not readily identifiable by standard psychometric measures, it is probably necessary for the clinician to note only that the conditions of a meditation experiment tend to create a type of uninterrupted "guilt-free" rest that is atypical of our society. Such rest can occur in the laboratory because the experimenter has carefully set up the conditions for it: Rest has become a *demand characteristic* of the experiment. Laboratory-induced rest may well possess some special therapeutic properties, particularly if, while resting, the participant experiences what Carrington (1977) has called the *meditative mood*.

Most people in our fast-paced society find it difficult, if not impossible, to truly rest during the day, and therefore a practice of meditation may supply a highly structured, especially effective form of enforced rest each day—one that is easier for the average person to observe than are vague therapeutic prescriptions to "take it easy and get more rest." In fact, meditation may be particularly effective in this respect because it is a novel, out-of-the-ordinary activity.

Such practical considerations as these constitute the focus of indications for mantra meditation in clinical practice. The discussion that follows summarizes the major clinical findings in this area.

Reduction in Tension/Anxiety

In research measuring the effects of mantra meditation on anxiety, results have consistently shown anxiety to be sharply reduced in a majority of participants after they commenced the practice of meditation (Carrington, 1998; Pearl & Carlozzi, 1994; Lehrer, Schoicket, Carrington, & Woolfolk, 1980; Travis et al., 2018; Zuroff & Schwarz, 1978). There is also some evidence suggesting that the regular practice of meditation may facilitate a reduction in anxiety for individuals with clinically elevated (i.e., high or average) anxiety levels, but that it shows a "floor" effect (i.e., not much change) in those with low anxiety (Delmonte, 1987). In addition, meditation may be less effective for some patients with long-term severe anxiety neurosis or those who suffer from panic disorder, because such patients can easily be overwhelmed by their symptoms and drop out of the practice. Glueck (1973), however, in a study conducted with a group of psychiatric inpatients, found that dosages of psychotropic drugs could be greatly reduced after these patients had been meditating for several weeks; in a majority of cases, the use of sedatives could also be reduced or eliminated in these patients. Meditation has also successfully been used to lower the anxiety experienced by patients preparing for cardiac surgery (Leserman, Stuart, Mamish, & Benson, 1989) and for ambulatory surgery (Domar, Noe, & Benson, 1987).

The quieting effects of mantra meditation differ, however, from the effects brought about by psychotropic drugs. Whereas the relaxation brought about by some tranquilizing drugs may slow the person down and cause grogginess, the relaxation resulting from meditation does not bring with it any loss of alertness. On the contrary, meditation seems, if anything, to sharpen alertness. Groups of meditators have been shown to have faster reaction times (Appelle & Oswald, 1974), to have better refined auditory perception (Pirot, 1978), to show increased vigor (Kirsten, 2001), and to perform more rapidly and accurately on perceptual–motor tasks (Rimol, 1978) than nonmeditating controls. Mantra meditation may, therefore, be indicated in cases in which anxiety is a problem, and it can often be used productively in place of tranquilizers or as a supplement to drug treatment.

Attenuated Stress Responses

Several studies have shown psychophysiological indicators of stress to be sharply reduced in persons who practice mantra meditation. Credidio (1982) found that CSM meditators showed significantly greater frontalis electromyographic (EMG) decreases and peripheral skin temperature increases than did a group practicing biofeedback. Lehrer et al. (1980) showed that participants practicing CSM meditation, compared with those practicing progressive relaxation or with controls, displayed greater cardiac deceleration, more frontal alpha, and fewer symptoms of cognitive anxiety than those in the other groups. These researchers concluded that meditation may prepare people to cope better with stress. Similarly, a study by Maclean and colleagues (1997) showed positive changes in neuroendocrine responses to laboratory stress in participants practicing transcendental meditation. These and similar studies confirm a number of clinical findings that suggest that mantra meditation can be a highly effective intervention when a person is under high stress.

Improvement in Stress-Related Illnesses

Many stress-related illnesses have proven responsive to meditation. Research has shown mantra meditation to be correlated with improvement in the breathing patterns of patients with bronchial asthma (Honsberger & Wilson, 1973); with decreased blood pressure in both pharmacologically treated and untreated hypertensive patients (Benson, 1977; Friskey, 1985; Hafner, 1982; Kondwani, 1998; Patel, 1973, 1975); with reduced premature ventricular contractions in patients with ischemic heart disease (Benson, Alexander, & Feldman, 1975); with reduced symptoms of angina pectoris (Tulpule, 1971; Zamarra, Besseghini, & Wittenberg, 1978); with reduced cardiovascular and all-cause mortality (Barnes, 1997); with reduced serum cholesterol levels in hypercholesterolemic patients (Cooper & Aygen, 1979); with reduced sleep-onset insomnia (Miskiman, 1978; Woolfolk, Carr-Kaffashan, McNulty, & Lehrer, 1976); with amelioration of stuttering (McIntyre, Silverman, & Trotter, 1974); with lowered blood sugar levels in diabetic patients (Heriberto, 1989); with amelioration of psoriasis (Gaston, 1988–1989); with reduced pain and bloating in irritable bowel syndrome (Keefer & Blanchard, 2002); and with reductions in the symptoms of psychiatric illness (Glueck & Stroebel, 1975). Studies have also shown that meditation may reduce salivary bacteria and thus may be useful in treating dental caries (Morse, 1982) and may decrease periodontal inflammation (Klemons, 1978). It may also reduce some coronary-prone behavior patterns (Muskatel, Woolfolk, Carrington, Lehrer, & McCann, 1984) and may be beneficial in lowering central nervous system responsivity to norepinephrine (Benson, 1989). Mantra meditation can thus be a useful intervention in a wide variety of stress-related illnesses.

Increased Productivity

Mantra meditation may bring out increased efficiency by eliminating unnecessary expenditures of energy, and a beneficial surge of energy is often noted in persons who have commenced the practice (e.g., Kirsten, 2001). This energy can manifest itself variously as a lessened need for daytime naps, increased physical stamina, increased productivity on the job, increased ideational fluency, the dissolution of writer's or artist's "block," or the release of hitherto unsuspected creative potential. Mantra meditation may therefore be useful when it is desirable to increase a client's available energy and/or when a client is experiencing a block to productivity.

Improvement in Cognitive Functioning

Several studies have shown improved cognitive functioning among those who meditate regularly. A study by Yucel (2001) showed the beneficial effects of meditation on memory in elderly persons. Benson, Wilcher, Greenberg, Huggins, and Ennis (2000) found that students who meditate tend to achieve higher grade-point averages, and Kirsten (2001), studying a group of secondary school teachers who were taught CSM, found that these meditators scored significantly lower on the Confusion–Bewilderment scale of the Profile of Mood States (POMS) than did nonmeditating teachers. These and similar studies suggest that mantra meditation can be used productively in the educational system.

Lessening of Self-Blame

A useful by-product of mantra meditation may be increased self-acceptance, often evidenced in clients as a lessening of unproductive self-blame. A spontaneous change in the nature of a meditator's self-statements—from self-castigating to self-accepting—suggests that the noncritical state experienced during the meditation session itself can generalize to daily life. Along with the tendency to be less self-critical, the meditator may show a simultaneous increase in tolerance for the human frailties of others, and concomitant improvement often occurs in interpersonal relationships. Mantra meditation may therefore be indicated when a tendency toward self-blame is excessive or when irrational blame of others has become a problem.

Antiaddictive Effects

Several studies (Benson & Wallace, 1971; Hawkins, 2003; Shafii, Lavely, & Jaffe, 1974, 1975) have shown that, at least in persons who continue meditating for long periods of time (usually for a year or more), a marked decrease may occur in the use of nonprescription drugs, such as marijuana, amphetamines, barbiturates, and psychedelic substances (e.g., LSD). Many long-term meditators, in fact, appear to have discontinued use of such drugs entirely. Similar antiaddictive trends have been reported in ordinary cigarette smokers and abusers of alcohol, as well (Murphy, Pagano, & Marlatt, 1986; Royer, 1994; Shafii, Lavely, & Jaffe, 1976). Mantra meditation may therefore be useful for a patient suffering from an addiction problem, particularly if that problem is in its incipient stage.

Mood Elevation

Research and clinical evidence suggest that people suffering from mild chronic depression or from reactive depression may experience distinct elevation of mood after commencing mantra meditation (Carrington et al., 1980; Kirsten, 2001). People with acute depressive reactions do not generally respond well to meditation, however, and are likely to discontinue practicing it (Carrington & Ephron, 1975). Meditation, therefore, appears indicated in mild or chronic depressive reactions but not in acute depressions.

Increase in Available Affect

Those who have commenced meditating frequently report experiencing pleasure, sadness, anger, love, or other emotions more easily than before. Sometimes they experience emotions that have previously been unavailable to them. Release of such emotions may occur during a meditation session or between sessions and may be associated with the recovery of memories that are highly emotionally charged (Carrington, 1977, 1998). Meditation is therefore indicated when affect is flat, when the client tends toward overintellectualization, or when access to memories of an emotional nature is desired for therapeutic purposes.

Increased Sense of Identity

Meditating clients frequently report that they have become more aware of their own opinions since commencing meditation, that they are not as easily influenced by others as they were previously, and that they can arrive at decisions more quickly and easily. They may also be able to sense their own needs better and thus may become more outspoken and self-assertive and more able to stand up for their own rights effectively. Such effects may not be easily measurable by any existing tests, although it is possible that the trait known as *field independence* may be relevant to some of the effects noted. Several studies (Hines, 1970, cited in Carrington, 1977; Pelletier, 1978; Sridevi & Krishna, 2003) have shown changes in the direction of greater field independence (or "inner-directedness") following the commencement of the practice of mantra meditation, whereas other researchers have found no such changes. The clinically important observation that there tends to be an increased sense of identity in meditators may not yet have been validly tested in an experimental setting.

One result of the increased sense of identity noted by clinicians may be marked improvement in the ability of a meditator to separate from significant others when such separation is called for. Meditation can thus be extremely useful in pathological bereavement reactions or in cases in which an impending separation (threatened death of a loved one, contemplated divorce, upcoming separation from growing children, etc.) presents a problem. Meditation is therefore indicated in cases in which separation anxiety is a problem. Because it is particularly useful in bolstering the inner sense of self that is necessary for effective self-assertion, it may also be helpful as an adjunct to assertiveness training.

Lowered Irritability

The meditating person may become markedly less irritable and impulsive in his or her interpersonal relationships within a relatively short period of time (Carrington et al., 1980). Kirsten (2001), for example, found that a group of teachers practicing CSM scored significantly lower on the Anger–Hostility and Hostility scales in the POMS and the Symptom Checklist–90 (SCL-90) after commencing meditation than did matched controls. A similar reduction in irritability and impulsivity could well be related to the lowered recidivism noted in prisoners practicing mantra meditation (Alexander, Walton, & Goodman, 2003; Anklesaria & King, 2003; Hawkins, 2003; Rainworth, Alexander, & Cavanaugh, 2003). Meditation thus appears indicated in cases in which impulsive outbursts or chronic irritability is a symptom. This recommendation includes cases of organic irritability, as preliminary observations have shown meditation to be useful in increasing overall adjustment in several cases of brain injury (Glueck, 1973).

How to Assess for Use of the Method

A few attempts have been made to identify personality characteristics of the meditation-responsive person. Most of these have led to inconclusive results, with the possible exception of the research on *absorption,* a component of hypnotic susceptibility. Absorption refers to the disposition to display episodes of total attention "during which the available representational apparatus seems to be entirely dedicated to experiencing and modeling the attentional object, be it a landscape, a human being, a sound, a remembered incident, or an aspect of one's self" (Tellegen & Atkinson, 1974, p. 274). Meditative skills such as focusing and receptivity may be reflected in items on the Tellegen Absorption Scale. For example, "When I listen to music I can get so caught up in it that I don't notice anything else" may reflect the focusing ability that Smith (1987) considers an essential meditative skill. In the same manner, the statement "I sometimes 'step outside' my usual self and experience an entirely different state of being" may reflect a receptivity to altered states of consciousness useful for meditation. Some evidence (Davidson & Goleman, 1977; Tjoa, 1975; Warrenburg & Pagano, 1982–1983) suggests that the absorption trait may predict a positive response to meditation, although this possibility has not been tested in clinical settings.

The majority of the studies attempting to predict what kind of person responds positively to meditation or stays with the practice over time have used nonclinical populations, and their criteria for "responsiveness to meditation" have generally not been relevant to problems involved in clinical assessment. One of the only measures of clinical improvement that has been experimentally addressed in meditation research in an attempt to identify a correlation with personality factors is improvement in anxiety. Beiman, Johnson, Puente, Majestic, and Graham (1980) noted that participants who reported more "internal locus of control" prior to learning meditation showed greater reductions in anxiety as measured by the Fear Survey Schedule. Smith (1978) found that reductions in trait anxiety following mantra meditation training were moderately correlated with two of Cattell's Sixteen Personality Factor Inventory (16-PF) factors: *Autia* (preoccupation with inner ideas and emotions) and *Schizothymia* (steadiness of purpose, withdrawal, emotional flatness, and "coolness"). However, when we (Carrington et al., 1980) studied employee stress in a large corporation, we found no significant correlations between any of the 16-PF factors (including anxiety) measured at pretest and subsequent drops in symptomatology as measured by the Symptom Checklist 90—Revised (SCL-90-R; Derogatis, Rickels, & Rock, 1976), a validated self-report inventory.

At this point, therefore, the research is too inconclusive to permit us to predict which clients will respond to meditation by means of standard personality tests. There has, however, been an attempt to identify predictive personality variables correlated with successful meditation practice on a theoretical basis. Davidson and Schwartz (1976) have suggested that relaxation techniques have varied effects, depending on the system at which they are most directly aimed. They categorize progressive relaxation as a "somatically oriented technique," because it involves learning to pay closer attention to physiological sensations, particularly muscle tension; they categorize forms of meditation in which a word or sound is internally repeated as "cognitively oriented techniques," as repeating a word (i.e., the mantra) presumably blocks other ongoing cognitive activity. In support of this idea, Schwartz, Davidson, and Goleman (1978) report questionnaire data that show that meditation produces greater decreases in cognitive symptoms of anxiety than does physical exercise, whereas exercise appears to produce greater decreases in somatic anxiety symptoms.

On the basis of the Davidson–Schwartz hypothesis, some clinicians have felt justified in advising meditation for clients who show symptoms of cognitive anxiety and in advising physiologically oriented techniques, such as progressive relaxation or autogenic training, for those who show symptoms of somatic anxiety. Although this criterion has the advantage of offering the therapist clear-cut guidelines, the empirical support for cognitive-versus-somatic specialization remains at best insubstantial. Given the absence of any solid predictive measures at present and the fact that even those that show promise (such as the Tellegen Absorption Scale) are not readily available in the clinic, a clinician attempting to assess the suitability of meditation for a particular client will do well to determine whether this client shows one or more of the meditation-responsive symptoms or difficulties.

If the therapist determines that the client possesses the requisite pathology for use of meditation, he or she should recognize that other modalities may also be used for treating these same symptoms. At this point, therefore, the decision to employ meditation becomes a practical one. The following are some of the factors that may guide this decision:

1. *Self-discipline*. The degree to which the client has a disciplined lifestyle may be an important factor to consider when deciding on mantra meditation as a stress management technique. This form of meditation requires less self-discipline than do most other methods currently used for stress control. The technique itself can usually be taught in a single session, with the remainder of the instruction consisting of training in practical management of the method. Unlike some other techniques, mantra meditation does not require memorizing and carrying out any sequential procedures. It does not even require the mental effort involved in visualizing muscle groups and their relaxation or in constructing "calm scenes" or other images. The modern forms of mantra meditation are simple one-step operations that soon become quite automatic. They are therefore particularly useful for those clients who may not be willing to make a heavy commitment in terms of time or effort or in situations in which relatively rapid results are desired. By contrast, mindfulness meditation often requires more commitment and self-discipline on the part of the learner.

2. *Self-reinforcing properties*. For many clients, the peaceful, drifting mental state of meditation is experienced as unusually pleasurable, a "vacation" from all cares. This self-reinforcing property of meditation makes it especially appealing to many clients. Other things being equal, a modern form of mantra meditation is more likely to be continued, once experienced, than are the more focused relaxation procedures. Therefore, when motivation to continue with a program for stress management is minimal, mantra meditation may be an especially useful strategy.

3. *Meditative skills*. Smith (1985, 1986) postulates three meditative skills: *focusing* (the ability to attend to a restricted stimulus for an extended period), *letting be* (the ability to put aside unnecessary goal-directed analytical activity), and *receptivity* (the willingness to tolerate and accept subjective experiences that may be uncertain, unfamiliar, and paradoxical). He suggests a skills-focused approach to meditation, which can both teach such skills in an organized manner and help select prospective meditators on the basis of whether or not they already possess some components of these skills. This is a promising approach, but one that awaits a test battery to measure "meditation readiness" before it can be applied in the clinic. Informal assessment of an individual as to whether or not he or she may possess meditative skills is a possibility, however.

SIDE EFFECTS AND CONTRAINDICATIONS

Side Effects of Tension Release

Like all techniques used to effect personality change, mantra meditation has its limitations. One of these is the stress-release component of meditation, which must be understood if this technique is to be used effectively. Particularly in the new meditator, physiological and/or psychological symptoms of a temporary nature may appear during or following any form of meditation. These have been described elsewhere (Carrington, 1977, 1998) and appear to be caused by the release of deep-seated nonverbal tensions. Their occurrence can be therapeutically useful, provided that the therapist is trained in handling them properly; however, too rapid a release of tension during or following meditation can cause difficulties and discouragement in a new meditator and may result in a client's backing off from meditation or even abandoning the practice altogether. For this reason, careful adjustments of meditation time and other key aspects of the technique must be made if this modality is to be used successfully. Such adjustments can usually eliminate problems of tension release in short order; accordingly, adjustment of the meditation to suit each client's individual needs is central to such modern forms of mantra meditation as CSM.

Rapid Behavior Change

Another potential problem in the use of meditation stems from the rapidity with which certain alterations in behavior may occur. Some of these changes may be incompatible with the lifestyle or defensive system of the client. Should positive behavioral change occur before the groundwork for it has been laid (i.e., before the client's value system has readjusted through therapy), an impasse can occur, which must then be resolved in one of two ways: (1) The pathological value system must be altered to incorporate the new attitude brought about by the meditation, or (2) the practice of meditation must be abandoned. If the meditator facing such an impasse has recourse to psychotherapy to work through the difficulties involved, this usually allows the individual to continue productively with meditation and make use of it to effect a basic change in lifestyle.

Some of the ways in which meditation-related behavioral changes may threaten a client's pathological lifestyle are as follows:

1. Meditation may foster a form of self-assertion that conflicts with an already established neurotic "solution" of being overly self-effacing. The tendency toward self-effacement must then be modified before meditation can be accepted into the person's life as a permanent and beneficial practice.
2. Meditation tends to bring about feelings of well-being and optimism, which may threaten the playing out of a depressive role that may have served an important function in the client's psychic economy.
3. The deeply pleasurable feelings that can accompany or follow a meditation session may cause anxiety. For example, clients with masturbation guilt may unconsciously equate meditation (an experience in which one is alone and gives oneself pleasure) to masturbation and thus may characterize it as a "forbidden" activity.
4. Meditation can result in an easing of life pace, which may threaten to alter a fast-paced, high-pressured lifestyle that is used neurotically as a defense or in the service of drives for power, achievement, or control. Clients who sense that this may happen may refuse to start meditating in the first place—or, if they

start, may quickly discontinue the practice—unless these personality problems are addressed.

5. A client may develop negative reactions to the meditation process or to a meditational object of focus, such as a mantra. Some individuals initially view meditation as being almost "magical." When they are inevitably forced to recognize that the technique varies in its effectiveness according to external circumstances or according to their own moods or states of health, they may then become angry and quit the practice unless the clinician can help them modify their irrational demands.

Fortunately, such complications as these do not occur in all meditating patients. Often, meditation assists the course of therapy in such a straightforward fashion that there is little necessity to be overly concerned with the client's reaction to it.

Contraindication for Clients with Excessive Need to Control

Clients who fear loss of control may equate meditation with hypnosis or forms of mind control and may thus be wary of learning the technique. If they do learn it, they may experience the meditation session as a form of punishment, a surrender, a loss of dominance, or a threat to a need on their part to manipulate others. These people may soon discontinue the practice unless therapeutic intervention brings about a sufficient change in attitude. Such overly controlling clients may prefer a more "objective" technique that they can manage through conscious effort (e.g., by tensing and relaxing muscles, dealing with biofeedback hardware). The response of a client to the clinician's initial suggestion that he or she learn meditation will often be the deciding factor: Those clients who fear loss of control during meditation will usually indicate this and will respond negatively to the suggestion that they learn the technique.

Cautions

1. An occasional person may be hypersensitive to meditation, so that he or she needs much shorter sessions than the average. Such a person may not be able to tolerate the usual 15- to 20-minute sessions prescribed in many forms of modern mantra meditation and may require drastic reductions in meditation time before benefiting from the technique. Most problems of this sort can be successfully overcome by adjusting the meditation time to suit the individual's needs.

2. Overmeditation can be dangerous. On the theory that "If one pill makes me feel better, taking the whole bottle should make me feel exceptionally well," some clients may, on their own, decide to meditate 3 or 4 hours (or more) per day instead of the prescribed 15–20 minutes only once or twice a day. Like a tonic or medicine, meditation may cease to have beneficial effects if it is taken in too-heavy doses and may become detrimental instead. Release of emotional material that is difficult to handle may occur with prolonged meditation; in a person with an adverse psychiatric history, the commencement of meditation training has been known to precipitate psychotic episodes (Carrington, 1977; Glueck & Stroebel, 1975; Lazarus, 1976; Sethi & Bhargava, 2003). Although it is not certain that overmeditation will lead to such serious results in relatively stable people, it is probably unwise for any person to enter into prolonged meditation sessions if they are not in special settings (such as a retreat) where careful supervision is available.

3. The fact that meditation may be a tonic and facilitator when taken in short, well-spaced dosages but may have an antitherapeutic effect when taken in unduly prolonged sessions is thus essential to consider when reviewing a psychiatric case history in which any form of meditation has previously been practiced by a client. Certain forms of meditation currently promoted by cults demand up to 4 hours of daily meditation from their followers—an important factor to note when assessing some of the "brainwashing" effects frequently reported by ex-members of these cults.

4. Meditation may enhance the action of certain drugs in some clients. Requirements for antianxiety and antidepressive drugs, as well as antihypertensive and thyroid-regulating medications, should therefore be monitored in patients who are practicing meditation. Sometimes the continued practice of meditation may permit a desirable low-dosage treatment using such drugs over more prolonged periods and occasionally may permit the discontinuance of drug therapy altogether.

To avoid such difficulties as these, meditation should be practiced in moderation, with the meditator following instructions in a reliable meditation training program. Full training in the management and adjustment of the technique, not just instruction in how to meditate, is essential for the effective clinical use of meditation.

THE METHOD

Optimal use of mantra meditation in a clinical setting depends on teaching the client to manage the technique successfully—a consideration that can all too easily be overlooked. Unless routine problems that arise during the practice of meditation are handled, however, the likelihood of obtaining satisfactory compliance is poor. On the other hand, if the technique is regulated to meet the needs of the particular client, adherence is often excellent.

It is doubtful whether meditation can ever be taught effectively through written instructions, as correct learning of the technique relies on the communication of the meditative mood—a subtle atmosphere of tranquility best transferred through nuances of voice and tonal quality. Meditation can be taught successfully by digital means, however, provided that those means effectively convey this elusive meditative mood (i.e., that they are not "cold" or "mechanical" in nature) and that the recorded teaching system is sufficiently detailed in terms of the information it conveys, so that the trainee is instructed in handling minor problems that may arise at first. The following discussion covers some of the ways in which CSM may be introduced to a client. Written handouts for learners with instructions for mantra meditation can be useful adjuncts to live instruction. An example of these instructions is provided in Tables 13.2, 13.3, and 13.4.

Introduction of the Method

Clinicians are in a strategic position to introduce to their clients the idea of learning CSM. This is best done by referring to specific difficulties or symptoms that a client has previously identified. Simply mentioning research that suggests that meditation may be useful for these problems is often all that is needed to motivate the client to learn the technique. To forestall misunderstandings, however, several aspects of the CSM method are useful to mention when the subject of meditation is first introduced.

TABLE 13.2. Handout: Choosing a Mantra

My mantra choice is: ____

The following is a list of Sanskrit and other sounds, which are considered soothing. Please read over the list, pronouncing each sound to yourself several times (either mentally or out loud in a soft voice) and then place a ”1” next to the one you like best, “2” next to the one you like next, and “3” next to the one you like third.

If you prefer to make up your own mantra, see below.

Note: “a” is usually pronounced “ah,” but do whatever pleases you.

____ah-nam	____ma-ha-yam
____vahn-day	____vis-ta
____shi-rim	____at-man
____sat-yam	____ra-ma
____ha-sam	____shan-ti
____in-dra	____ra-yim
____see-tah-ram	____ma-na
____ta-sam	____shee-vo-humm

If you decide to create *your own mantra,* be sure to keep the following in mind:

1. It should be soothing and resonant. Avoid all sharp, staccato words.
2. It should not be emotionally stimulating. Do not use names of people or other words that have special “loaded” meanings to you.
3. “Meaningless” sounds are particularly useful because they have fewer associations and seem to have a more “mysterious” quality, but they are not absolutely essential. One person, for example, successfully used the self-created phrase “inner peace” as a mantra.

When you have selected your mantra, write it down at the top of the page where space is provided. Later you may transfer it to your reminder schedule for future reference. If you wish to tell your mantra to a close friend or relative, do so in a respectful, serious manner and ask them to keep it in confidence. We will explain this further after you have received further instruction.

TABLE 13.3. Handout: Preparation for a Meditation Lesson

1. The type of meditation you will be learning is a Westernized version of an ancient Indian practice known as *mantra* meditation, where a soothing sound (a mantra) is repeated mentally for 20 minutes under quiet conditions. It is easy to learn and does not require conscious effort or concentration.
2. IMPORTANT: Do not take any nonprescription drugs (such as marijuana) or any alcohol for 24 hours prior to your instruction in meditation. Learning meditation requires a special degree of alertness.
3. Do not eat and do not drink any caffeine beverage (such as coffee, tea, or cola), for 1 hour prior to your meditation instruction. Meditation should always be practiced on an empty stomach, and stimulants (such as coffee) should be regularly avoided beforehand.
4. Among the many benefits that some people report from regular meditation with this method are a lessening of a sense of urgency about life in general, an easing of tensions, decreased anxiety, greater energy, and more restful sleep. In many ways, meditating can be thought of as giving yourself a “vacation” twice a day. It is often described as being like lying on a quiet beach with the sound of the surf lulling you into a peaceful state or like resting on a boat gently rocking in a calm sea. Meditation Western-style is a time when you are “nice” to yourself. Most of us who practice it have found it a very enjoyable addition to our lives. We hope you will too.

TABLE 13.4. Handout: Points to Remember about Meditation

- You will be meditating twice daily, for your selected number of minutes.
- Ordinarily you will not meditate on a full stomach, although it is fine to meditate after a very light meal (without caffeine).
- An ideal time for your first meditation is in the morning before breakfast and before morning coffee (caffeine, being a stimulant, may counteract the effects of meditation).
- First, get out of bed and sit in a chair (or on the floor if preferred). It may be useful to walk about a bit and wash up so as to be thoroughly awakened before commencing meditation, but no *strenuous* physical exercises before meditating.
- If you miss doing meditation before breakfast, meditate either after finishing a very light breakfast, at midmorning, or just before lunch. Whichever time you choose, make sure to space your two daily meditations so that they are separated in time by at least 4 hours.
- An ideal time for your second meditation is in the late afternoon before dinner if possible. If this is not possible, wait about an hour after eating before meditating (never meditate on a full stomach).
- Unless you plan to stay up, you may not want to meditate late in the evening since meditation increases available energy in some people, and about 20% of people find they do not feel like sleeping after meditation. On the other hand, about 80% of people find that they go to sleep easily and restfully directly after meditation even if the hour is late. You can find out, by trial and error, which way you react to a late-evening meditation.
- Your meditation should be practiced in a reasonably quiet place where you are not too likely to be disturbed for at least 20 minutes. If you are disturbed (e.g., by a telephone ringing), remember to play for time. Take 20 to 30 seconds to come out of meditation before dealing with the interruption, giving yourself ample time to "surface." After disposing of the interruption, then return to meditation for the time that is left in it (not starting over again from the beginning but continuing where you left off).
- Falling asleep during meditation occasionally happens. When it does, it shows that you need sleep more than meditation at that particular moment. Never fight sleep. If it comes, just lie down, sleep, and when you awaken, return to the meditation and complete the approximate time you need to in order to have done it for 20 minutes' total time.
- Meditation may be timed by occasionally peeking at a watch or clock with one eye. (This is much less apt to arouse you from meditation than using both eyes.) Many people learn to sense when their number of minutes is up after they have been practicing meditation for several weeks.
- When sitting down to meditate, the first 30 seconds sit quietly with eyes closed, so that you have a chance to "settle down" before commencing to think the mantra.
- After finishing meditation (and before opening your eyes), it is most important that you spend 2 minutes in silence (without thinking the mantra). This quiet interval will help you to carry over the effects of the meditation into your daily life and to dissolve stresses that may have surfaced during meditation.
- When meditating, *do not* "force" the mantra or "clutch" at it. If the mantra appears or disappears, if it gets loud or soft, if it is accompanied by many thoughts or by no thoughts—all these things make no difference. The meditation will be just as beneficial with much thinking of the mantra as with little thinking of the mantra, with many thoughts as with few. Simply take it as it comes.

Note. If you have any questions about your experiences during your home meditation in the next few days, please feel free to call me on my office phone at ____________. Leave a message if I do not pick up.

The clinician will want to emphasize that this form of meditation is strictly noncultic in nature. It does not rest on a religious theoretical foundation, although religion can certainly be combined with meditation and often is in various forms of meditation. Clients with religious convictions will not want their beliefs violated by competitive belief systems and can be relieved to learn that CSM is a "scientifically developed" form of meditation. In addition, clients who are uncomfortable with seemingly unconventional interventions will also benefit from being reassured about the noncultic (not religious) nature of the method. The clinician will also want to emphasize that the technique is easily learned, because one of the most prevalent misconceptions about meditation is the notion that it requires intense mental concentration. Most people are reassured by the knowledge that a modern mantra meditation technique such as CSM does not require forced concentration at all but actually proceeds automatically once it has been mastered. The clinician should also routinely check on the client's knowledge about and/or previous experience with meditation in order to clear up any further questions about the method.

The preliminary discussion between therapist and client is typically brief, but certain clients may need to be introduced to meditation in a more planned manner. "Type A" clients, for example, may resist learning meditation (or any other relaxation technique) because the idea of slowing down threatens their lifestyle, which is often hectic and high-pressured. When a clinician is recommending CSM to a Type A person, therefore, a useful strategy is to indicate that the time that this person will take out of his or her day for meditation practice is likely to result in increased efficiency. Much research suggests that this is so, and Type A individuals are typically achievement-oriented.

Type A, or extremely active, people can also be helped to accept meditation by being informed that they can break up their practice into a series of what have been termed *mini-meditations* (Carrington, 1978, 1998). These are short meditations of 2 or 3 minutes (sometimes only 30 seconds) in duration, which can be scattered throughout the day. Frequent mini-meditations may be much more acceptable to an impatient, driven sort of person than longer periods of meditation may be (although these can be used, too), and they have the advantage of helping the client reduce transient elevations in stress levels as these occur.

A final strategy useful when recommending meditation to Type A or exceedingly active persons can be to inform them that they can use CSM while simultaneously engaged in some solitary sport that they may already practice and enjoy. Meditation can be successfully combined with solitary, repetitive physical activities such as jogging, walking, bicycling, or swimming, and this practice may be a salutary one. Benson, Dryer, and Hartley (1978) have shown, for example, that repeating a mantra mentally while exercising on a stationary bicycle can lead to increased cardiovascular efficiency.

The Physical Environment and Equipment

During the instruction session, the trainee repeats his or her mantra out loud in imitation of the instructor and meditates silently in harmony with the instructor. Potential problems involved in meditation practice are explained, and the trainee is instructed to practice independently during the following week, adjusting the technique to personal needs. The clinician may assist by making clinically relevant suggestions.

Most clients learn CSM in their homes (or hospital or dormitory rooms) and make their own arrangements for a suitable instruction environment. When clients are taught on the premises of the clinician (usually so that the latter can advise immediately on adjusting the technique), a quiet, uncluttered room in which the client can be alone while learning should be made available. This room typically contains a comfortable straight-backed chair and some visually pleasant object, such as a plant or vase, on which the trainee can gaze when entering and exiting from meditation. The arrangements are simple, but they should be carefully observed for maximum effect.

Therapist–Client Relationship

There is a standardized procedure for teaching CSM. People who have successfully used other meditation techniques often make excellent instructors of CSM by using such a protocol because almost all meditation techniques have a number of points in common. Even those trained in some of the more disciplined forms of meditation have been able to teach the permissive approach of CSM after first learning the technique themselves and practicing it for several months prior to teaching it. Personal experience with CSM is essential for the prospective instructor to understand the basic permissiveness of the technique.

Procedure

Trainees will first select a mantra from a list of 16 mantras in the workbook. They are instructed to choose the one that sounds most pleasant and soothing to them or to make up a mantra according to simple instructions. The mantras used in this method are resonant sounds (often ending in the nasal consonants *m* or *n*) that have no meaning in the English language but that, in pretesting, have been shown to have a calming effect on many people. Such sounds as *ahnam, shi-rim,* and *ra-mah* are among those used. Long vowels and consonants tend to help produce a relaxing effect. The mantra should not have a meaning in any language known by the trainee, so that it becomes a conditional stimulus associated with a relaxed meditative state. After the trainee has selected a mantra, training is conducted in a peaceful setting removed from any disturbances that may detract from the meditative mood. The instructor walks quietly, speaks in low tones, and typically conveys by his or her behavior a respect for the occasion of learning meditation. An instruction sheet with a list of suggested mantras can be given to the trainee, although the trainee should feel free to make up an individual mantra. See Table 13.2 earlier in the chapter.

When teaching meditation, the instructor repeats the trainee's mantra out loud in a rhythmical manner to demonstrate how this is done. The trainee then repeats the mantra in unison with the instructor, and finally alone. He or she is next asked to "whisper it" and then simply to "think it to yourself" silently, with eyes closed. Instructor and trainee then meditate together for a period of 10 minutes, after which the trainee remains seated for a minute or two with eyes closed, allowing the mind to return to "everyday thoughts." The trainee is then asked to open his or her eyes very slowly. At this point the instructor answers any questions the trainee may have about the technique, corrects any misconceptions he or she may have, and then leaves the room so that the trainee can meditate alone for a stated period of time (usually 20 minutes). The experience of meditating on one's own is included in order to wean the trainee as soon as possible from dependency on the instructor's presence when meditating.

In a postinstruction interview, procedures for a home meditation practice are clarified, and instructions are given for the trainee's meditation program for the following week. The trainee is then apprised of possible side effects of tension release (Carrington, 1998) and is taught how to handle these should they occur.

Individual follow-up interviews are later held at intervals, or group meetings are scheduled in which new meditators can gather to share meditation experiences, meditate in a group, and pick up new pointers on handling any problems that may arise in their practice. These trainees then learn to adjust their techniques to suit their own individual needs and lifestyles. Close clinical supervision of the meditation practice is strongly advised. A careful follow-up program ensures much greater participation in a continued program of meditation.

Resistance, Adherence, and Maintenance of Behavior Change

Problems of resistance were discussed in the earlier section, "Side Effects and Contraindications." Adherence and maintenance of behavior change are now considered.

Adherence

Researchers have found long-term use of the modern forms of mantra meditation to be about 50% among adults in a typical community, with about half of those who learn to meditate discontinuing the practice within 3 years of having learned it and an even larger number cutting down to once instead of twice a day or to only occasional use of the practice (Carrington, 1998). Several problems emerge when we try to evaluate the existing adherence figures, however. The trend has been to define *adherence* as "regular daily practice" of the meditation technique in question, a viewpoint undoubtedly influenced by the firm conviction of TM's founder, Maharishi Mahesh Yogi, that twice-daily practice is necessary in order to obtain benefits from meditation. Some recent findings cast doubt on the necessity of daily meditation for all people, however, and suggest that the degree of adherence that is necessary to produce benefits may in many cases be an individual matter.

When we (Carrington et al., 1980) studied the use of two mantra meditation techniques (CSM and ROM) in a working population self-selected for symptoms of stress, we found that after 5½ months of practicing meditation, these participants showed highly significant reductions in symptoms of stress as measured by the SCL-90-R in comparison with controls. However, when the groups were broken down into (1) *frequent practicers* (individuals who practiced their technique several times a week or more), (2) *occasional practicers* (individuals who practiced it once a week or less), and (3) *stopped practicers* (individuals who no longer practiced their technique), the results were unexpected. Although SCL-90-R improvement scores for stopped practicers and controls did not differ (as might be expected), no differences in degree of symptom improvement were found between frequent and occasional practicers when the scores for these two groups were compared, contrary to our expectation. Nevertheless, when frequent and occasional practicers were collapsed into a single "practicers" group and stopped practicers and controls were placed into a single "nonpracticers" group, the difference in degree of symptom reduction between these two groups was highly significant. As long as participants practiced at all, they were likely to show improvement in symptoms of stress. When they did not practice, they were unlikely to improve more than controls.

The finding in this study—that *frequent* practice appears unnecessary to produce symptomatic improvement—disagrees with those in several studies using the TM technique. The latter studies have reported positive effects of frequent (as opposed to occasional) practice of meditation on neuroticism (Ross, 1978; Tjoa, 1978; Williams, Francis, & Durham, 1976), trait anxiety (Davies, 1978), autonomic instability (Orme-Johnson, Kiehlbauch, Moore, & Bristol, 1978), intelligence test scores (Tjoa, 1975), and measures of self-actualization (Ross, 1978). However, our findings (Carrington et al., 1980) are in agreement with research that has reported no differences between frequent and occasional practicers with respect to anxiety reduction (Lazar, Farwell, & Farrow, 1978; Ross, 1978; Zuroff & Schwarz, 1978).

It should be noted that there were several differences between our study with the employee group and those studies using the TM technique that did show effects for frequency of practice. All but one of the TM investigations were conducted with participants who had signed up to learn TM at TM training centers. These participants were not selected for high initial stress levels (although in some cases perceived stress may have played a role in their decision to learn meditation). It is therefore unlikely that they were under the same degree of stress as the employees in the Carrington et al. (1980) study, who had been self-selected for this variable and whose initial SCL-90-R scores fell at the edge of the clinical range. Possibly, when stress symptoms approach clinical levels, even a moderate amount of meditation, or the use of meditation when needed, is sufficient to achieve sharp reductions in symptomatology. When the initial stress levels are close to the norm, however, it may be necessary to practice meditation more frequently in order to reduce symptoms to a still lower level.

Another factor differentiating the employee study from the TM studies is that teachers of the TM technique prohibit the use of mini-meditations, which they consider harmful to proper meditation practice. In the employee study, however, strong emphasis was laid on the use of mini-meditations in addition to full meditation sessions, and the effectiveness of this teaching was demonstrated by the fact that at the end of 54 months, 88% of the employees who had learned meditation reported that they were using mini-meditations. Mini-meditations may therefore have exerted a leveling effect, causing a blurring of expected distinctions between frequent and occasional practicers.

Although frequency of practice could not predict stress reduction in the employee study, this should not be taken to mean that frequent practice of a meditation technique is not valuable. For *some* participants in the study, regular daily practice may have been necessary to acquire noticeable benefits. Realizing this, such people may have developed the habit of meditating frequently. Other participants, however, may have found it unnecessary to practice their technique more than a few times a week to obtain noticeable symptom improvement. It is also not presently known whether frequent practice will in time produce beneficial changes in some practicers who do not report benefits from frequent practice during the first 6 months, whether physiological (as opposed to psychological) measures respond to occasional practice as well as they do to frequent practice, or whether effective control of a maladaptive form of behavior (e.g., drug addiction) requires the frequent practice of meditation in order to alter this behavior. Research findings such as those described herein, coupled with clinical reports on the benefits of using meditation on a contingency basis (and/or of using frequent mini-meditations), suggest the wisdom of reconsidering our present criteria for adherence. This might serve to lessen some of the current confusion in the field. For example, in the Carrington et al. (1980) study, when "practicing at all" (whether frequent or occasional) was used as the criterion for adherence, 81% of the participants using CSM and 76% of the participants using ROM were still practicing their respective techniques at the end of 5½ months. However, when "frequent practice only" was used as the criterion for adherence, only 50% of the CSM participants and 30% of the ROM participants were practicing their techniques by the end of this time.

Spontaneous comments offered by participants in this study (on a postexperimental questionnaire) may offer some clues. When these comments were examined in relation to frequency of practice, analysis revealed that more occasional practicers than frequent practicers were using their techniques for strategic purposes (i.e., as needed); that more frequent practicers than occasional practicers made strong positive statements about the benefits derived from their technique; and that only occasional practicers qualified their statements about their benefits (e.g., "Under extreme pressure in my department, I don't feel as tensed up, but don't find meditation as beneficial as I had hoped" or "I think there has been some possible effect"). The tentative nature of the statements of many of the occasional practicers attest to the "in-between" quality of their evaluative statements, as opposed to the certainty that characterized those of the frequent practicers.

We might summarize the findings to date, then, by saying that the effects of frequency of practice are only partially known. Frequency may not play a major role in symptom reduction for certain patients (although it may for others), but it may be positively related to perceived benefits in other areas, such as personal growth and job performance, and it seems clearly related to the degree of enthusiasm that participants express for their technique. In a practical sense, therefore, it would seem wise to encourage regularity of practice for a client whenever possible, without being unduly alarmed if that client should shift from meditating regularly to using the technique for strategic purposes only or to relying mainly on mini-meditations. The deciding factor should be the degree of benefit that the client is deriving from the practice. If this factor remains satisfactory in the estimation of the clinician, then even if the client meditates only occasionally or uses only mini-meditations, his or her decision to employ the technique in this manner should be supported.

Also relevant to adherence is the manner in which meditators stop practicing. In the Carrington et al. (1980) study, the timetable for quitting in the stopped practicers was revealing. The practice of meditation appears to have stabilized markedly within the first 3 months. One-third of the stopped practicers reported that they had abandoned their technique within the first 2 weeks after having learned it; another 27% reported that they abandoned it between 2 and 6 weeks; and still another 37% reported having abandoned it between 6 weeks and 3 months. Only one participant had abandoned the technique between 3 and 5½ months (during the final 2½ months of the study). It would seem, therefore, that during the first 3 months of their practice, a more or less permanent commitment was made by these trainees to continue their meditation practice. Thereafter, although a trainee might shift from frequent to occasional practice (or back again), he or she was extremely unlikely to stop practicing entirely. This timetable of attrition strongly suggests that once meditation has been successfully adopted and practiced for several months, it may become a permanent coping strategy that can then be called on by a trainee when he or she has need for it—in short, that the *strategic* use of meditation is not likely to be abandoned.

It has also been observed that meditators may stop practicing meditation temporarily for a variety of reasons. These "vacations" from meditation appear to be a normal part of the practice for many people and are not evidence of nonadherence. It is important, therefore, that the clinician not label a cessation of meditation practice as "dropping out" unless such a fact has been proven correct. The client should be helped instead to understand that such "vacations" can be normal occurrences and that meditators frequently return to their regular practice later with renewed enthusiasm. In CSM, use of a special renewal-of-practice audio by the client is recommended as a useful means of reinstating the meditation practice after having taken a break from it.

The clinician should also be aware that even if a client eventually abandons his or her technique, this is not necessarily a negative finding. Reports from a corporate program using CSM at New York Telephone (G. H. Collings, personal communication, 1982) suggest that after an extended period (e.g., 1 year or more) of successful meditation practice, some people may no longer need to practice meditation on a formal basis, because its benefits have been incorporated into their lifestyles. One telephone company employee reported that he no longer needed to meditate because he had begun to spend his lunch hour eating by the fountain in the courtyard where he worked, just watching the water rise and fall. He described this as so peaceful that afterward "I feel better for the rest of the day." He typically spent 20 minutes watching the fountain, but he said that before he learned to meditate (approximately a year earlier), he would never have thought of such a thing, because then he was always in a hurry, even when he had a lunch break. When asked whether his experience of gazing into the fountain had any features in common with meditation, he replied that, although this had not occurred to him before, actually the two processes seemed exactly the same to him, except that he didn't use his mantra when he watched the fountain.

Similar reports from other long-term meditators, collected at New York Telephone, suggest that formal meditation may be phased out by some clients as a "meditative approach" to life is phased in. Such people appear to have substituted their own meditation equivalents for formal meditation sessions. This is by no means the case with all meditators, however. A sizable number of people need to continue with the formal practice of meditation indefinitely in order to maintain the beneficial changes brought about using the technique.

Maintenance of Behavioral Change

As noted, maintenance of behavioral change is large and may be closely linked with adherence in some, but not necessarily in all, instances. However, cases have occasionally been reported in which, after several years of mantra meditation, meditators have ceased to notice any more benefits accruing from their practice. Because these have all been anecdotal reports, it is unclear whether the people involved were no longer benefiting from their meditation practice or whether benefits were still occurring but were not perceived because the meditators' tension levels had been reduced for so long a time. In clinical practice, an empirical test can be applied in the event of reports of diminished benefits. The client can be asked to stop practicing meditation for a stated period; if cessation of meditation brings no change in the clinical condition, or if it results in a beneficial change, then meditation (at least as originally learned and practiced) may have ceased its usefulness for the client. Substitution of another variant of meditation or another approach described in this volume, if this is desired, is sometimes useful at this point and can result in a revival of beneficial effects in some cases. The reasons for these occasional apparent habituations to the method are unclear, as are the causes of reports of certain meditators' having experienced adverse effects after having practiced their techniques for prolonged periods of time. Although the latter problem can usually be brought under control by proper readjustment of the meditation routine, discontinuance of the practice is in order if it cannot.

CASE EXAMPLE

Training in meditation was recommended for a middle-aged female client because her chronic tension headaches had consistently resisted all other forms of intervention, even though her other physical symptoms (e.g., gastric ulcer and colitis) had abated with psychotherapy. After she commenced meditation, this client's headaches worsened for about a week (temporary symptom acceleration is not unusual following commencement of meditation) and then abruptly disappeared; the patient remained entirely free of headaches for 4 months for the first time in many years.

During this period, however, she noticed personality changes that disturbed her and that she attributed to meditation. Formerly self-sacrificing and playing the role of a "martyr" to her children, husband, and parents, she now began to find herself increasingly aware of her own rights and impelled to stand up for them, sometimes so forcefully that it alarmed her as well as her family. Although she was apparently effective in this new self-assertion (her adolescent sons began to treat her more gently, making far fewer scathing comments), other members of her family commented that she was no longer the "sweet person" that she used to be, and the client soon complained that "meditation is making me a hateful person."

At the same time, this client also noticed that she was no longer talking compulsively—a change for which she received favorable comments from others but that bothered her because she was now able to sense the social uneasiness that had been hidden beneath her compulsive chatter. She related this tendency to remain quieter in social situations directly to meditation, as it was more apt to occur soon after a meditation session. Unable to assimilate the personality changes she was noticing, the client stopped meditating, despite the fact that her tension headaches then returned.

It was necessary at this point in therapy to trace the origins of this client's need to be self-effacing before she could consider reinstating the practice of meditation. In doing so, it was discovered that her competition with an older sister was at the root of much of her difficulty in this respect. This sister had been considered a "saint" by their parents, whereas the client had always been considered a troublesome, irritating child. During her childhood she had despaired at this state of affairs; however, in her adolescence, she developed an intense compulsion to become more "saintly" than her exalted sister, although this often meant total sacrifice of her own wishes or needs to those of others. Even the simple pleasure of a normal meditation session seemed to this client to be a self-indulgence out of character for so "self-sacrificing" a person.

After working on these problems in therapy, and after some role playing with respect to positive forms of self-assertion, the client finally agreed to resume daily meditation. It was soon discovered, however, that the meditative process was once again pushing her toward self-assertion at too rapid a rate for her to handle. The therapist then suggested that she reduce her meditation to once weekly. Her meditation session was to take place only at the start of each therapy session, and the therapist was to meditate with the client at these times, giving tacit support to the client's right to independence and self-assertion and serving as a role model in terms of acceptance of a meditation practice in one's life.

These weekly joint meditative sessions proved extremely productive; the client described her sessions with her therapist as being "deeply restful," pleasurable, and constructive. Because her emotional responses to each meditation session could be promptly dealt with in the discussion that followed, guilt over self-indulgence was prevented. With this approach, the client's headaches again disappeared, and she began to experience personality changes typical of regular daily meditators, such as marked enrichment of a previously impoverished fantasy life. She repeatedly stated, however, that this weekly meditation session was all she could "take" of meditation at one time without feeling "pounded" by it. In this moderate dose, the client appeared well able to assimilate the changes in self-concept brought about by meditation, and the client–therapist relationship was used to enhance the effectiveness of the meditation through the joint meditation sessions.

As this case illustrates, the use of psychotherapy along with meditation can be crucial in certain instances to the success of the technique. It is important to note that in most cases, however, meditation contributes to the patient's therapeutic progress with few, if any, complications.

CURRENT CONTRIBUTIONS OF MANTRA MEDITATION TO CLINICAL PRACTICE

What was already known about mantra meditation's potential role in stress management has been well supported by the research that has been conducted since the last edition of this book. Although recent research has yielded less in the way of such basic discoveries as were initially reported in the early 1970s, the findings remain decidedly positive.

Since mantra meditation is well within the reach of the general public, it has grown in popularity and familiarity for the average person in recent years. The era of surprises is over now, and we know that it works for many health issues, that it can be extremely effective for emotional distress, and that it can be administered with little cost or difficulty.

Several meta-analyses of the data have been reported (Ooi, Giovino, & Pak, 2017; Park & Han, 2017; Shi et al., 2017), and these reviews generally confirm the earlier findings that mantra meditation is of decided value for health and emotional stability. These studies frequently call for more rigor in the design of studies and ask for larger samples. This is sound advice, except for the fact that funding for research on meditation is not easy to come by due to its seemingly esoteric nature, which still marks it as somewhat outside of the commonly accepted parameters. For this reason, research funds for more elaborate studies may not be readily obtainable. Hopefully, adequate support will bring forth such larger studies in the future.

It seems noteworthy that, although no truly unique realm of effectiveness has been identified in the past few years, no findings have supported the conclusion that this is *not* an important potential treatment for the average person, and basically the previous positive findings remain unchallenged. A few subareas are now being studied that were relatively neglected before. One of these is the effectiveness of mantra meditation for posttraumatic stress disorder (PTSD). PTSD is a notoriously difficult area to study due to the difficulties in pinning down the diagnosis and acquiring a suitable population for study, for follow-up, and for obtaining reliable outcome measures. Despite such difficulties, however, the general conclusion that mantra meditation can be highly effective in the treatment of many *components* of PTSD has been repeatedly confirmed (Kang et al., 2018; Metcalf et al, 2016; Cushing & Braun, 2018; Lang et al., 2012; Park & Han, 2017; Harne & Hiwale, 2018), and anxiety remains a major area in which mantra meditation seems to offer considerable clinical help (Cooney Roxbury, 2018; Travis et al., 2018).

One of the difficulties that somewhat hamper research on mantra meditation is the diversity of methods used to bring about the meditative state, which can result in comparing techniques whose dissimilarities make these methods difficult to study. This has resulted in some of the studies perhaps comparing apples to oranges, or presenting similar awkwardness.

Commendable for its consistency in comparing identical conditions in its study of mantra meditation has been the research conducted under the auspices of the

Transcendental Meditation™ organization. Researchers using TM as a subject of study have the advantage of a rigorous and strictly defined procedure to examine, one that can be reexamined with reliability for various purposes. Accordingly, the TM researchers observe rigorous scientific standards when conducting their research so that, when reviewing their studies, we can know exactly what has been studied and to exactly what it has been compared.

Interestingly, the careful standards of the TM investigators have not only resulted in excellent comparability between their various studies, but also (rather surprisingly, as it is an organization that might be said to have a "vested interest" in positive outcomes) they have done some of the few investigations as yet conducted into the nature of the basic meditative state itself.

The TM studies go well beyond simple attempts to show that mantra meditation works to reduce stress. Some outstanding examples of the TM organization-sponsored current research are studies by Infante and colleagues (2014) and Oman and Bormann (2015). Early research has been reviewed in a volume of collected research papers (Kanellakos, 1974).

In summary, the fact that mantra meditation can be an effective stress management tool in clinical practice has now been replicated in study following study, and the method is showing itself to be an ever more attractive alternative for clinicians in the mental health field. This conclusion is now even more solidly research-based than formerly, and we can hopefully look forward to even more extensive research evidence of its clinical effectiveness in the decades to come.

SUMMARY

A note of caution seems appropriate at this point. Although it is clearly desirable to be clinically oriented in one's approach to meditation training, this need not be defined as making the instruction of the technique impersonal in nature. The clinician should be aware that in his or her zeal for objectivity, he or she could inadvertently "throw the baby out with the bathwater." The attitude of quiet respect and the peaceful surroundings that have traditionally accompanied the teaching of meditation have something important to teach us. They cannot, it seems, be lightly dispensed with without losing something essential to the meditative process. Properly taught, meditation can be a compelling subjective experience. To hand a client a sheet of instructions and tell him or her to "go home and meditate" is therefore likely to result in a serious decrease in the importance the client will attach to learning the meditation, as well as to deprive him or her of a role model to demonstrate the subtle meditative mood.

Following the adage "easy come, easy go," clients who are taught meditation in an abbreviated fashion and without attention to the conveying of the delicate mood inherent in this practice are apt to treat meditation casually and may soon discontinue its practice. When field-testing versions of CSM instructions, I (P.C.) discovered, for example, that clients' adherence increased in direct proportion to the inclusion of informal, "personal," mood-setting recordings. Similarly, when giving personal instruction in meditation, clinicians are advised to give careful attention to the setting and the mood that accompany the teaching of meditation. The instruction need not reflect a particular belief system, but it should be pleasant, peaceful, and in some sense special in nature. Learning meditation is an important moment in an individual's life. If it is treated as such, the entire practice takes on a deeper meaning.

A somewhat related issue is the tendency of some clinicians to view meditation as so "simple" that it can be taught in one session merely by imparting the technique itself and that the client can then be left to his or her own devices. As Smith (1987) has pointed out, many therapists and researchers tend to use truncated versions, or "analogues," of authentic meditation training on the assumption that these are equivalent to the full training. In fact, the analogues merely supply components of meditation isolated from their context. For proper training in meditation, *context* is extremely important. Although the actual techniques of meditation can be taught in a single, carefully structured session, this does not mean that a successful practice of meditation has been established by doing this. The latter requires that a number of changes be made in the trainee's daily routine, that individual regulation of the technique be provided, and that knowledge of ways to handle problems that may arise in meditation practice be taught. Without full training in the management of meditation, in fact, learning the technique alone can be detrimental in that it may lead a trainee to believe that he or she is not a likely candidate for meditation (because he or she may have run into some problems with its practice), when, in fact, this may not be the case.

The clinician who recommends meditation to a client must, therefore, be careful to supply complete training in all the practical aspects and adjustments of the technique. Only in this manner can the method have the best opportunity for success.

In summary, present experimental, as well as clinical, evidence supports the conclusion that—provided it is imparted with full respect for both its inherent ease and any potential problems involved in learning it—meditation can be a potent tool for personality change—one that greatly extends the clinician's repertoire.

NOTE

Patricia Carrington passed away before updates on this chapter from the previous edition could be completed. Updates on the chapter were primarily done by Paul M. Lehrer.

REFERENCES

Alexander, C. N., Walton, K. G., & Goodman, R. S. (2003). Walpole study of the transcendental meditation program in maximum security prisoners: 1. Cross-sectional differences in development and psychopathology. *Journal of Offender Rehabilitation, 36*(1–4), 97–125.

Allison, J. (1970). Respiratory changes during the practice of transcendental meditation. *Lancet, 7651,* 833–834.

Anklesaria, F. K., & King, M. S. (2003). The transcendental meditation program in the Senegalese penitentiary system. *Journal of Offender Rehabilitation, 36*(1–4), 303–318.

Appelle, S., & Oswald, L. E. (1974). Simple reaction time as a function of alertness and prior mental activity. *Perceptual and Motor Skills, 38,* 1263–1268.

Banquet, J. (1973). Spectral analysis of the EEG in meditation. *Electroencephalography and Clinical Neurophysiology, 35,* 143–151.

Barnes, V. A. (1997). Reduced cardiovascular and all-cause mortality in older African-Americans practicing the transcendental meditation program. *Dissertation Abstracts International, 57*(8B), 4999.

Barnes, V. A., & Orme-Johnson, D. W. (2012). Prevention and treatment of cardiovascular disease in adolescents and adults through the transcendental meditation program: A research review update. *Current Hypertension Reviews, 8*(3), 227–242.

Beck, D., Cosco, H. L., Bukaro, J., Andres, T., Andrews, T., Lino, L., et al. (2017). Efficacy of the mantram repetition program for insomnia in veterans with posttraumatic stress disorder. *Advances in Nursing Science, 40,* E1–E12.

Beiman, I. H., Johnson, S. A., Puente, A. E., Majestic, H. W., & Graham, L. E. (1980). Client characteristics and success in TM. In D. H. Shapiro & R. N. Walsh (Eds.), *The science of meditation* (pp. 283–296). Chicago: Aldine.

Benson, H. (1975). *The relaxation response.* New York: Morrow.

Benson, H. (1977). Systemic hypertension and the relaxation response. *New England Journal of Medicine, 296,* 1152–1156.

Benson, H. (1989). The relaxation response and norepinephrine: A new study illuminates mechanisms. *Australian Journal of Clinical Hypnotherapy and Hypnosis, 10*(2), 91–96.

Benson, H., Alexander, S., & Feldman, C. L. (1975). Decreased premature ventricular contractions through use of the relaxation response in patients with stable ischaemic heart disease. *Lancet, 2,* 380.

Benson, H., Dryer, T., & Hartley, H. L. (1978). Decreased CO_2 consumption during exercise with elicitation of the relaxation response. *Journal of Human Stress, 4,* 38–42.

Benson, H., & Friedman, R. (1985). A rebuttal to the conclusions of David S. Holmes's article: "Meditation and somatic arousal reduction." *American Psychologist, 40*(6), 725–728.

Benson, H., & Stark, M. (1996). *Timeless healing: The power and biology of life.* New York: Scribner.

Benson, H., & Wallace, R. K. (1971). Decreased drug abuse with transcendental meditation: A study of 1,862 subjects. *Congressional Record,* 92nd Cong., 1st Session, Serial No. 92–1.

Benson, H., Wilcher, M., Greenberg, B., Huggins, E., & Ennis, M. (2000). Academic performance among middle-school students after exposure to a relaxation response curriculum. *Journal of Research and Development in Education, 33*(3), 156–165.

Bernardi, N. F., Bordino, M., Bianchi, L., & Bernardi, L. (2017). Acute fall and long-term rise in oxygen saturation in response to meditation. *Psychophysiology, 54*(12), 1951–1966.

Brat, M. (2018). Effects of Zen mindfulness meditation on student counselor's stress attention and self-comparison. *Dissertation Abstracts International, 79* (8-A(E)), 201.

Brown, F. M., Stewart, W. S., & Blodgett, J. I. (1971, November). *EEG kappa rhythms during transcendental meditation and possible threshold changes following.* Paper presented to the Kentucky Academy of Science, Lexington.

Carrington, P. (1977). *Freedom in meditation.* Garden City, NY: Doubleday/Anchor.

Carrington, P. (1978). *Clinically standardized meditation (CSM) professional pack.* Monroe Township, NJ: Pace Educational Systems.

Carrington, P. (1998). *The book of meditation.* Monroe Township, NJ: Pace Educational Systems.

Carrington, P., Collings, G. H., Benson, H., Robinson, H., Wood, L. W., Lehrer, P. M., et al. (1980). The use of meditation–relaxation techniques for the management of stress in a working population. *Journal of Occupational Medicine, 22,* 221–231.

Carrington, P., & Ephron, H. S. (1975). Meditation as an adjunct to psychotherapy. In S. Arieti (Ed.), *New dimensions in psychiatry: A world view.* New York: Wiley.

Cooney Roxbury, A. (2018). Does adding mantra-based meditation training improve the efficacy of the unified protocol for individuals with generalized anxiety disorder? *Dissertation Abstracts International, 78*(8-B(E)).

Cooper, M. J., & Aygen, M. M. (1979). A relaxation technique in the management of hypercholesterolemia. *Journal of Human Stress, 5,* 24–27.

Credidio, S. G. (1982). Comparative effectiveness of patterned biofeedback vs. meditation training on EMG and skin temperature changes. *Behaviour Research and Therapy, 20*(3), 233–241.

Cushing, R. E., & Braun, K. L. (2018). Mind–body therapy for military veterans with posttraumatic stress disorder: A systematic review. *Journal of Alternative and Complementary Medicine, 24,* 106–114.

Das, N. N., & Gastaut, H. (1957). Variations de l'activité électrique du cerveau, du coeur, et des muscles squelletiques au cours de la méditation et de l'extase yogique. [Variations in electrical activity of the brain, the heart, the head, and the skeletal muscles during meditation and yogic ecstasy.] *Electroencephalography and Clinical Neurophysiology, 6*(Suppl.), 211–219.

Davidson, R., & Goleman, D. (1977). The role of attention in meditation and hypnosis: A psychobiological perspective on transformation of consciousness. *International Journal of Clinical and Experimental Hypnosis, 25,* 291–308.

Davidson, R., Goleman, D., & Schwartz, G. (1976). Attentional and affective concomitants of meditation: A cross-sectional study. *Journal of Abnormal Psychology, 85,* 235–238.

Davidson, R., & Schwartz, G. (1976). The psychobiology of relaxation and related states: A multiprocess theory. In D. I. Mostofsky (Ed.), *Behavior control and the modification of* *physiological activity* (pp. 364–392). Englewood Cliffs, NJ: Prentice Hall.

Davies, J. (1978). The transcendental meditation program and progressive relaxation: Comparative effects on trait anxiety and self-actualization. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 449–452). Livingston Manor, NY: Maharishi European Research University Press.

Delmonte, M. M. (1984). Physiological responses during meditation and rest. *Biofeedback and Self-Regulation, 9,* 181–200.

Delmonte, M. M. (1987). Personality and meditation. In M. West (Ed.), *The psychology of meditation* (pp. 118–132). New York: Oxford University Press.

Derogatis, L. R., Rickels, K., & Rock, A. F. (1976). The SCL-90 and the MMPI: A step in the validation of a new self-report scale. *British Journal of Psychiatry, 128,* 280–290.

Domar, A. D., Noe, J. M., & Benson, H. (1987). The preoperative use of the relaxation response with ambulatory surgery patients. *Journal of Human Stress, 13*(3), 101–107.

Earle, J. B. (1981). Cerebral laterality and meditation: A review of the literature. *Journal of Transpersonal Psychology, 13,* 155–173.

Engstrom, M., Pihlsgard, J., Lundberg, P., & Soderfeldt, B. (2010). Functional magnetic resonance imaging of hippocampal activation during silent mantra meditation. *Journal of Alternative and Complementary Medicine, 16*(12), 1253–1258.

Fox, K. C. R., Dixon, M. L., Nijeboer, S., Girn, M., Floman, J. L., Lifshitz, M., et al. (2016). Functional neuroanatomy of meditation: A review and meta-analysis of 78 functional neuroimaging investigations. *Neuroscience and Behavioral Reviews, 65,* 208–228.

Friskey, L. M. (1985). Effects of a combined relaxation and meditation training program on hypertensive patients. *Dissertation Abstracts International, 46*(1-B), 300.

Gainotti, G. (2019). The role of the right hemisphere in emotional and behavioral disorders of patients with frontotemporal lobar degeneration: An updated review. *Frontiers in Aging Neuroscience.* Available online at *https://www.frontiersin.org/articles/10.3389/fnagi.2019.00055/full.*

Gaston, L. (1988–1989). Efficacy of imagery and meditation techniques in treating psoriasis. *Imagination, Cognition, and Personality, 8*(1), 25–38.

Gellhorn, E., & Kiely, W. F. (1972). Mystical states of consciousness: Neurophysiological and clinical aspects. *Journal of Nervous and Mental Disease, 154,* 399–405.

Glueck, B. C. (1973, March). *Current research on transcendental meditation.* Paper presented at the Transcendental Meditation Conference, Rensselaer Polytechnic Institute, Hartford Graduate Center, Hartford, CT.

Glueck, B. C., & Stroebel, C. F. (1975). Biofeedback and meditation in the treatment of psychiatric illness. *Comprehensive Psychiatry, 16,* 302–321.

Goldstein, C. M., Josephson, R., Xie, S., & Hughes, J. W. (2012). Current perspectives on the use of meditation to reduce blood pressure. *International Journal of Hypertension, 2012,* 578397.

Goleman, D. (1971). Meditation as a metatherapy: Hypothesis toward a proposed fifth state of consciousness. *Journal of Transpersonal Psychology, 3,* 1–25.

Goleman, D. J., & Schwartz, G. E. (1976). Meditation as an intervention in stress reactivity. *Journal of Consulting and Clinical Psychology, 44,* 456–466.

Haaga, D. A., Grosswald, S., Gaylord-King, C., Rainforth, M., Tanner, M., Travis, F., et al. (2011). Effects of the transcendental meditation program on substance use among university students. *Cardiology Research and Practice, 2011,* 537101. Available at *www.hindawi.com/journals/crp/2011/537101.*

Hafner, R. J. (1982). Psychological treatment of essential hypertension: A controlled comparison of meditation and meditation plus biofeedback. *Biofeedback and Self-Regulation, 7,* 305–316.

Harne, B. P., & Hiwale, A. S. (2018). EEG spectral analysis on OM mantra meditation: A pilot study. *Applied Psychophysiology and Biofeedback, 43,* 123–130.

Hawkins, M. A. (2003). Effectiveness of the transcendental meditation program in criminal rehabilitation and substance abuse recovery: A review of the research. *Journal of Offender Rehabilitation, 36*(1–4), 47–65.

Heriberto, C. (1989). The effects of clinically standardized meditation (CSM) on type II diabetics. *Dissertation Abstracts International, 49*(8-B), 3432.

Holmes, D. S. (1984). Meditation and somatic arousal reduction: A review of the experimental evidence. *American Psychologist, 39,* 1–10.

Honsberger, R. W., & Wilson, A. F. (1973).

Transcendental meditation in treating asthma. *Respiratory Therapy, 3,* 79–80.

Infante, J. R., Peran, F., Rayo, J. I., Serrano, J., Dominguez, M. L., Garcia, L., et al. (2014). Levels of immune cells in transcendental meditation practitioners. *International Journal of Yoga, 7*(2), 147–151.

Innes, K. E., Selfe, T. K., Kandati, S., Wen, S., & Huysmans, Z. (2018). Effects of mantra meditation versus music listening on knee pain, function, and related outcomes in older adults with knee osteoarthritis: An exploratory randomized clinical trial (RCT). *Evidence-Based Complementary and Alternative Medicine, 2018,* 7683897. Available at *www.ncbi.nlm.nih.gov/pmc/articles/PMC6136530*.

Janowiak, J. J., & Hackman, R. (1994). Meditation and college students' self-actualization and rated stress. *Psychological Reports, 75*(2), 1007–1010.

Kanellakos, D. (Ed.). (1974). *The psychobiology of transcendental meditation*. Menlo Park, CA: Benjamin.

Kang, S. S., Erbes, C. S., Erbes, C. R., Lamberty, G. L., Thuras, P., Sponheim, S. R., et al. (2018). Transcendental meditation for veterans with post-traumatic stress disorder. *Psychological Trauma Theory, Research Practice and Policy, 10*(6), 675–680.

Keefer, L., & Blanchard, E. B. (2002). A one-year follow-up of relaxation response meditation as a treatment for irritable bowel syndrome. *Behaviour Research and Therapy, 40*(5), 541–546.

Kirsten, G. J. V. (2001). *The use of meditation as a strategy for stress management and the promotion of wellness in teachers: An educational psychology study*. Unpublished doctoral dissertation, Potchefstroom University for Christian Higher Education, Potchefstroom, South Africa.

Klemons, I. M. (1978). Changes in inflammation in persons practicing the transcendental meditation technique. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 287–291). Livingston Manor, NY: Maharishi European Research University Press.

Kondwani, K. A. (1998). Nonpharmacologic treatment of hypertensive heart disease in African-Americans: A trial of the transcendental meditation program and a health education program. *Dissertation Abstracts International, 59*(6B), 3114.

Kozasa, E. H., Tanaka, L. H., Monson, C., Little, S., Leao, F. C., & Peres, M. P. (2012). The effects of meditation-based interventions on the treatment of fibromyalgia. *Current Pain and Headache Reports, 16*(5), 383–387.

Lang, A. J., Strauss, J. L., Bomyea, J., Bormann, J. E., Hickman, S. D., Good, R. C., et al. (2012). The theoretical and empirical basis for meditation as an intervention for PTSD. *Behavior Modification, 36*(6), 759–786.

Lawrence, A. V., Alkozei, A., Irgens, M. S., Acevedo-Molina, M. C., Brener, S. A., Chandler, A. B., et al. (2020). Think again: Adaptive repetitive thought as a transdiagnostic treatment for individuals predisposed to repetitive thinking styles. *Journal of Psychotherapy Integration*. Advance online publication.

Lazar, Z., Farwell, L., & Farrow, J. T. (1978). The effects of the transcendental meditation program on anxiety, drug abuse, cigarette smoking, and alcohol consumption. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 524–535). Livingston Manor, NY: Maharishi European Research University Press.

Lazarus, A. A. (1976). Psychiatric problems precipitated by transcendental meditation. *Psychological Reports, 10,* 39–74.

Lehrer, P. M., Sasaki, Y., & Saito, Y. (1999). Zazen and cardiac variability. *Psychosomatic Medicine, 61,* 812–821.

Lehrer, P., Schoicket, S., Carrington, P., & Woolfolk, R. L. (1980). Psychophysiological and cognitive responses to stressful stimuli in subjects practicing progressive relaxation and clinically standardized meditation. *Behaviour Research and Therapy, 18*(4), 293–303.

Leserman, J., Stuart, E. M., Mamish, M. E., & Benson, H. (1989). The efficacy of the relaxation response in preparing for cardiac surgery. *Behavioral Medicine, 15*(3), 111–117.

Maclean, C. R. K., Walton, K. G., Wenneberg, S. R., Levitsky, D. K., Mandarino, J. P., Waziri, R., et al. (1997). Effects of the transcendental meditation program on adaptive mechanisms: Changes in hormone levels and responses to stress after 4 months of practice. *Psychoneuroendocrinology, 22*(4), 277–295.

Mather, M. (2019, September 27–29). *Can we improve brain mechanisms of emotion regulation by increasing heart rate variability?* Paper presented at the annual meeting of the Society for Psychophysiological Research, Washington, DC.

McIntyre, M. E., Silverman, F. H., & Trotter, W. D. (1974). Transcendental meditation and stuttering: A preliminary report. *Perceptual and Motor Skills, 39,* 294.

Metcalf, O., Varker, T., Forbes, D., Phelps, A., DiBattista, A., Ralph, N., et al. (2016) Efficacy of fifteen emerging interventions for the treatment of posttraumatic stress disorder: A systematic review. *Journal of Traumatic Stress, 29*(1), 88–92.

Miskiman, D. E. (1978). Long-term effects of the transcendental meditation program in the treatment of insomnia. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 299–306). Livingston Manor, NY: Maharishi European Research University Press.

Morse, D. R. (1982). The effect of stress and meditation on salivary protein and bacteria: A review and pilot study. *Journal of Human Stress, 8*(4), 31–39.

Murphy, T. J., Pagano, R. R., & Marlatt, G. A. (1986). Lifestyle modification with heavy alcohol drinkers: Effects of aerobic exercise and meditation. *Addictive Behaviors, 11*(2), 175–186.

Muskatel, N., Woolfolk, R. L., Carrington, P., Lehrer, P. M., & McCann, B. S. (1984). Effect of meditation training on aspects of coronary-prone behavior. *Perceptual and Motor Skills, 58,* 515–518.

Nidich, S., Mills, P. J., Rainforth, M., Heppner, P., Schneider, R. H., Rosenthal, N. E., et al. (2018). Non-trauma-focused meditation versus exposure therapy in veterans with post-traumatic stress disorder: A randomised controlled trial. *Lancet. Psychiatry, 5*(2), 975–986.

Oman, D., & Bormann, J. E. (2015). Mantram repetition fosters self-efficacy in veterans for managing PTSD: A randomized trial. *Psychology of Religion and Spirituality, 7*(1), 34–45.

Ooi, S. L., Giovino, M., & Pak, S. C. (2017). Transcendental meditation for lowering blood pressure: An overview of systematic reviews and meta-analyses. *Complementary Therapies in Medicine, 34,* 26–34.

Orme-Johnson, D. W., Kiehlbauch, J., Moore, R., & Bristol, J. (1978). Personality and autonomic changes in prisoners practicing the transcendental meditation technique. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 556–561). Livingston Manor, NY: Maharishi European Research University Press.

Ornstein, R. (1972). *The psychology of consciousness.* San Francisco: Freeman.

Park, E. R., Traeger, L., Vranceanu, A.-M., Scult, M., Lerner, J. A., Benson, H., et al. (2013). The development of a patient-centered program based on the relaxation response: The Relaxation Response Resiliency Program. *Psychosomatics: Journal of Consultation and Liaison Psychiatry, 54*(2), 165–174.

Park, S. H., & Han, K. S. (2017). Blood pressure response to meditation and yoga: A systematic review and meta-analysis. *Journal of Alternative and Complementary Medicine, 23*(9), 685–695.

Parks, G. A., & Marlatt, G. (2004). Review of transcendental meditation in criminal rehabilitation and crime prevention. *Behavior Therapist, 27*(8), 179–182.

Patel, C. H. (1973). Yoga and bio-feedback in the management of hypertension. *Lancet, 2,* 1053–1055.

Patel, C. H. (1975). 12-month follow-up of yoga and bio-feedback in the management of hypertension. *Lancet, 1,* 62–64.

Pearl, J. H., & Carlozzi, A. F. (1994). Effect of meditation on empathy and anxiety. *Perceptual and Motor Skills, 78*(1), 297–298.

Pelletier, K. R. (1978). Effects of the transcendental meditation program on perceptual style: Increased field independence. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 337–345). Livingston Manor, NY: Maharishi European Research University Press.

Pirot, M. (1978). The effects of the transcendental meditation technique upon auditory discrimination. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 331–334). Livingston Manor, NY: Maharishi European Research University Press.

Rainworth, M. V., Alexander, C. N., & Cavanaugh, K. L. (2003). Effects of the transcendental meditation program on recidivism among former inmates of Folsom Prison: Survival analysis of 15-year follow-up data. *Journal of Offender Rehabilitation, 36*(104), 181–203.

Rimol, A. G. P. (1978). The transcendental meditation technique and its effects on sensory-motor performance. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on* *the transcendental meditation program: Collected papers* (Vol. 1, pp. 326–330). Livingston Manor, NY: Maharishi European Research University Press.

Ross, J. (1978). The effects of the transcendental meditation program on anxiety, neuroticism, and psychoticism. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 594–596). Livingston Manor, NY: Maharishi European Research University Press.

Royer, A. (1994). The role of the transcendental meditation technique in promoting smoking cessation: A longitudinal study. *Alcoholism Treatment Quarterly, 11*(1–2), 221–239.

Salk, L. (1973). The role of the heartbeat in the relations between mother and infant. *Scientific American, 228,* 24–29.

Schwartz, G., Davidson, R., & Goleman, D. (1978). Patterning of cognitive and somatic processes in the self-regulation of anxiety: Effects of meditation versus exercise. *Psychosomatic Medicine, 40,* 321–328.

Sethi, S., & Bhargava, S. C. (2003). Relationship of meditation and psychosis: Case studies. *Australian and New Zealand Journal of Psychiatry, 37*(3), 382.

Shafii, M., Lavely, R. A., & Jaffe, R. D. (1974). Meditation and marijuana. *American Journal of Psychiatry, 131,* 60–63.

Shafii, M., Lavely, R. A., & Jaffe, R. D. (1975). Meditation and the prevention of alcohol abuse. *American Journal of Psychiatry, 132,* 942–945.

Shafii, M., Lavely, R. A., & Jaffe, R. D. (1976). Decrease of smoking following meditation [German]. *Maharishi European Research University Journal, 24,* 29.

Shapiro, D. H. (1985). Clinical use of meditation as a self-regulation strategy: Comments on Holmes's conclusion and implications. *American Psychologist, 40*(6), 719–722.

Shi, L., Zhang, D., Wang, L., Zhuang, J., Cook, R., & Chen, L. (2017). Meditation and blood pressure: A meta-analysis of randomized clinical trials. *Journal of Hypertension, 35*(4), 696–706.

Shulte, M. F. (2018). *A comparison of adult responses to the use of mantra, mindfulness and spiritual meditation as a stress management tool.* Available from ProQuest Dissertations and Theses database.

Smith, J. C. (1978). Personality correlates of continuation and outcome in meditation and erect sitting control treatments. *Journal of Consulting and Clinical Psychology, 46,* 272–279.

Smith, J. C. (1985). *Relaxation dynamics: Nine world approaches to self-relaxation.* Champaign, IL: Research Press.

Smith, J. C. (1986). *Meditation: A sensible guide to a timeless discipline.* Champaign, IL: Research Press.

Smith, J. C. (1987). Meditation as psychotherapy: A new look at the evidence. In M. A. West (Ed.), *The psychology of meditation* (pp. 136–149). New York: Oxford University Press.

Sridevi, K., & Krishna, P. V. (2003). Temporal effects of meditation on cognitive style. *Journal of Indian Psychology, 21*(1), 38–51.

Stahl, J, E., Dossett, M. L., LaJoie, A. S., Denninger, J. W., Mehta, D. H., Goldman, R., et al. (2015). Relaxation response and resiliency training and its effect on healthcare resource utilization. *PLOS ONE, 10*(10), ArtID e0140212.

Suler, J. R. (1985). Meditation and somatic arousal: A comment on Holmes's review. *American Psychologist, 40*(6), 717.

Tellegen, A., & Atkinson, G. (1974). Openness to absorbing and self-altering experiences ("absorption"): A trait related to hypnotic susceptibility. *Journal of Abnormal Psychology, 83,* 268–277.

Thomas, J., Jamieson, G., & Cohen, M. (2014). Low and then high frequency oscillations of distinct right cortical networks are progressively enhanced by medium and long term Satyananda Yoga meditation practice. *Frontiers in Human Neuroscience, 8,* 197.

Tjoa, A. (1975). Increased intelligence and reduced neuroticism through the transcendental meditation program. *Gedrag: Tijdschrift voor Psychologie, 3,* 167–182.

Tjoa, A. (1978). Some evidence that the transcendental meditation program increases intelligence and reduces neuroticism as measured by psychological tests. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 363–367). Livingston Manor, NY: Maharishi European Research University Press.

Torkamani, F., Aghayousefi, A., Alipour, A., & Nami, M. (2018). Effects of single-session group mantra-meditation on salivary immunoglobulin A and affective state: A psychoneuroimmunology viewpoint. *Explore, 14*(2), 114–121.

Travis, F., Haaga, D. A. F., Hagelin, J., Tanner, M., Arenander, A., Nidich, S., et al. (2010). A self-referential default brain state: Patterns of coherence, power, and eLORETA sources during eyes-closed rest and Transcendental

Meditation practice. *Cognitive Processes, 11,* 21–30.

Travis, F., Valosek, L., Konrad, A., Link, J., Salerno, J., Scheller, R., et al. (2018). Effect of meditation on psychological distress and brain functioning: A randomized controlled study. *Brain and Cognition, 125,* 100–105.

Travis, F., & Wallace, R. K. (1999). Autonomic and EEG patterns during eyes-closed rest and transcendental meditation practice: The basis for a neural model of TM practice. *Consciousness and Cognition, 8*(3), 302–318.

Tulpule, T. (1971). Yogic exercises in the management of ischemic heart disease. *Indian Heart Journal, 23,* 259–264.

Vaschillo, E., Vaschillo, B., Buckman, J., & Bates, M. (2019, February 24–27). *New approach for brain stimulation.* Paper presented at the 3rd International Brain Stimulation Conference, Vancouver, BC.

Waelde, L. C., Meyer, H., Thompson, J. M., Thompson, L., & Gallagher-Thompson, D. (2017). Randomized controlled trial of inner resources meditation for family dementia caregivers. *Journal of Clinical Psychology, 73*(12), 1629–1641.

Wallace, R. K. (1970). Physiological effects of transcendental meditation. *Science, 167,* 1751–1754.

Wallace, R. K., Benson, H., & Wilson, A. F. (1971). A wakeful hypometabolic state. *American Journal of Physiology, 221,* 795–799.

Walton, K. G., & Levitsky, D. K. (2003). Effects of the transcendental meditation program on neuroendocrine abnormalities associated with aggression and crime. *Journal of Offender Rehabilitation, 35*(1–4), 67–87.

Warrenburg, S., & Pagano, R. R. (1982–1983). Meditation and hemispheric specialization: Absorbed attention in long-term adherents. *Imagination, Cognition, and Personality, 2*(3), 211–229.

West, M. A. (1985). Meditation and somatic arousal reduction. *American Psychologist, 40*(6), 717–719.

Williams, P., Francis, A., & Durham, R. (1976). Personality and meditation. *Perceptual and Motor Skills, 43,* 787–792.

Woolfolk, R. L., Carr-Kaffashan, K., McNulty, T. F., & Lehrer, P. M. (1976). Meditation training as a treatment for insomnia. *Behavior Therapy, 7,* 359–365.

Yogitha, B., Nagarathna, R., John, E., & Nagendra, H. (2010). Complimentary effect of yogic sound resonance relaxation technique in patients with common neck pain. *International Journal of Yoga, 3*(1), 18–25.

Yucel, H. G. (2001). The effects of the practice of the transcendental meditation technique and exercise on cognitive and psychophysiological measures in the elderly. *Dissertation Abstracts International, 62*(4B), 2100.

Zamarra, J. W., Besseghini, I., & Wittenberg, S. (1978). The effects of the transcendental meditation program on the exercise performance of patients with angina pectoris. In D. W. Orme-Johnson & J. T. Farrow (Eds.), *Scientific research on the transcendental meditation program: Collected papers* (Vol. 1, pp. 270–278). Livingston Manor, NY: Maharishi European Research University Press.

Zuroff, D. C., & Schwarz, J. C. (1978). Effects of transcendental meditation and muscle relaxation on trait anxiety, maladjustment, locus of control, and drug use. *Journal of Consulting and Clinical Psychology, 46,* 264–271.

CHAPTER 14

Mindfulness Meditation for Stress Management

Jean L. Kristeller

Mindfulness meditation is one of two traditionally identified forms of meditative practice, along with concentrative meditation (see Carrington & Lehrer, Chapter 13, this volume, on mantra meditation; Goleman, 1988; Goleman & Davidson, 2017). Over the last 15 years, *mindfulness meditation,* also referred to as "insight meditation" or "Vipassana practice," drawn from Buddhism, has become the predominant focus in defining how meditation can contribute to therapeutic growth and personal development (Creswell, 2017; Shapiro & Carlson, 2017). First identified within Buddhist traditions as *sati,* it can be argued that *mindfulness* is the first meaningful construct from Eastern philosophy to be substantively engaged by contemporary psychology.

Probably the most commonly cited definition of mindfulness is used by Jon Kabat-Zinn (2005, p. 108): "moment to moment non-judgmental awareness cultivated by paying attention in a specific way . . . in the present moment, and as non-reactively, as non-judgmentally, and as openheartedly as possible." The concept of "nonjudgment" is highly important. It refers to removing the positive or negative evaluations ("judgments") we make to virtually all experience. "Paying attention" perhaps seems obvious, but it speaks to cultivating a quality of sustained attention, with an intended focus, rather than letting attention quickly jump from one to another aspect of our external or internal environment, as it often does. Doing so without undue reactivity, judgment—and with an open heart—are all elements that can be cultivated, as will be spoken to further below.

The core practice involves attending to the feelings of the breath, a neutral object of attention, then noticing when attention has moved to something else—a thought, a feeling, a sound, for example—then observing that object without judging or reacting, before intentionally moving attention back to the breath, repeating this process over and over. This trains the capacity to engage mindfulness throughout the day, such as in a high-stress situation, and also to appreciate the value of sitting for a period of mindfulness meditation, for the sense of deep calm, wonder, and/or wisdom often evoked.

With the integration of mindfulness practice into an increasingly wider range of therapeutic uses, including online resources, the amount of recommended training varies greatly. The original clinical application, the mindfulness-based stress reduction (MBSR) program (Kabat-Zinn, 1990/2013), includes eight sessions, now a widely used model. The MBSR program, while using breath awareness practice from the beginning, also incorporates body awareness (the body scan), gentle yoga, and walking meditation. Research has explored the value of briefer training, though with mixed results (Gilmartin et al., 2017; Schumer, Lindsay, & Creswell, 2018).

Evidence (Dunn, Hartigan, & Mikulas, 1999; Xu et al., 2014) suggests that concentrative and mindfulness practices engage somewhat different neuropsychological processes. Although all meditation techniques cultivate the ability to focus and manage attention, mindfulness meditation cultivates an ability to bring nonjudgmental sustained awareness to any object of attention, in that the breath is an inherently "softer" focus that does not engage a specific cognitive focus as does a sound or word. Because of this, while initially more challenging to learn, the skill involved in maintaining focus on the breath may be at a higher level than focusing on a more concrete object. Focus on the breath is also softer in that one may more easily shift attention mindfully onto other content, such as thoughts, feelings (such as pain), or a behavior (eating, walking, etc.), thereby providing more flexibility and mindful engagement than does concentrative meditation, either for personal or therapeutic value (Shapiro & Schwartz, 2000).

HISTORY OF MINDFULNESS MEDITATION FOR STRESS MANAGEMENT

The therapeutic use of mindfulness meditation is still most often associated with the MBSR program or a variant of it. However, there is a rapidly expanding literature on other mindfulness-based interventions (MBIs), integrating mindfulness meditation into group-based approaches such as mindfulness-based cognitive therapy (MBCT; Segal, Williams, & Teasdale, 2002/2013), mindful self-compassion training (MSC; Germer & Neff, 2013, 2019), and mindfulness-based eating awareness training (MB-EAT; Kristeller, 2015; Kristeller, Wolever, & Lieberstein, in press), among others. (See Table 14.1 for a summary list of mindfulness-related abbreviations used in the chapter.)

Programs are also in use for an increasingly wide range of individuals, from children and teens to worksite settings (Biegel, Chang, Garrett, & Edwards, 2014; Dunning et al., 2018; Germer, Siegel, & Fulton, 2013). Mindfulness perspectives and techniques, including brief meditation, are used in dialectical behavior therapy (DBT; Linehan, 2015; Linehan & Wilks, 2015) and acceptance and commitment therapy (ACT; Hayes, Follette, & Linehan, 2004). Other therapeutic uses of mindfulness meditation practices include very traditional retreat-based programs (Hart, 1987) and, alternatively, use of meditation-type practices within individual therapy sessions (Germer et al., 2013).

These approaches have been informed by traditional mindfulness meditation practices mostly based in Buddhism, although meditative practices exist in virtually all religious traditions (Walsh & Shapiro, 2006). For example, DBT melds elements of Zen practice with cognitive-behavioral therapy. Buddhism also contains a wide range of traditions, with distinct meditative practices. Mindfulness meditation entered psychology through several different routes: through the Zen tradition; Southeast Asian Vipassana or Theravadan traditions; and Tibetan practices. Mindfulness is often linked to Americans who entered monastic training in Asia, particularly in the Thai Theravadan tradition, notably psychologist Jack Kornfield (1993) and Sharon Salzberg (1999), who then

TABLE 14.1. Mindfulness-Related Abbreviations: Interventions and Related Assessment Tools

Abbreviation	Term	Description
Interventions		
ACT	Acceptance and commitment therapy	Developed by Steven Hayes, ACT has increasingly incorporated mindfulness components.
DBT	Dialectical behavior therapy	Developed by Marsha Linehan for treatment of borderline personality disorder, DBT merges Zen-based theory and practice with cognitive-behavioral therapy.
MBCR	Mindfulness-based cancer recovery	An adaptation of MBSR by Linda Carlson and Michael Speca for cancer patients and family members.
MBCT	Mindfulness-based cognitive therapy	A melding of MBSR and CBT developed by Zindel Segal, Mark Williams, and John Teasdale for major depression, now modified for a wide range of clients.
MBFP	Mindfulness-based flourishing program	An online program developed by Itai Ivtzan for cultivating self-compassion and self-efficacy.
MB-EAT	Mindfulness-based eating awareness treatment	MB-EAT draws on MBSR, self-regulation theory, and eating research, developed by Jean Kristeller and associates for binge-eating disorder, now more broadly applicable.
MBIs	Mindfulness-based interventions	Refers to the growing number of programs mostly modeled on MBSR.
MBSR	Mindfulness-based stress reduction	The first secular program, developed by Jon Kabat-Zinn for chronic pain patients; now applied more broadly, throughout the world.
MBRP	Mindfulness-based relapse prevention	Developed by G. Alan Marlatt and associates for individuals with alcohol and drug issues.
MORE	Mindfulness-oriented recovery enhancement	An MBI focused on enhancing hedonic balance, developed by Eric Garland, for opioid addiction and chronic pain.
MSC	Mindful self-compassion training	MSC training incorporates a wide range of research by Christopher Germer and Kristin Neff on cultivating compassion both for self and for others.
Assessment		
CAMS-R	Cognitive and Affective Mindfulness Scale—Revised	Widely used, with a single dimension assessing attention, awareness, and acceptance of thoughts and feelings.
FFMQ	Five-Facet Mindfulness Questionnaire	Widely used measure of dispositional mindfulness, developed by Ruth Baer, incorporating several previously published measures, and defining five factors.
FMI	Freiburg Mindfulness Inventory	Developed originally for experienced meditators; sensitive to change during intensive retreats.
MAAS	Mindful Attention Awareness Scale	A reverse-scored measure developed by Kirk Brown and Richard Ryan valid in both experienced and general populations.
TMS	Toronto Mindfulness Scale	A measure intended to assess the immediate effects of mindfulness practice.

founded the Insight Meditation Society in Barre, Massachusetts, in 1976. Burmese traditions are reflected in the 10-day retreat programs led by Goenka (Hart, 1987). Tibetan meditation was substantively introduced into the United States in the early 1970s by Chogyam Trungpa Rinpoche, who founded the Naropa Institute in Boulder, Colorado, now Naropa University, dedicated to Tibetan and Buddhist studies and psychology. Interest in Tibetan practices has grown with the influence of the Dalai Lama and with deeper understanding of the impact of Tibetan meditation practices on emotional and physical self-regulation. Cultivation of positive emotion plays a central role in Tibetan Buddhism (Ricard, 2006).

Another highly influential Asian teacher has been Thich Nhat Hanh, a Vietnamese monk who resided for many years in France and whose lineage is influenced by both Theravadan and Chinese Zen (Ch'an) Buddhism. His approachable writings universalize mindfulness approaches, from his first, now classic, book, *The Miracle of Mindfulness*, in 1975, to the many that have followed (e.g., Hanh, 1996, 2017). He is particularly associated with contemplative walking meditation (Hanh, 1991) and loving-kindness meditation (Hanh, 1997). Loving-kindness meditations are brief guided practices in which individuals silently and slowly repeat phrases to themselves such as "Breathing in, may I be well . . . may I be happy . . . may I be free from suffering. . . . Breathing in, may you be well . . . may you be happy . . . may you be free from suffering . . . " (also see Alidina, 2015).

Zen meditation is not always considered a mindfulness tradition, but many aspects of Zen practice are essentially mindfulness practices. *Shikantaza* ("just sitting") refers to letting the mind be aware of whatever comes into consciousness, similar to "open awareness" practice. See the following online overviews for more understanding: *https://kirkville.com/just-sitting-the-zen-practice-of-shikantaza* is an introductory overview; *http://shoresofzen.com/index_htm_files/DefinitionsOfShikantaza.pdf* is a compiled set of commentary, from both Zen and Western perspectives.

Zen also had early influence on the incorporation of Buddhist perspectives into psychodynamic psychotherapy (Fromm, 1994; Horney, 1945, 1987) and autogenic training (see Linden, Chapter 18, this volume), and it continues to influence therapeutic practices through the work of Linehan (1993), Rubin (2016), and others (Germer & Neff, 2019). Zen practice in the United States also draws on Korean traditions (Coleman, 2001), which influenced Kabat-Zinn's work, and Chinese Zen (Ch'an) practice following the teachings of Sheng Yen, a Taiwanese Zen master, and others, including Hsing Yun, a Chinese/Taiwanese proponent of humanistic Buddhism.

Shonin and his colleagues have strongly argued that a deep understanding of Buddhist perspectives and concepts is central to fully comprehending mindfulness-based therapies (Shonin, Van Gordon, & Griffiths, 2014; Shonin, Van Gordon, & Singh, 2015). Indeed, consider that Buddhism, unlike Western traditions, has not separated religion from psychology. Therefore, one can consider Buddhism to encompass both spiritual and psychological principles and teachings. Models of underlying processes in meditation practice continue to evolve, particularly in how they link into contemporary psychological theory, while still drawing on their traditions.

THE METHOD: BASIC ELEMENTS OF MINDFULNESS MEDITATION

Mindfulness practices distinguish between focused awareness and open awareness (Lutz, Slagter, Dunne, & Davidson, 2008). Focused awareness encompasses breath awareness, brief focused practices, and guided mindfulness practices. Open awareness and focused awareness are often used in combination. Evidence, although limited, suggests differential effects for types of practice (Perlman, Salomons, Davidson, & Lutz, 2010).

Breath Awareness and Brief Focused Awareness

Vipassana practice, or insight meditation, uses a focus on the breath as a core way to both cultivate attention and reengage it when the mind becomes distracted. The breath is potent, always present, sensitive to stress reactions, inherently rhythmic in nature, and, in that attending to it is more subtle, it cultivates higher levels of attentional capacity. Further, cultivating awareness of the richness of something as simple as the breath is also an intention of mindfulness practice (Hanh, 1996). Yet when moments of racing thoughts or distractions occur, adding a cognitive element, such as briefly saying a mantra-type word or counting breaths, which may be carried even into advanced practice, may help manage awareness. (See Table 14.2 for brief instructions.)

Learning to shift one's attention to the breath at times of stress may also cultivate a positive physiological feedback system, with deeper, slower "belly" breathing engaging the vagus nerve, bringing sympathetic and parasympathetic responses into balance (Gerritsen & Band, 2018; see Lehrer, Chapters 2 and 10, and van Dixhoorn, Chapter 12, in this volume for further detail). Maintaining slower breath is an aspect of several meditation traditions, including Tibetan and Zen Rinzai practices (Lehrer, Sasaki, & Saito, 1999), but it is optional in clinical applications.

Open Awareness

Open awareness (or "open monitoring") is a core element of mindfulness practice, as shown in Table 14.2. In open awareness, one gently rests attention on whatever rises into consciousness; as that fades, attention rests on the next object of awareness. Such practice has several goals: (1) to bring awareness to experiences in the body and in the mind; (2) to disengage the reactive and analytical mind, in regard to both behavioral impulses and to content of thought (ruminating) rather than to simply observing them; (3) to train the ability to engage mindfulness more easily during daily activities.

A useful teaching metaphor is to imagine oneself sitting on the banks of a river, observing whatever is floating by: leaves, branches, perhaps a piece of trash. Our analytical mind might analyze, judge, or have associations to each object: "What type of leaf is that? . . . Where did that branch fall in? . . . Oh, who threw that trash in? Isn't that terrible . . . oh, I forgot to put out the garbage." In contrast, mindfulness involves simply observing or "noting": "leaf . . . branch . . . trash . . . " without letting the mind be carried along. A metaphor, offered by a client, is the difference between "mall walking" for exercise versus window shopping. Both are fine, but they serve different intentions. When window shopping, one may stop to chat with friends or enter a store to browse. When mall walking, doing so would defeat the intent of walking for exercise, but one would still greet friends or make a mental "note" of a window display to return to later.

This type of "noting" is often valuable, particularly when first learning to meditate—silently naming the type of thought or experience one is having, such as "analyzing," "pain," "desire to move," "phone ringing," "impatience"—and then moving back to the breath. This helps train the mind simply to be aware of, rather than "grabbing onto," content of an experience or thought. Many find mindfulness training very powerful because they now realize they have this capacity simply to observe, rather than "to analyze, judge, or react with undue worry or concern."

TABLE 14.2. Basic Instructions in Breath Awareness and Mindfulness Meditation

1. Find a quiet place and time. As preferred, set a soft timer, perhaps in the next room, for your intended length of practice time. Become comfortable in your chair, one you can sit in with a relaxed but straight, erect posture, balanced but not straining. Loosen any tight clothing that will restrict your stomach. Let your hands rest comfortably in your lap. Gently close your eyes.
2. Simply allow your body to become still. Allow your shoulders, chest, and stomach to relax. Focus your attention on the feeling of your breathing. Breathing through your nose, take two or three deeper breaths from your diaphragm, letting the air flow all the way into your stomach, without any push or strain, and then flow gently back out again. Repeat these two or three deep breaths, noticing an increased sense of calm and relaxation as you breathe in the clean, fresh air and breathe out any sense of tension or stress.
3. Now let your breathing find its own natural, comfortable rhythm and depth. Focus your attention on the feeling of your breath as it comes in at the tip of your nose, moves through the back of your throat, into your lower diaphragm, and back out again, letting your stomach rise and fall naturally with each breath.
4. Allow your attention to stay focused on your breath, away from the noise, thoughts, feelings, and concerns that usually fill your mind. Perhaps explore the nature of the breath: changes in temperature in your nose, feelings in your throat, changes in rhythm and depth.
5. As you continue, you will notice that your awareness will move to thoughts, feelings, sounds. You may find yourself remembering something from your past or thinking about the future. This is to be expected. This is the nature of the mind. As you notice this occurring, simply observe the process of the mind. You might note to yourself the nature of the thought or experience: "worry," "planning," "pain," "sound." Then gently return your attention to the breath.
6. As you notice your mind wandering off, try not to be critical of yourself. Again, understand that this is the nature of the mind—to become attached to daily concerns, to feelings, to memories, adding positive or negative self-judgment to these experiences. If you find your mind becoming preoccupied with a thought, simply notice that, rather than pursuing it at this moment. Then return your attention to your breathing. See the thought, feeling or memory as simply an activity that your mind is familiar with. Being mindful also involves just noticing where your mind goes, without judgment.
7. As you gain more experience, you may choose to use open awareness to let your mind stay with the thought, feeling, or emotion that arises, for a few moments, doing so with intention, much as you might watch a bird flying across the sky, with curiosity and gentle observation. With even more experience, you might practice open awareness for longer, using the breath only occasionally to ground your awareness.
8. When you are ready, or with the sound of the timer, gently bring your attention back just to the breath, taking a few deeper breaths. Now bring your attention back into the space of your body and into the space of the room. Move around gently in the space of the chair. When you are ready, open your eyes and gently stretch out.

Guided Awareness

In guided meditation practice, one brings awareness to a chosen experience, but again in a mindful, rather than analytical or judgmental, way. The goal is to first increase awareness of the targeted object, observing any cognitive, behavioral, or emotional reactivity, then to shift them into more reflective "responses." Guided meditations may be elements of general mindfulness practice, such as occurs in the MBSR program related to symptoms such as pain or anxiety, or as fully "scripted" meditations. Such scripted meditations may be as brief as a loving-kindness meditation, or they may make up a substantial part of a treatment program, as in the MBCT program for depression (Segal et al., 2002/2013) or related to eating in the MB-EAT program (Kristeller, Wolever, &

Sheets, 2013; Kristeller et al., in press); they can also be integrated into individual therapy. Within individual therapy, the therapist, trained in one or more mindfulness-based approaches, may introduce a client to mindfulness practice in general and then to particular focused mindfulness practices that may last only a few minutes or perhaps longer.

How do such focused or guided meditations differ from imagery work or hypnosis-based therapy? There is, of course, overlap in the use of focused attention and disengagement of usual thought processes. The processes in autogenic training overlap more fully. The distinctions from hypnosis are clearer; hypnosis more generally cultivates mental processing of images and experience, both spontaneous and suggested (see Karlin, Chapter 19, this volume), whereas mindfulness practice cultivates "bare awareness" that may be brought to any life situation, feeling, or thought; guided practices provide experience in doing so (Thompson, Waelde, Tisza, & Spiegel, 2016). In my experience, individuals may also experience hypnosis as something "done to them," whereas mindfulness meditation cultivates a greater sense of discovery and internalization of awareness and self-control. However, there has been long-standing interest in combining these approaches clinically.

It is also useful to consider body-focused practices as a distinct type of guided meditation practice. Body practices include body scanning, mindful yoga, walking meditation, and guided meditations on the senses or interoceptive experience. The word *yoga* comes from the Sanskrit term *yuj,* meaning "to yoke," as in yoking the mind, spirit, and body. From a therapeutic perspective, the type and degree of emphasis on body work can be adjusted to therapeutic goals and the needs (or limitations) of a particular client or population.

Mindfulness: Treatment Formats

Perhaps the best known and most fully researched mindfulness approach is the MBSR group program (Kabat-Zinn, 1990/2013). The basic structure includes eight weekly sessions of 2½–3 hours each, with a full-day silent retreat after Session 6. Typically, about 25 people attend; group sharing is an important aspect of the program. After learning breath awareness, body scan, gentle yoga, and walking meditation in the first few sessions, they continue with formal sitting mindfulness meditation. Daily home practice includes use of audio files of guided practices, 45 minutes in length, reflecting traditional practice time. Information is also provided on stress management and creating a healthy lifestyle. While informed by Buddhist practice, presentation of material is strictly secular. Orientation sessions occur prior to the program; assessment includes medical and psychiatric symptom checklists, but individuals are rarely screened out. While individuals may sometimes experience highly charged emotional reactions, rarely (less than 1%) do these require referral or withdrawal (Kabat-Zinn, personal communication, June 2004). Meta-analyses and reviews have generally supported the value of MBSR for a range of issues/populations (see Table 14.3 later in the chapter). Training and certification programs for MBSR leaders are offered by the Center for Mindfulness, linked to the University of Massachusetts Memorial Health Care. Other MBIs also offer certification programs.

MBCT, the second most widely used and researched program, adapted the MBSR program to address the downward spiral of negative thinking and emotion that contribute to relapse in clinical depression (Teasdale et al., 2000). MBCT, structured similarly to MBSR, gradually engages awareness of mood states, expanding in Sessions 4–6 to negative automatic thoughts, cultivating acceptance, and seeing thoughts as "just thoughts." The "cognitive" components integrated into the MBCT program draw elements from cognitive-behavioral therapy (CBT). Mindfulness is used, rather than formal "thought logs," to cultivate awareness of such patterns. The last two sessions focus on positive self-care, creating mastery, and relapse prevention. Excellent overviews of the clinical flow of MBCT are provided by Kuyken and Evans (2014) and Segal et al. (2002/2013). Gotink and his associates (2015) provide an overview of the meta-analyses exploring both MBSR and MBCT.

Other mindfulness-based group programs are available for an increasingly wide range of issues and populations. Perhaps most notable is Linehan's DBT, originally developed to use for borderline personality disorder (Linehan, 2015; Linehan & Wilks, 2015). Mindfulness practice is core to DBT but in a more limited way than in MBSR or MBCT. DBT is now extended to eating disorders (Linardon, Gleeson, Yap, Murphy, & Brennan, 2018; Robinson & Safer, 2012), generalized anxiety disorder (Roemer, Fuchs, & Orsillo, 2014), and anger and aggressive behavior (Frazier & Vela, 2014). ACT, originally developed to enrich cognitive therapy with self-acceptance practices and self-awareness of intention, now incorporates more active mindfulness into the program. ACT, along with DBT, MBSR, MBCT, and other mindfulness-based programs such as MB-EAT, are often referred to as third-wave cognitive-behavioral therapies (Springer, 2012).

The value of mindfulness practice in becoming more aware of subtle or complex feelings is compatible with insight-oriented psychodynamic approaches (Epstein, 2007; Rubin, 1996, 2016). The evidence that mindfulness meditation helps access higher levels of wisdom, inner awareness, or spiritual experience when facing stress or anxiety also makes it compatible with humanistic/transpersonal approaches to therapy (Khong & Mruk, 2009).

Much briefer introductions of mindfulness practice are gaining attention. A meta-analysis found small to medium effect sizes on improvement of mood for programs ranging from a single session to 2-week programs, with stronger effects for community relative to student samples. One caution was evidence of publication bias reducing effect size (Schumer et al., 2018).

In contrast, a traditional form of Vipassana meditation, a 10-day intensive retreat model, was developed by Goenka, from Burma, in India about 30 years ago (Hart, 1987). Silence is maintained, with 10 hours per day spent in meditation. After 3 days of breath awareness, the program shifts to mindful observation of physical and mental experiences. Each evening presents a secular Buddhist perspective on suffering, stress, and the value of meditative practice. The program has been used in both the India and US prison systems (Bowen et al., 2006), with the transformative impact documented in the film *Doing Time, Doing Vipassana* (Menahemi & Ariel, 1997), and is also available more generally.

Integrating mindfulness meditation practice into individual therapy has garnered more interest in the past 10 years. A study compared MBCT delivered individually or in the standard group, finding comparable effects (Schroevers, Tovote, Snippe, & Fleer, 2016). Mindfulness can be a primary component of treatment or can draw on clients' own practice experience to complement psychotherapy. There are increasing resources for self-teaching mindfulness (Mikulas, 2015), the value of which is supported by a meta-analysis (Cavanagh, Strauss, Forder, & Jones, 2014).

Length and Type of Practice

Formal mindfulness practice entails practicing for certain lengths of time, such as 20, 30, or 45 minutes, once or twice per day. Shorter periods of time, such as 5–10 minutes, may be helpful initially in teaching children or in special settings. The 3-minute mindfulness pause is a core part of MBCT used to diffuse depressive thoughts/feelings. In our MB-EAT program, we emphasize using “mini-meditations” of just a few moments when eating to bring mindful awareness to the food to move from “reaction” to “response” (Kristeller & Epel, 2014). Attention is brought to the breath, and then to related experiences of hunger, food choice, fullness, and so forth, cultivating choice, rather than habitual reaction.

During daily activities, a person may shift attention to the breath or simply stop and attend mindfully to whatever he or she is doing. A client of mine was struggling with almost incapacitating anger and anxiety at work. She had experience with mantra meditation but had a difficult time using her mantra in daily activities without “zoning out.” After a weekend mindfulness retreat, we explored using mini-meditations at work. She stuck small red dots in her office (on her computer monitor, her telephone, etc.) as reminders, if she felt agitated, to shift her attention gently to her breath. She returned the next week noting that this had been very helpful—and that she had also imagined a red dot on the forehead of her boss.

Meditation retreats, lasting from several hours to months, held mostly in silence, cultivate deeper awareness of the mind and body and provide a path into altered states or deeper spiritual experiences (Kozasa et al., 2015; Austin, 1998). A unique randomized clinical trial explored the effects of a 3-month retreat on experienced meditators, finding broad improvement in various aspects of self-regulation (Sahdra et al., 2011). A meta-analysis of MBIs has found positive effects of half- to full-day retreats (Visted, Vøllestad, Nielsen, & Nielsen, 2015).

THEORETICAL FOUNDATIONS: MINDFULNESS AS A COGNITIVE PROCESS

Several thousand studies have explored mindfulness-based techniques, and mindfulness is now considered a measurable human capacity, referred to as *dispositional* mindfulness (Baer et al., 2008; Bishop et al., 2004). The therapeutic value of meditation practice, originally framed mostly as a function of physical relaxation, is now better understood as a function of the cognitive–attentional processes engaged (Keng, Smoski, & Robins, 2011; Lutz, Jha, Dunne, & Saron, 2015; Shapiro & Carlson, 2017; Wallace, 2006).

Skill in engaging moment-to-moment, nonjudgmental awareness of one’s present experience, whether narrowly or more broadly focused, develops over time. Often referred to as *decentering,* particularly within the MBCT program, this process refers to engaging the human capacity to observe experiences in a more detached manner, rather than overidentifying with them, and assigning them a stable reality (Shoham, Goldstein, Oren, Spivak, & Bernstein, 2017). A related concept is *decoupling,* or the separation of an emotional pull from previously conditioned reactions/behavior (Levin, Luoma, & Haeger, 2015). In the model outlined in Figure 14.1 (later in the chapter), such processes mediate the wide range of effects of mindfulness practice, including physical awareness/relaxation, emotional balance, behavioral regulation, relationship to self and others, and spiritual engagement.

As these capacities are cultivated over time, longer term meditation practice is associated with an enhancement of cerebral areas related to attention and increased synchrony across brain pathways (Lutz, Greischar, Rawlings, Ricard, & Davidson, 2004). Neuroimaging studies have shown that mindfulness practice activates the prefrontal cortex (PFC) and the anterior cingulate cortex (ACC) and modulates attentional processes and meta-awareness in self-regulatory ways (Fox et al., 2014; Lutz et al., 2008; Lutz et al., 2009; Goleman & Davidson, 2017; Davidson et al., 2003), consistent with helping individuals lessen their reactivity to challenging issues such as chronic pain or depression (Farb, Anderson, & Segal, 2012; Zeidan & Vago, 2016). Hölzel and colleagues (2013) found that individuals with generalized anxiety disorder improved to an equivalent degree with either MBSR or with a stress education control intervention, but only the MBSR program improved neurolinkages between the PFC and the amygdala, correlated with symptom improvement.

MBIs are often compared to cognitive-behavioral therapy, also informed by the premise that we construct much of our reality through imposed meaning. Cognitive therapy acts by directing us to substitute alternative content—by substituting optimistic thoughts for pessimistic thoughts or by reframing the meaning of particular experiences. Behavioral therapy works by repeatedly changing the pairing of actual triggers and responses through extinction, exposure, or practice. Mindfulness acts somewhat differently by providing a means to disengage reactive attention from whatever signal is impinging on the mind, whether threatening or engaging, by simply observing the occurrence of patterns of conditioned reacting, a type of reflective self-monitoring and deconditioning. That doing so is possible with relatively little training suggests that this is an accessible and even universal capacity (Raffone, Srinivasan, & Barendregt, 2014). Yet as the construct of mindfulness enters contemporary vocabulary and is being recognized as a meaningful human capacity, the term *mindfulness* is being discussed in increasingly complex ways (Van Dam et al., 2018).

One first becomes aware that most physical or emotional experiences are in flux; they rise and fall, rather than being constant. Then, by disengaging the stimulus from conditioned reactions over and over again, the mind creates different patterns of responding, consistent with contemporary learning theory. With this, one becomes aware of an inherent ability to purposefully disengage from the usual chatter of the conscious mind, often experienced as a sense of space, liberation, and freedom, a release from operating on "automatic." Finally, this process of disengaging immediate associative reactions allows a broader range of connections and perspectives. Patients often report experiencing their alternative choices as fresh and in some way unexpected, along with a growing sense of insight and wisdom, emerging from their own capacities rather than being directed or prescribed from the outside, as a reintegration or synchronization of existing neural networks becomes possible (Germer & Siegel, 2012).

Lutz and his associates (2015), as part of a special issue on mindfulness in *American Psychologist,* assert that mindfulness is best conceptualized as a multidimensional process engaging complex neurocognitive elements. Although studies exploring meditational processes are still limited in number, a meta-analysis (Gu, Strauss, Bond, & Cavanagh, 2015), primarily focused on MBSR and MBCT, found that decreases in cognitive and emotional reactivity were indeed mediators of change.

CLINICAL EFFECTS OF MINDFULNESS MEDITATION: APPLYING THE MULTIDOMAIN MODEL

Because meditation practice affects basic processes by which we encode and respond to meaning in our perceptual and internal experience, effects of meditation practice can appear across all areas of functioning. Based on contemporary psychological theory, clinical application, and research to date, the following six domains, as noted above, are heuristically useful in framing meditation effects: (1) attentional/cognitive, (2) physiological, (3) emotional, (4) behavioral, (5) relation to self and to others, and (6) spiritual, as depicted in Figure 14.1.

Stage of Development		Attentional/Cognitive	Physical	Emotional	Behavioral	Relation to Self/ Others	Spiritual
	Advanced	Integration of Effects/Exceptional Capacities/Sustained Insight and Spiritual Wisdom					
	Intermediate	• Altered states • Attentional flexibility • Decrease in ruminative thinking • Increased mindfulness	• Pain reduction • Pain control • Change in physiological processes • Breath control	• Sustained equanimity • Positive emotion • Engagement in the moment • Reduced anxiety/ anger/depression	• Compassionate behavior • Addictive behavior • Adaptive behavior • Deconditioning	• Dissolving attachment to sense of self • Connectedness to others • Empathy • Self-integration • Deeper compassion	• Altered states • Mystical experiences • Awareness of "transcendence" • Unselfish love • Heightened sense of inner peace/ calm
	Initial	• Ability to focus • Awareness of mind/thoughts	• Awareness of breath • Awareness of the body • Relaxation response	• Awareness of reactivity • Awareness of emotional patterns • Decreasing reactivity/ judgment	• Awareness of behavior patterns • Impulse control	• Self-acceptance • Sense of self • Compassion for self and others	• Spiritual engagement • Sense of awe
Domain		Attentional/Cognitive	Physical	Emotional	Behavioral	Relation to Self/ Others	Spiritual

FIGURE 14.1. A multidomain model of meditation effects in stress management. The order of effects within the intermediate stage may vary considerably across individuals. The dashed lines between domains reflect that these domains interact with each other.

The order of columns in Figure 14.1 is not arbitrary. Cognitive elements are placed first, as the primary mediating processes and as objects of practice. Physical effects are next; most clients, on first experiencing meditation, note how physically relaxing it feels; further, body awareness is an easily accessible focus. Emotional effects encompass decreases in negative affect and heightened positive experiences. Behavioral change may be more challenging, benefiting from guided practices. Shifts in relation to self and to others proceed as self-judgment decreases and with directed compassion practices. Finally, greater spiritual engagement and well-being is a virtually universal goal of meditative traditions. The dashed vertical lines in Figure 14.1 reflect that as effects develop within each domain, they also interact with each other. The dashed horizontal line indicates that initial effects (below the line) may occur with relatively little practice. The second level represents effects that follow with further practice, but with considerable variability in how readily such effects are experienced. Practice within a particular domain—for example, with use of guided meditations—may cultivate more rapid growth.

More advanced effects such as spiritual awakening, often beyond the goals of therapeutic work, are also depicted in Figure 14.1. A hallmark of this level is sustainability of effects, despite life challenges; another is cultivation of certain exceptional capacities. Yet because the traditional literature is replete with references to extraordinary states, insight, and spiritual enlightenment, beginning meditators may be confused about what to expect, perhaps leading to unrealistic expectations. Fleeting experiences with unusual states may occur early in practice on occasion, contributing to this confusion, to frustration at not being able to sustain such effects, or to a lack of appreciation for the value of practice for more readily accessible effects.

Mindfulness and Empirical Evidence across Domains

The following sections review the current research and are organized by domains/subareas, linked with Figure 14.1, in relation to demonstrated efficacy. Also refer to Table 14.3 for a listing of related key meta-analyses and reviews organized by and within domains.

Overall Effects

By mid-2013, a meta-analysis of MBIs drew on 209 studies (Khoury, Lecomte, Fortin, et al., 2013), showing comparable effects of MBIs to CBT and psychopharmacology and greater effects in comparison to other control conditions. Other meta-analyses are now showing effects for stress reduction in healthy individuals (Khoury, Sharma, Rush, & Fournier, 2015), in primary care (Demarzo et al., 2015), in older adults (Hazlett-Stevens, Singer, & Chong, 2018), and in the prison population (Shonin, Van Gordon, Slade, & Griffiths, 2013). An inclusive meta-analysis of psychiatric disorders (Goldberg, Tucker, Greene, Davidson, et al., 2018) examined efficacy by five types of control groups (from wait-list to evidence-based intervention), finding significant improved value both immediately after intervention and at follow-up for most comparisons, with MBIs being comparable to evidence-based alternatives. There are now enough meta-analyses in core areas for a meta-analysis of the meta-analytic studies (Gotink et al., 2015). With the focus primarily on MBSR and MBCT, results show significant improvement in depressive symptoms, anxiety, stress, quality of life, and general physical functioning.

Most meta-analyses focus on specific areas of application. The next sections review the literature across the six domains, some of which can be mapped onto patterns of underlying mechanism processes (Gu et al., 2015). The primary clinical focus of the

TABLE 14.3. Mindfulness-Related Review Papers and Meta-Analyses

Domain	Studies
Overall effects	Creswell (2017); Demarzo et al. (2015); Goldberg et al. (2018); Gotink et al., (2015); Gu, Strauss, Bond, & Cavanagh (2015); Hazlett-Stevens, Singer, & Chong (2018); Khoury, Lecomte, Fortin, et al. (2013); Khoury, Sharma, Rush, & Fournier (2015); Shonin, Van Gordon, Slade, & Griffiths (2013); Visted, Vøllestad, Nielsen, & Nielsen (2015)
Cognitive/attentional	
General/executive functioning	Levin, Luoma, & Haeger (2015); Mak, Whittingham, Cunnington, & Boyd (2018)
ADHD	Cassone (2015); Chimiklis et al. (2018); Mitchell, Zylowska, & Kollins (2015)
Thought disorders	Khoury, Lecomte, Gaudiano, & Paquin (2013)
Physiological/health	
Overall effects/telomerase	Carlson (2015); Demarzo et al. (2015); Gotink et al. (2015); Schutte & Malouff (2014)
Chronic pain/IBS/fibromyalgia/ inflammatory response	Hilton et al. (2016); Lauche (2013); Veehof et al. (2016); Zeidan & Vago (2016)
Immune functioning/cancer-related physiology/HIV	Sanada et al. (2017); Riley & Kalichman (2015)
Cancer care/depression/ posttraumatic growth	Carlson (2015); Jones et al. (2013); Rush & Sharma (2017); Shiyko et al. (2017); Zhang, Xu, Wang, & Wang (2016)
Cardiovascular health/ autonomic nervous system	Abbott et al. (2014)
Neuroregulation	Fox et al. (2014); Hölzel et al. (2013); Lutz et al. (2015)
Emotion regulation	
Overall effects	Burton, Burgess, Dean, Koutsopoulou, & Hugh-Jones (2017); Demarzo et al. (2015); Dunning et al. (2018); Lomas, Medina, Ivtzan, Rupprecht, & Eiroa-Orosa (2018); Schumer, Lindsay, & Creswell (2018)
Depression	Goldberg et al. (2019); Kuyken et al. (2016); Wang et al. (2018)
Anxiety disorders/PTSD	Borquist-Conlon et al. (2019); Hopwood & Schutte (2017); Singh & Gorey (2018); Vøllestad, Nielsen, & Nielsen (2012)
Mood—general/stress/anxiety/ anger	Khoury et al. (2015); Rush & Sharma (2017); Shi & MacBeth (2017)
Behavioral	
Eating disorders/obesity	Godfrey, Gallo, & Afari (2015)
Alcohol/drug abuse/smoking/ gambling	Chiesa & Serretti (2014); Marlatt et al. (2004) Maynard et al. (2018); Oikonomou et al. (2017); Roos, Bowen, & Witkiewitz (2017)

(continued)

TABLE 14.3. *(continued)*

Relationship to self/others	
Overall/personal growth	Kirby, Tellegen, & Steindl (2017)
Self-acceptance/self-compassion	Ferrari et al. (2019); Marsh, Chan, & MacBeth (2017)
Compassion for others/marital adjustment	Galante, Galante, Bekkers, & Gallacher (2014); Hofmann, Grossman, & Hinton (2011); Luberto et al. (2018)
Spiritual	Chiesa & Serretti (2009); Shiyko et al. (2017)
Self-help/practice levels/dose response/component analysis	Cavanagh et al. (2014); Gilmartin et al. (2017); Parsons et al. (2017); Schumer et al. (2018)
Special populations	
Children/adolescents/perinatal/health care providers/veterans/work environments/therapist training	Borquist-Conlon et al. (2019); Dunning et al. (2018); Jones et al. (2013); Mak et al. (2018); Marsh et al. (2017); Shi & MacBeth (2017); Shonin et al. (2013)

Note. Some references are repeated across categories, as applicable. Full citations are contained within the References section of the chapter.

research may or may not lie within the domain noted (e.g., Teasdale's work on major depression, relevant to emotional effects, is also cited within the cognitive domain because of the underlying mechanisms being tested).

Cognitive Effects

As reviewed earlier, meditation fundamentally is a cognitive process that involves focusing the attention at will onto an object of choice, such as bodily feelings, thereby disengaging usual conditioned reactivity or elaborative processing—the stress reaction. In a now-classic study, Lazar and her colleagues (2005) showed thickening in parts of the right prefrontal cortex in experienced meditators, indicating heightened cognitive capacity; another study showed that cortical thickening varied by level of practice (Grant et al., 2013).

Applications within the cognitive domain address the process and power of thought content and the process of attention itself. In our work, we introduce a model in which the first step is heightening awareness of the "chattering" or "monkey mind," followed by awareness of the more usual "thinking mind," and finally moving to flashes of the "wise mind." The wise mind emerges as preoccupation with everyday concerns decreases, and brain functioning shifts into "insight" mode. The cognitive construct *mind wandering* describes the *default network* of the mind when not actively engaged in a focused task (Smallwood & Schooler, 2015). Relevant to mindfulness (Vago & Zeidan, 2016), mind wandering was initially framed as particularly relevant to mood disorders, but it is also related to positive mood, as creative perspectives arise during mindfulness practice or at other times (Horan, 2009; Franklin et al., 2013).

The fact that mindfulness meditation practice can heighten objective self-awareness and disengage ruminative thinking patterns, a key element of stress, was utilized by

Teasdale and his colleagues to inform the original development of MBCT (Segal et al., 2002/2013). Teasdale (1999) differentiated between metacognitive *knowledge* (*knowing* that thoughts are not always accurate reflections of reality) and metacognitive *insight* (*experiencing* thoughts as events, rather than as being necessarily reflective of reality). This can be broadened to include neural processes underlying executive functioning, referred to above as *wise mind*. For example, executive functioning increased in children after a biweekly (30-minute session) 8-week program (Flook et al., 2010) and in older adults enrolled in an MBSR program (Moynihan et al., 2013).

Applications include attention-deficit/hyperactivity disorder (ADHD) in adults (Mitchell, Zylowska, & Kollins, 2015), students (van de Weijer-Bergsma, Formsma, Bruin, & Bögels, 2012), and children (Cassone, 2015). Use of DBT with college students found over 50% recovery from ADHD symptoms, with higher quality of life (Fleming, McMahon, Moran, Peterson, & Dreessen, 2015), with similar effects for MBCT (Gu, Xu, & Zhu, 2018). These results are consistent with those I observed for a student with ADHD taking a course I taught on the psychology of meditation. Although most students reported value within a week or two from their required practice (four times/week), she struggled with her "chattering mind," taking about 6 weeks before experiencing shifts in her ability to stay focused. By the end of the semester, she reported dramatic changes and was feeling far more hopeful for her future.

Experiencing thoughts as "just" thoughts is extremely powerful for those struggling with schizophrenia/psychotic-type thinking (Chadwick et al., 2016), supported in a meta-analysis (Khoury, Lecomte, Gaudiano, & Paquin, 2013). This effect has been shown for mindfulness in a vocational training program for individuals with stabilized schizophrenia (Davis et al., 2015) and on an individual basis in a promising pilot study of a 4-week intervention (Louise, Rossell, & Thomas, 2019). I observed such responses in a woman with several hospitalizations for paranoid psychosis, although she was otherwise highly functioning, married, and working in a responsible position. In treatment, she became aware that under stress she would construe even mild criticism as very harsh, ruminating on it with increasing paranoid ideation. With mindfulness, she became able to simply observe negative thoughts rather than reacting to them, interrupting an escalating course of paranoid ideation.

Mind-Body and Health Benefits

Much of stress is experienced in the body. Further, stress contributes to chronic health issues—and health issues lead to stress. Almost immediately, meditation elicits a sense of physical relaxation for most people. Part of this effect is due to vagal nerve stimulation that occurs with deeper, slower breathing, as noted above, identified as the "relaxation response" established in early research, primarily on mantra-based meditation (Benson, 1975; Lehrer, 1983). Very early work (Kasamatsu & Hirai, 1969) also identified dominance of alpha and theta brain rhythms, consistent with greater relaxation, work that still informs brain rhythm research (Kerr, Sacchet, Lazar, Moore, & Jones, 2013) and extends into clinically oriented studies, within the MBSR program (Hunt, Al-Braiki, Dailey, Russell, & Simon, 2018). Telomeres, the "caps" at the end of DNA strands sensitive to stress, are also responsive to meditation practice across multiple studies (Schutte & Malouff, 2014).

MBIs are valuable for a wide range of health-related issues (Carlson, 2015). Chronic pain, a disorder in which decreased symptom reactivity plays a key ameliorative role, has been a primary focus of the MBSR program, documented in the now-classic book *Full*

Catastrophe Living by Kabat-Zinn (1990/2013), though meta-analyses of chronic pain research have shown somewhat mixed effects (Hilton et al., 2016; Veehof, Trompetter, Bohlmeijer, & Schreurs, 2016). A meta-analytic review related to fibromyalgia (Lauche, Cramer, Dobos, Langhorst, & Schmidt, 2013) found effects of MBSR relative to active control conditions, for quality of life and for pain.

Peripheral physiological effects of MBSR include enhanced endocrine and immune system functioning (Davidson et al., 2003). In psoriasis, brief guided mindfulness meditation, delivered during standard light treatment by audio recorder, proved highly effective as an adjunctive treatment (Bernhard, Kristeller, & Kabat-Zinn, 1988; Kabat-Zinn et al., 1998), with more rapid improvement of the scaly, itchy skin patches. Effects on cardiac health (Abbott et al., 2014) have been explored in limited studies. The MBSR program has extended value for cancer patients across a range of psychosocial measures, including fatigue (Johns et al., 2015).

Emotional Benefits

Mindfulness can be considered one of the few tools for cultivation of emotional equanimity, a more advanced level of stress tolerance (Walsh & Shapiro, 2006; Desbordes et al., 2015), modulating underlying neurocognitive processes in emotion regulation. Davidson found that meditation enhances activity in the left PFC underlying positive emotion, both in novice meditators (Davidson et al., 2003) and, to a striking amount, in highly adept (> 10,000 hours of practice) Buddhist monks and other practitioners (Goleman, 2003). The mindfulness-based flourishing program (MBFP), an 8-week online training, draws on positive psychology, showing increased self-compassion, loving kindness, and self-efficacy, including in an ethnic Chinese sample (Ivtzan et al., 2017). Engaging positive elements has been explored for treating opioid misuse in chronic pain management as part of the mindfulness-oriented recovery enhancement (MORE) program (Garland, Howard, Zubieta, & Froeliger, 2017). Further, effects may be additive. A creative study (Kutz et al., 1985) added a 10-week intervention modeled on MBSR to ongoing individual psychodynamic therapy for 20 patients with an average of 4 previous years of treatment. Both therapists and patients noted improved anxiety and quality of life and greater insight; 80% of patients indicated that daily practice was the most valuable part, engaging a deeper sense of well-being in their daily lives.

ANXIETY

Meditation practice may be particularly powerful for anxiety disorders. The first study of MBSR on anxiety (Kabat-Zinn et al., 1992) found lower anxiety, panic, and general dysphoria, sustained 3 years later, for panic attacks and agoraphobia, declining gradually for generalized anxiety disorder (GAD; Miller, Fletcher, & Kabat-Zinn, 1995). More recent work with GAD has confirmed the value of MBSR and ACT in the shorter term (Hölzel et al., 2013; Roemer et al., 2014), suggesting that individual therapy be added as an appropriate complementary treatment for GAD. A 12-week training for young teenagers (primarily Latino and Asian) showed improvement across multiple outcome measures, with effects for perceived stress mediated by reductions in expressive suppression and rumination (Fung et al., 2018). Meta-analyses have documented the value of MBIs for anxiety disorders in adolescents (Borquist-Conlon, Maynard, Brendel, & Farina, 2019) and adults (Singh & Gorey, 2018). A meta-analysis for treatment of PTSD (Hopwood & Schutte, 2017) found moderate effects, somewhat dependent on length of training.

DEPRESSION

Mindfulness meditation interrupts cascades of negative thinking that contribute to psychobiological dysregulation. The MBCT program includes guided practices that heighten awareness of depressive-type thoughts while cultivating an ability to disengage from them. Mediation analyses highlight the value of practicing these decentering skills to prevent relapse (Segal et al., 2019). A meta-analysis found clear effects for MBCT for prevention of relapse, particularly for previously higher levels of depression (Kuyken et al., 2016). For active depression, a meta-analysis of MBCT found effects comparable to other interventions (Goldberg et al., 2019); Wang et al. (2018) found short-term effects but lack of maintenance at follow-up.

ANGER AND BROADER EMOTIONAL ADJUSTMENT

Anger management may be well suited to mindfulness approaches in that awareness, acceptance, and the ability to suspend immediate reaction are core to disengaging anger reactions. Decreases in anger rumination mediate the effects on anger expression (Peters et al., 2015). Woolfolk (1984) used a single-case reversal design with a 26-year-old construction worker who had lost several jobs due to anger. The client used mantra meditation, separately and in combination with mindful-awareness practice, to deal with typical precursors to his angry outbursts. In 4 weeks, the effects were clear; only the combination of practices, rather than mantra meditation alone, affected expression of anger. A randomized controlled trial (RCT) examined a wide range of emotional regulation responses to MBSR, finding significant effects for anger (Robins, Keng, Ekblad, & Brantley, 2012). Brantley's book *Calming Your Angry Mind* (2014), for the layperson, is an excellent accompaniment to anger-management therapy. DBT addresses the emotionally chaotic inner lives, often marked by anger, of individuals diagnosed with borderline personality disorder (Linehan, 1993; Frazier & Vela, 2014).

Research on emotional adjustment has also focused on specific populations. Much of the value of MBIs for medical patients lies in relieving distress related to challenges of treatment and fears of disability or mortality (Sagula & Rice, 2004). MBSR may be particularly powerful for patients dealing with cancer, shown in several reviews and meta-analyses (Rush & Sharma, 2017; Zainal, Booth, & Huppert, 2013; Zhang, Xu, Wang, & Wang, 2016). An adaptation, the mindfulness-based cancer recovery (MBCR) program (Carlson & Speca, 2011), has shown sustained benefits, particularly for stress. A recent meta-analysis (Veehof et al., 2016) including MBSR, MBCT, and ACT for chronic pain patients found comparable effects for emotion regulation across interventions, with higher impact of ACT on anxiety and depression. Another meta-analysis found mixed effects for brief (1–2 week) MBIs (Schumer et al., 2018). A meta-analysis of mindfulness for perinatal women found particularly strong effects for anxiety (Shi & MacBeth, 2017).

Mindfulness practice also contributes to better coping in high-stress work environments, such as for medical and other health care students and professionals, confirmed in a meta-analysis (Burton, Burgess, Dean, Koutsopoulou, & Hugh-Jones, 2017). These effects extend to business environments (Davidson et al., 2003; Wolever et al., 2012), also supported by meta-analyses (Lomas, Medina, Ivtzan, Rupprecht, & Eiroa-Orosa, 2018).

Changing Behavior Mindfully

Chronic stress is often associated with behavioral issues, whether as a cause of the behaviors or triggered by them. MBIs have been applied to drug and alcohol addiction, smoking, eating behavior, gambling, and Internet gaming. Mindfulness may help by improving emotional regulation, interrupting the chain of behavioral reactions, increasing receptivity to behavioral recommendations, or learning to ride out waves of craving (Brewer, 2017; Tapper, 2018; Witkiewitz, Lustyk, & Bowen, 2013). Mindfulness first increases awareness of behavioral patterns, followed by a sense of a general "deconditioning," of being somehow "freed" from the power of earlier patterns of avoidance or compulsions, followed then by increases in purposeful, focused, or "wiser" action. A meta-analysis of research on a range of addictive behaviors, up through 2011, found overall positive effects for MBIs (Chiesa & Serretti, 2014). The extent to which such changes occur as a function of general meditation practice or of practice explicitly focused on the behavioral intentions is not yet clear.

Eating behavior appears particularly responsive to mindfulness practice. A number of eating disorder and weight-control programs have incorporated mindfulness, with a meta-analysis of studies focusing on binge-eating disorders showing large effects (Godfrey, Gallo, & Afari, 2015). In our MB-EAT program for binge eaters, we begin with mindfully eating four raisins, adapted from the MBSR program, followed by more challenging foods, including a buffet meal. There are also guided meditations on awareness of physical hunger, taste, satiety, and emotional eating—and using nutritional information in a nonjudgmental manner. Evidence shows comparable effects to a CBT-based intervention for decreasing bingeing, but with greater improvement on internalization of change, related to amount of mindfulness practice (Kristeller et al., 2013). The application of MB-EAT has been broadened (Kristeller & Wolever, 2014), showing effectiveness for type 2 diabetes (Miller, Kristeller, Headings, & Nagaraja, 2014), individuals with a range of obesity (Daubenmier et al., 2016; Mason et al., 2016), and for post–bariatric surgery patients (Wnuk et al., 2017). Larger effects have also been shown for individuals with stress or compulsive eating than those without (Radin et al., 2020).

Gambling is another area in which people may behave in a compulsive way. A meta-analysis (Maynard, Wilson, Labuzienski, & Whiting, 2018) showed moderate to large effects for both gambling urges and gambling behavior. An RCT comparing the MORE program to a support group for Internet gambling disorder (IGD) found that reduction in IGD and craving were mediated by the decrease in maladaptive cognitions (Li, Garland, & Howard, 2018).

Applying mindfulness to smoking cessation seems particularly suitable, given the role of paced inhalation as part of smoking, the compelling nature of craving for nicotine, and highly conditioned associations with smoking. Brewer (Brewer et al., 2011) found clear effects for an 8-session MBI compared with the American Lung Association's Freedom from Smoking program. In another study, those in the mindfulness condition reported better volitional control over smoking than those in the CBT group (Spears et al., 2017). A meta-analysis (Oikonomou, Arvanitis, & Sokolove, 2017) found an overall 4-month quit rate of 25.2%, double the 12.6% for comparison treatments. A very strong study of underlying mechanisms of self-regulation (Tang, Tang, & Posner, 2016) showed that, following brief mindfulness training, smokers increased activity in the anterior cingulate cortex (ACC) and adjacent medial prefrontal cortex (mPFC) areas, comparable to nonsmokers.

Research has consistently found a reduction in drug and alcohol use as a result of practicing mindfulness meditation (Li, Howard, Garland, McGovern, & Lazar, 2017). An early application was the mindfulness-based relapse prevention (MBRP) program (Bowen, Chawla, Grow, & Marlatt, 2021), integrating mindfulness into relapse prevention approaches (Marlatt & Gordon, 1985). Although the CBT-based control condition was initially more effective, MBRP showed stronger effects at 1 year, particularly for those with more severe dependence and higher anxiety or depression (Roos, Bowen, &

Witkiewitz, 2017). Marlatt and his associates (Bowen et al., 2006) compared prison inmates about to be discharged, who volunteered for a 10-day Vipassana retreat as created by Goenka (described earlier), with those in the usual process. Three months later, these individuals had significantly lower levels of marijuana, crack cocaine, and alcohol use, in comparison with the controls, along with improvement on impulse control, psychiatric symptoms, and optimism.

Relationship to Self and Others

Harsh self-judgment is a source of stress, and lack of social connectedness contributes to poor adjustment. Gilbert, in his wonderful book *The Compassionate Mind* (2009), opened the door wider to the psychology of mindfulness in cultivating self-compassion and compassion toward others. Since then, further research is evidenced in Kirby, Tellegen, and Stiendl's meta-analysis of compassion-based interventions (2017), along with a rich overview in Germer and Siegel's edited volume *Wisdom and Compassion in Psychotherapy* (2012).

One paradox of meditation practice is how a clearly inner-focused undertaking can cultivate empathy and altruism, elements central to Buddhism. The answer may lie within the process of decreasing self-protective reactivity, one that can be supplemented with guided meditations that cultivate self-acceptance and caring for others. Exploring, understanding, and shifting one's relationship to sense of self and to others can be considered core to both traditional and contemporary use of mindfulness practices (Kristeller & Johnson, 2005).

UNDERSTANDING THE "SELF"

The mind constructs meaning out of experience, encapsulated by conscious thoughts (Mahoney, 2003). A central tenet of Buddhist psychology is that conditioned desires distort perception, contribute to an illusionary sense of self, and, by producing craving and attachment, are the primary source of distress. For example, addictions are powerfully directed by constructed thoughts and conditioned reactions, which the individual experiences both as uncontrollable and as integral aspects of "self." Indeed, Albert Ellis notably linked his work in rational–emotive behavior therapy to Zen perspectives in regard to the value of disengaging the identity of the "self" from the content of one's thought (Kwee & Ellis, 1998). A core process in mindfulness is "dis-identification," observing experiences without overinvesting a sense of self into them (Walsh & Shapiro, 2006). Suspending this identification of self, laden with judgment, either positive or negative, promotes self-acceptance and self-growth, deepening engagement of inner sources of strength and higher capacities. It also incorporates Buddhist concepts of multiple facets of self, in contrast to Western perspectives that there is one "true" self. It can be quite releasing for clients to recognize that they can experience different aspects of self, none of which can—or should—fully define who they are. I have found this perspective particularly powerful in helping people disengage from overidentification with a diagnosis—that they are much more than their depression, bipolar disorder, or phobia.

SELF-ACCEPTANCE AND SELF-COMPASSION

Much as we project positive or negative valuation on experience, we also do so in relation to self-perception. Higher levels of self-compassion are confirmed as key mediating processes in the MBSR program (Keng, Smoski, Robins, Ekblad, & Brantley, 2012).

Greenberg and his associates (2018) found that in an MBCT program, greater self-compassion protected against mind wandering at baseline and at the end of treatment, decreasing links to depression. Mindful awareness may also aid the clinician in cultivating empathic concern (Bruce, Shapiro, Manber, & Constantino, 2010). Such effects have been supported in a meta-analysis for adolescents (Marsh, Chan, & MacBeth, 2017).

Guided self-acceptance practices are powerful for cultivating positive self-awareness, as shown in Neff and Germer's (2013) important work with MSC, maintained out to 1 year. A meta-analysis supports the value of guided practices for cultivation of these qualities (Ferrari et al., 2019). Two trials explored a brief mindfulness intervention, finding that the addition of acceptance components created more positive affect (Lindsay, Chin, et al., 2018) and less stress reactivity (Lindsay, Young, Smyth, Brown, & Creswell, 2018). Shorter programs have also found that increases in self-kindness mediated decreases in depression and stress in younger breast cancer patients (Boyle, Stanton, Ganz, Crespi, & Bower, 2017) and led to a range of positive effects in university students, still present after 6 months (Dundas, Binder, Hansen, & Stige, 2017).

COMPASSION TOWARD OTHERS

Buddhist practices (Davidson & Harrington, 2002) incorporate a strong focus on cultivating compassion, both toward others and toward oneself. Kornfield (1993) has written eloquently of meditation as a path to loving kindness and to opening the heart, as has Sharon Salzberg (1999). Thich Nhat Hanh's (1997) now-classic brief meditations on loving kindness are particularly powerful and easily incorporated into psychotherapy. The value of such components are now supported in a meta-analysis (Galante, Galante, Bekkers, & Gallacher, 2014). In one of the few RCTs exploring mindful engagement in couples, Carson, Carson, Gil, and Baucom (2004) found that a loving-kindness meditation therapy improved aspects of the relationship, even when the quality was already high. Susan Lord (2017) reports on a process, meditative dialogue, designed to help couples develop empathy. A study introducing couples to mindful preparation for an expected birth found effects particularly clear for the fathers (Gambrel & Piercy, 2015). Preschool children have been shown to increase their kindness toward others while involved in a 12-week mindful-kindness program (Flook, Goldberg, Pinger, & Davidson, 2015).

Compassion toward others links to spiritual experience, widely recognized as such within religious traditions. Yet, in that individuals who do not consider themselves religiously or spiritually inclined may still engage such experiences, a sense of compassion can be placed within both areas in the current model, leading us into the final domain.

Meditation and Spiritual Well-Being

Spiritual well-being has received increasing attention within therapeutic contexts as an important component of optimal coping (Kristeller, 2011; Plante, 2016) in the face of significant life stressors, such as cancer and trauma (Aldwin, Park, Jeong, & Nath, 2014; Shiyko, Hallinan, & Naito, 2017). Traditionally, spiritual growth is a goal of all meditative traditions, with meditative practice playing a core role in many religious training traditions (Walsh, 1999; Trammel, 2017).

Although examination of the neurophysiological processes underlying spiritual experience remains at an exploratory stage (Newberg, 2014; Austin, 1998; Walach, Schmidt, & Jonas, 2013), meditation practice is almost universally used to cultivate such experiences. The processes appear to involve disengagement from other preoccupations, followed by potentiation of neurological functions specific to spiritual experience. Brain imaging provides intriguing evidence regarding brain responses during spiritual experience in meditators (Newberg, 2014; Newberg & Waldman, 2009). In a broad range of participants, a review (Barnby, Bailey, Chambers, & Fitzgerald, 2015) suggests an overlap in the underlying processes, with meditation engaging the prefrontal cortex and spiritual processes reducing activity in the parietal cortex.

Despite the long-standing association between meditation and cultivation of spiritual experience, most contemporary approaches to mindfulness meditation secularize meditation practice. However, as attention to spirituality as an appropriate therapeutic focus has grown (Marlatt & Kristeller, 1999), measures of spiritual engagement have been developed (Monod et al., 2011), and research is documenting effects of meditation and related practice on spiritual well-being, even within secular programs (Falb & Pargament, 2012). An early randomized study (Shapiro, Schwartz & Bonner, 1998) with medical students showed substantial changes across all measures of well-being, including spirituality, within a 7-week mindfulness meditation program.

Increases in spiritual well-being occur in the MBSR program, and may even mediate improvement in physical well-being (Carmody, Reed, Kristeller, & Merriam, 2008; Greeson et al., 2011) and levels of depression (Greeson et al., 2015). Similar effects have been documented with medical populations, including cancer patients (Carlson et al., 2016), with changes in mindfulness mediating increases in spiritual well-being (Labelle, Lawlor-Savage, Campbell, Faris, & Carlson, 2015). Another study found that increases in spirituality occurring over the course of the MB-EAT intervention appeared to mediate other indicators of improvement, including decreases in binge-eating symptoms (Kristeller & Jordan, 2018). A meta-analysis of the effects of MBSR in healthy individuals also showed consistent effects on spiritual well-being (Chiesa & Serretti, 2009).

This research suggests that when spiritual experience is engaged through mindfulness practice, even when it is not a focus of the practice, personal enhancement of these experiences occur, deepening self-regulatory capacity. The growing interest in understanding this aspect of mindfulness practice is also reflected in an increased call for MBIs to more explicitly engage spiritual aspects of traditional Buddhist teachings (Lomas, 2017; Shonin et al., 2014).

In summary, these six domains represent core aspects of human functioning, the regulation of which can be heightened and strengthened through the practice of mindful attention and awareness. This happens, regardless of the domain, by shifting neurological processes out of highly conditioned reactivity, opening access to alternative responses less focused on survival, self-protection, and immediate gratification. As capacity for this shift deepens, access becomes easier and more fluid across domains and in the face of stronger challenges.

ASSESSMENT OF MINDFULNESS AND MINDFULNESS MEDITATION

As mindfulness becomes accepted as a viable human capacity, interest in measurement of dispositional mindfulness has emerged. Multiple assessment tools are now available; Eisenlohr-Moul, Peters, and Baer (2015) provide an excellent overview of these, including the Freiburg Mindfulness Inventory (FMI) (Walach, Buchheld, Buttenmüller, Kleinknecht, & Schmidt, 2006), developed with highly experienced meditators; the Mindful Attention Awareness Scale (MAAS) (Brown & Ryan, 2003); and the Cognitive and Affective Mindfulness Scale-Revised (CAMS-R) (Feldman, Hayes, Kumar, Greeson, & Laurenceau, 2007). Widely used is Baer's 39-item Five-Facet Mindfulness Questionnaire (FFMQ), created by giving previously developed mindfulness scales to a large sample of undergraduates (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006). Five factors emerged: Nonreactivity, Observing, Acting with Awareness, Describing, and Nonjudging. Using responders with little or no meditation experience limited the research, but meta-analyses have found consistent changes on the FFMQ and other measures of mindfulness following meditation training (Goldberg, Tucker, Greene, Simpson, et al., 2018; Quaglia, Brown, Lindsay, Creswell, & Goodman, 2015). Somewhat distinct, the Toronto Mindfulness Scale (TMS; Bishop et al., 2004) assesses experiences during meditation practice itself.

It can also be valuable, both for research and in a therapeutic context, to assess the quality of meditative practice that clients are engaging. On a daily basis, individuals can track (1) type of meditation (i.e., sitting/guided/walking meditation); (2) the quality of experience (e.g., distracted, focused); and (3) therapeutic impact noticed. This can be done informally or using a self-monitoring form suiting the practice/needs of the clients. I suggest a form with five columns: time of day, type of practice, length of practice, benefits, and problems/issues that arose.

MINDFULNESS MEDITATION: CLINICAL APPLICATIONS, CHALLENGES, AND PRACTICAL ISSUES

A key question is how to deliver mindfulness meditation instruction most effectively, both clinically and in regard to patient receptivity, burden, and cost. Two excellent resources are by McCown, Reibel, and Micozzi (2011, 2016). For therapeutic value, most approaches use group programs, which entail substantive but valuable teacher training. Guidelines for integration of mindfulness approaches into psychotherapy are also being developed (Michalak et al., 2019). Wilson et al. (2017) provides an overview of implementation related to behavioral addictions. Audio programs and apps are increasingly available for home use. Reviewing them for the needs of a client is recommended; such apps often include music, nature sounds, or frequent instruction, valuable but not providing enough open awareness space for optimal mindfulness practice.

Client Engagement in Practice

Considerable attention has been given to maintaining high levels of involvement in the MBSR program (Salmon, Santorelli, Sephton, & Kabat-Zinn, 2009). Over a 2-year period in the program at the University of Massachusetts Medical Center, 76.3% of 784 individuals who were enrolled completed the program (Kabat-Zinn & Chapman-Waldrop, 1988), but they did not necessarily complete assigned home practices. The daily 45-minute meditation sessions raise one concern for compliance. A meta-analysis (Parsons, Crane, Parsons, Fjorback, & Kuyken, 2017) found small but consistent effects of the relationship of practice to outcome ($r = 0.26$, $p < .01$), with typical reported length at about 30 minutes/day. As an alternative, both the MBCT and MB-EAT programs introduce sitting meditation with 10-minute practices, increasing to 20 and then 30 minutes.

Many teachers emphasize regularity of practice over the length of practice. Sitting for even 10–20 minutes per day would be preferable to skipping days—or weeks. Although even 3 minutes may bring a mindful perspective to a range of daily activities or tasks (Harp, 1996), it would not be long enough to elicit deeper awareness. In my experience, regular periods help people move toward valuing the transformative elements of meditation. Using the analogy of learning a musical instrument or a new sport helps people understand that regular practice heightens the skills needed under the more challenging circumstances of a performance, game—or life!

Therefore, a key issue in clinical use of meditation is how formal practice of mindfulness carries over into everyday activities. Sitting every day or most days deepens and sustains the effects achieved, heightening the transfer of mindfulness to everyday life. Both work together. Carlson has examined change over the course of her MBCR program for cancer patients (Labelle, Campbell, Faris, & Carlson, 2015) and found early effects of MBCR on the FFMQ factors of Observing and Nonjudging, as well as on rumination; these changes then led to later effects, for example, on nonreactivity.

One question often raised is whether individuals will practice on their own after formal instruction ends, implying that there is little value in learning meditation if it is not practiced on an extended basis. It is also a question grappled with in many areas of therapeutic practice and behavioral change. Mindfulness training is analogous to other types of therapeutic interventions, such as cognitive restructuring, in that they cultivate a set of skills, in addition to inducing a particular state of being. The individual who participates in substantial mindfulness experience learns to focus more easily, shift attention at will, use the breath to find physical relaxation, recognize emotional reactivity better, shift from reactivity to "wise" responses, and cultivate a state of compassion toward him- or herself and others. Such abilities exist independent of meditation practice.

In the 3-year follow-up of Kabat-Zinn's study of individuals with anxiety and panic attacks (Miller et al., 1995), effects were well maintained, yet only about half of people reported any continued use of meditation practice, mostly irregular. Follow-up of chronic pain patients (Kabat-Zinn, Lipworth, Burney, & Sellers, 1986) revealed similar patterns; about half still used some breath awareness through 4 years, with 30–40% reporting regular sitting practice (three or more times/week for 15 minutes or more). A practice effect on pain experience was evident but did not reach statistical significance. Much like many skills, the basic ability to engage in mindfulness is retained, but regular practice will continue to deepen it. Many individuals do maintain regular sitting, coupled perhaps with occasional retreats, finding that this allows them to deepen their self-management skills, accessing inner wisdom and insight more readily.

Cautions and Concerns

Other Challenges to Practice

Gunaratana, a Sri Lankan Buddhist monk and meditation teacher, in his classic small book *Mindfulness in Plain English* (1991), outlines 11 problems that may arise when meditating, including physical pain, "odd" sensations, drowsiness, inability to concentrate, boredom, fear, agitation, and trying too hard. He addresses each one, with a common thread being his encouragement simply to observe these experiences as aspects of the mind and the self that may arise even for experienced meditators. If these states occur during meditation, they may be present in the background of other activities, reflecting issues to be dealt with more broadly.

Typically, most individuals find enough calm in the midst of these experiences to keep practicing. But they may need reassurance that "clearing the mind" of all thoughts or feelings is not the goal of practice—a common misperception. In contrast, someone may find his or her mind racing so much, even without ADHD, as noted earlier, that he or she is unable to find any relaxation at all during the initial experiences, regardless of whether the content of the thoughts is distressing. Reassurance that such agitation is not uncommon and that with more practice this should improve, potentially providing an even greater value of meditation, can help increase someone's willingness to stay with developing a practice. Also, as mentioned earlier, using a mantra, the technique of "noting," or starting with shorter time periods can be helpful for anyone. In contrast, someone who deeply resonates to the practice may report a sense of physical "floating." During our usual daily activities, our body awareness comes from even low-level movements that generate feedback to the brain. The floating sensation occurs when someone is able to still his or her body to a substantial degree; although unusual in beginners, when it first occurs, it can be disconcerting. Simply explaining the reason to the individual—and to the whole group—is reassuring. And the person experiencing it can be gently congratulated for a high level of inner and outer quiet and stillness.

The perceived religious context of mindfulness practice in Buddhism may be concerning for those who hold a strong identity with another religion, even to a sense of betrayal of their beliefs. I have found it very helpful first to explore this concern and then to point out that the program draws on Buddhist psychology, rather than Buddhist religion. Paradoxically, it is then helpful to point out that virtually all world religions have developed such practices to help people access inner wisdom and connect to a higher sense of spirit. These two messages together seem to quell these concerns, creating a greater sense of comfort with practicing mindfulness.

As noted earlier, another challenge may occur with more advanced meditators—or those who have read extensively in the Buddhist literature. They may misunderstand Buddhist-based teachings as requiring that one give up the ego or any sense of identity with the self. Rather than cultivating mindful awareness of the natural fluctuations of their experience, they suppress the presence of craving or desire, trying to meet a goal of spiritual attainment that is unrealistic. Epstein (1995) discusses this as confusion between "egolessness"—defined as the realization that desires or aversions do not define the "self"—and a steady state of "no-self" that can rarely be sustained. Shapiro, Siegel, and Neff (2018) usefully address these issues in a model contrasting (1) acceptance versus change, (2) escape versus engagement, (3) effort versus nonstriving, and (4) self-focus versus non-self.

Uncovering Memories, Dissociation, and Trance Experiences

Mindfulness meditation cultivates the ability to "fall awake." At the same time, meditative practice can induce trance states, access hidden memories, or create dissociative experiences (Vieten et al., 2018). Kutz and his colleagues (1985), while exploring meditation as an adjunct to psychodynamic psychotherapy, mentioned earlier, also assessed the occurrence of unpleasant reactions; four patients recovered memories of a past traumatic event when meditating. Others reported feelings of "defenselessness," leading to emotionality, anger, fear, and despair. Even so, such experiences were balanced by an enhanced sense of inner centeredness.

An excellent qualitative study of over 60 individuals with extended meditation experience (70% had over 5,000 hours of lifetime practice; Lindahl, Fisher, Cooper, Rosen, & Britton, 2017) found that although 12% noted having had a challenging experience (e.g., emotional, trauma memories) in the first few days of practice, for the majority (70%) such experiences did not happen until after at least a year of practice, mostly (72%) associated with intense retreat experience. There was a wide range of effects, in over 50 different categories: for example, 47% reporting delusional or paranormal beliefs; 72% unusual anxiety or fear; 43% traumatic memories; and 75% unusual positive affect.

Although this sample is clearly nonrepresentative of individuals enrolling in MBIs, it illustrates a wide range of challenging experiences that may occur, yet these individuals had nevertheless continued with their practice.

As noted before, the prevalence of traumatic reactions within the MBSR program, which draws from a general population, has tended to be very low, generally under 1%. Within a psychiatric setting, such experiences may be more prevalent. Within my own therapy practice, they have covered a range. A woman in the MB-EAT program found the mild dissociation she could induce so appealing that she began to "zone out" to avoid engaging with her husband ("I could be right there, and he didn't even know I was somewhere else"). An older man I saw for an anxiety disorder recovered memories of childhood sexual abuse within a few days of practice, leading him to better understand the avoidance issues he had dealt with for 50 years. A woman trying meditation to stop smoking was immediately flooded with images related to past sexual abuse, which she believed she had fully dealt with in previous therapy.

For the first woman, we reviewed appropriate use of meditation practice and explored the need for marital counseling. In the second case, the client decided he wished to continue meditating but followed it with journaling so we could more readily use recovered material in therapy, also enabling him to recognize some "false" memory components that emerged. In the third case, the woman became aware that she had been using her smoking as a way to suppress these memories of abuse and decided to return to her previous therapist for further work.

Meditating may also evoke vivid and even bizarre imagery, without entailing a significant psychiatric issue. Such individuals may need to modulate the depth or type of meditation used, consult with senior meditation teachers, or explore the significance of the imagery in a therapy context. Contraindications are rare. People with unstable psychiatric issues should wait until other treatment is established, as noted earlier. In a group context, creating a sense of quiet is important, so those who have difficulty managing social expression or who dominate group discussion might better be introduced to mindfulness in individual therapy.

Therapist Training and Practice

It is very important that a therapist have substantive personal experience with mindfulness practice before using it professionally. Internal experiences are being cultivated, and it is difficult to understand the reports of clients in regard to such experiences without personal practice. The MBSR and other certification programs assume that individuals already have a personal sitting practice. Piet, Fjorback, and Santorelli (2016) provide an excellent overview of teaching needs, incorporating sensitivity to both contemporary science and Buddhist traditions that have informed the core programs, MBSR and MBCT. Saki Santorelli, with Jon Kabat-Zinn, led the Center for Mindfulness at the University of Massachusetts Medical Center for many years. At the same time, practicing at the level of a meditation "master" is not necessary for using basic techniques in a therapeutic context or for teaching such techniques to others.

RESOURCE MATERIAL

A substantial number of resources are available to help beginning meditators understand the value of practice, including reading materials and local sitting groups and retreat centers. One consideration is whether a client is interested in the spiritual context of meditative traditions. A highly readable guide to mindfulness from a secular perspective is *Mindfulness for Dummies* (Alidina, 2015). I generally ask clients what they have read or downloaded from meditation audio files. If I am unfamiliar with it, I ask the client to bring it in to show me or to upload the app during therapy; this helps clarify previous experience or identify sources of misunderstanding.

Most sitting groups and retreat environments focus on a particular tradition (i.e., Zen, Tibetan, or Vipassana), so that should be investigated and considered in relation both to therapeutic goals and to specific training or spiritual messages that might be conveyed. A range of residential programs is offered at the Omega Institute, Rhinebeck, New York (*www.omega.com*) and at Kripalu Center for Yoga and Health, Lenox, Massachusetts (*www.kripalu.org*). Focused on mindfulness practice are the training programs at Spirit Rock Meditation Center near San Francisco (*www.spiritrock.org*) and the Insight Meditation Society near Boston (*www.dharma.org*), among others.

CASE EXAMPLE

Choosing a case example to illustrate use of mindfulness meditation in a therapeutic context is challenging. Applications are varied and are becoming increasingly so; further, much of the validated use is within a group context. In regard to the MBSR program, *Full Catastrophe Living* (Kabat-Zinn, 2013) presents substantial case material related to chronic pain management. Segal and his colleagues (Segal et al., 2002/2013), in their manual on MBCT for depression, also present useful case material. For individual therapy, Germer and his colleagues (Germer et al., 2013) illustrate varying applications of mindfulness practice in psychotherapy, as does Ron Siegel in *The Mindfulness Solution* (2010). Jeffrey Rubin (1996) and Tara Brach (2003) also draw on rich case material to illustrate their applications of mindfulness-based approaches in therapy.

The case presented here is a 40-year-old woman who participated in our MB-EAT program and then continued in individual therapy under my supervision. This case illustrates someone drawing benefit from a group program utilizing general sitting practice and guided meditations and then moving deeper with individual therapy. Ms. W entered our MB-EAT program for binge-eating disorder. She weighed more than 250 pounds and struggled with knee and hip pain. She was vivacious, professionally employed, and acknowledged turning to food as a primary way to manage stress. She had tried many diets, losing considerable weight and then gaining it back. Her family had placed substantial importance on physical fitness, but she had struggled with weight and admitted to binge-eating issues since age 15. Otherwise, she had no other notable psychiatric issues. Although projecting confidence, she was in reality extremely hard on herself, with much of her self-judgment focused on her inability to control her eating and weight.

During the MB-EAT program, she responded very positively to meditation practice, noting how valuable the mindfulness exercises were in staying away from automatic eating. Unlike many participants, she lost little weight initially. However, she noted that her relationship to food changed markedly. She said that she learned to “honor my hunger,” became aware of satiety, and came to “care what I put in my body.” Her eating patterns continued to improve during the 4-month follow-up. Several months later, she began therapy with one of the group leaders, focusing on interpersonal relations and other issues. Two and a half years later, she focused again on weight issues, re-enrolling in Weight Watchers, noting, “now I’m doing this for myself, and not for them” and lost over 50 pounds. At that time, she was binge eating only a few times a year and would be mindful of the circumstances, using the episode to examine why her stress was high enough to trigger a binge. She also noted that while she no longer craved rich foods, she enjoyed food more, and it played a better role in her life. Although rarely practicing formal meditation, she frequently engaged in mindfulness and breathing awareness, attributing much of her self-growth to the meditation training, saying "it helps me hook into my inner wisdom. Meditation slows you down enough to be in touch with God . . . and God lives in all of us."

COMMENTS AND REFLECTIONS

I have tried to convey the potential value of mindfulness meditation within the therapy context. There is a rapidly expanding appreciation of mindfulness as a cognitive process that is powerful in its potential for heightening self-regulation and for disengaging the type of automatic reactivity, whether emotional or behavioral, that leads to suffering. Meditation practice helps cultivate the ability to bring mindfulness into moment-to-moment activity, a capacity that I would assert virtually everyone can develop. Over the past 30 years, the range and complexity of mindfulness meditation practices is being increasingly recognized and appreciated. The empirical foundation for understanding the value of mindfulness-based approaches is growing steadily, within both the framework of "stress management" and, more broadly, as a means to understanding how optimal functioning may require optimal management of stress-inducing situations. As with a number of other stress management approaches outlined in this volume, it is important to keep in mind that many of these approaches go far beyond relatively simple "relaxation" effects in their value to individuals. Mindfulness meditation provides clients with tools to engage the full range of their capabilities without becoming caught up in patterns of overdetermined and long-standing emotional and behavioral reactions to stress situations.

REFERENCES

Abbott, R. A., Whear, R., Rodgers, L. R., Bethel, A., Coon, J. T., Kuyken, W., et al. (2014). Effectiveness of mindfulness-based stress reduction and mindfulness-based cognitive therapy in vascular disease: A systematic review and meta-analysis of randomised controlled trials. *Journal of Psychosomatic Research, 76*(5), 341–351.

Aldwin, C. M., Park, C. L., Jeong, Y.-J., & Nath, R. (2014). Differing pathways between religiousness, spirituality, and health: A self-regulation perspective. *Psychology of Religion and Spirituality, 6*(1), 9–21.

Alidina, S. (2015). *Mindfulness for dummies* (2nd ed.). Chichester, West Sussex, UK: Wiley.

Austin, J. H. (1998). *Zen and the brain: Toward an understanding of meditation and consciousness*. Cambridge, MA: MIT Press.

Baer, R. A., Smith, G. T., Hopkins, J., Krietemeyer, J., & Toney, L. (2006). Using self-report assessment methods to explore facets of mindfulness. *Assessment, 13*(1), 27–45.

Baer, R. A., Smith, G., Lykins, E., Button, D., Krietemeyer, J., Sauer, S., et al. (2008). Construct validity of the Five Facet Mindfulness Questionnaire in meditating and nonmeditating samples. *Assessment, 15*(3), 329–342.

Barnby, J. M., Bailey, N. W., Chambers, R., & Fitzgerald, P. B. (2015). How similar are the changes in neural activity resulting from mindfulness practice in contrast to spiritual practice? *Consciousness and Cognition, 36,* 219–232.

Benson, H. (1975). *The relaxation response*. New York: Morrow.

Bernhard, J. D., Kristeller, J., & Kabat-Zinn, J.

(1988). Effectiveness of relaxation and visualization techniques as an adjunct to phototherapy and photochemotherapy of psoriasis. *Journal of the American Academy of Dermatology, 19*(3), 572–574.

Biegel, G. M., Chang, K., Garrett, A., & Edwards, M. (2014). Mindfulness-based stress reduction for teens. In R. A. Baer (Ed.), *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications* (2nd ed. pp. 189–212). San Diego, CA: Elsevier Academic Press.

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., et al. (2004). Mindfulness: A proposed operational definition. *Clinical Psychology: Science and Practice, 11*(3), 230–241.

Borquist-Conlon, D. S., Maynard, B. R., Brendel, K. E., & Farina, A. S. J. (2019). Mindfulness-based interventions for youth with anxiety: A systematic review and meta-analysis. *Research on Social Work Practice, 29*(2), 195–205.

Bowen, S., Chawla, N., Grow, J., & Marlatt, G. A. (2021). *Mindfulness-based relapse prevention for addictive behaviors: A clinician's guide* (2nd ed.). New York: Guilford Press.

Bowen, S., Witkeiwitz, K., Dillworth, T., Chawla, N., Simpson, T., Ostafin, B., et al. (2006). Mindfulness meditation and substance use in an incarcerated population. *Psychology of Addictive Behaviors, 20*(3), 343–347.

Boyle, C. C., Stanton, A. L., Ganz, P. A., Crespi, C. M., & Bower, J. E. (2017). Improvements in emotion regulation following mindfulness meditation: Effects on depressive symptoms and perceived stress in younger breast cancer survivors. *Journal of Consulting and Clinical Psychology, 85*(4), 397–402.

Brach, T. (2003). *Radical acceptance: Embracing your life with the heart of a buddha*. New York: Bantam Books.

Brantley, J. G. (2014). *Calming your angry mind*. Oakland, CA.: New Harbinger.

Brewer, J. (2017). *The craving mind: From cigarettes to smartphones to love—why we get hooked and how we can break bad habits*. New Haven, CT: Yale University Press.

Brewer, J. A., Mallik, S., Babuscio, T. A., Nich, C., Johnson, H. E., Deleone, C. M., et al. (2011). Mindfulness training for smoking cessation: Results from a randomized controlled trial. *Drug and Alcohol Dependence, 119*(1–2), 72–80.

Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology, 84*(4), 822–848.

Bruce, N. G., Shapiro, S. L., Manber, R., & Constantino, M. J. (2010). Psychotherapist mindfulness and the psychotherapy process. *Psychotherapy: Theory, Research, Practice, Training, 47*(1), 83–97.

Burton, A., Burgess, C., Dean, S., Koutsopoulou, G. Z., & Hugh-Jones, S. (2017). How effective are mindfulness-based interventions for reducing stress among healthcare professionals?: A systematic review and meta-analysis. *Stress and Health, 33*(1), 3–13.

Carlson, L. E. (2015). Mindfulness-based interventions for physical conditions: A selective review. In K. W. Brown, J. D. Creswell, & R. M. Ryan (Eds.), *Handbook of mindfulness: Theory, research, and practice*. (pp. 405–425). New York: Guilford Press.

Carlson, L. E., & Speca, M. (2011). *Mindfulness-based cancer recovery*. Oakville, CA: New Harbinger.

Carlson, L. E., Tamagawa, R., Stephen, J., Drysdale, E., Zhong, L., & Speca, M. (2016). Randomized-controlled trial of mindfulness-based cancer recovery versus supportive expressive group therapy among distressed breast cancer survivors (MINDSET): Long-term follow-up results. *Psycho-Oncology, 25*(7), 750–759.

Carmody, J., Reed, G., Kristeller, J. L., & Merriam, P. (2008). Mindfulness, spirituality, and health-related symptoms. *Journal of Psychosomatic Research, 69*(4), 393–403.

Carson, J. W., Carson, K. M., Gil, K. M., & Baucom, D. H. (2004). Mindfulness-based relationship enhancement. *Behavior Therapy, 35*(3), 471–494.

Cassone, A. R. (2015). Mindfulness training as an adjunct to evidence-based treatment for ADHD within families. *Journal of Attention Disorders, 19*(2), 147–157.

Cavanagh, K., Strauss, C., Forder, L., & Jones, F. (2014). Can mindfulness and acceptance be learnt by self-help?: A systematic review and meta-analysis of mindfulness and acceptance-based self-help interventions. *Clinical Psychology Review, 34*(2), 118–129.

Chadwick, P., Strauss, C., Jones, A.-M., Kingdon, D., Ellett, L., Dannahy, L., et al. (2016). Group mindfulness-based intervention for distressing voices: A pragmatic randomised controlled trial. *Schizophrenia Research, 175*(1–3), 168–173.

Chiesa, A., & Serretti, A. (2009). Mindfulness-based stress reduction for stress management in healthy people: A review and meta-analysis. *Journal of Alternative and Complementary Medicine, 15*(5), 593–600.

Chiesa, A., & Serretti, A. (2014). Are mindfulness-based interventions effective for substance use disorders?: A systematic review of the evidence. *Substance Use and Misuse, 49*(5), 492–512.

Chimiklis, A. L., Dahl, V., Spears, A. P., Goss, K., Fogarty, K., & Chacko, A. (2018). Yoga, mindfulness, and meditation interventions for youth with ADHD: Systematic review and meta-analysis. *Journal of Child and Family Studies, 27,* 3155–3168.

Coleman, J. W. (2001). *The new Buddhism.* New York: Oxford University Press.

Creswell, J. D. (2017). Mindfulness interventions. *Annual Review of Psychology, 68,* 491–516.

Daubenmier, J., Moran, P. J., Kristeller, J., Acree, M., Bacchetti, P., Kemeny, M. E., et al. (2016). Effects of a mindfulness-based weight loss intervention in adults with obesity: A randomized clinical trial. *Obesity, 24*(4), 794–804.

Davidson, R. J., & Harrington, A. (Eds.). (2002). *Visions of compassion: Western scientists and Tibetan Buddhists examine human nature.* New York: Oxford University Press.

Davidson, R. J., Kabat-Zinn, J., Schumacher, J., Rosenkranz, M., Muller, D., Santorelli, S. F., et al. (2003). Alterations in brain and immune function produced by mindfulness meditation. *Psychosomatic Medicine, 65*(4), 564–570.

Davis, L. W., Lysaker, P. H., Kristeller, J. L., Salyers, M. P., Kovach, A. C., & Woller, S. (2015). Effect of mindfulness on vocational rehabilitation outcomes in stable phase schizophrenia. *Psychological Services, 12*(3), 303–312.

Demarzo, M. M. P., Montero-Marin, J., Cuijpers, P., Zabaleta-del-Olmo, E., Mahtani, K. R., Vellinga, A., et al. (2015). The efficacy of mindfulness-based interventions in primary care: A meta-analytic review. *Annals of Family Medicine, 13*(6), 573–582.

Desbordes, G., Gard, T., Hoge, E. A., Hölzel, B. K., Kerr, C., Lazar, S. W., et al. (2015). Moving beyond mindfulness: Defining equanimity as an outcome measure in meditation and contemplative research. *Mindfulness, 6*(2), 356–372.

Dundas, I., Binder, P. E., Hansen, T. G. B., & Stige, S. H. (2017). Does a short self-compassion intervention for students increase healthy self-regulation?: A randomized control trial. *Scandinavian Journal of Psychology, 58*(5), 443–450.

Dunn, B. R., Hartigan, J. A., & Mikulas, W. L. (1999). Concentration and mindfulness meditations: Unique forms of consciousness? *Applied Psychophysiology and Biofeedback, 24*(3), 147–165.

Dunning, D. L., Griffiths, K., Kuyken, W., Crane, C., Foulkes, L., Parker, J., et al. (2018). Research review: The effects of mindfulness-based interventions on cognition and mental health in children and adolescents—a meta-analysis of randomized controlled trials. *Journal of Child Psychology and Psychiatry, 60*(3), 244–258.

Eisenlohr-Moul, T., Peters, J. R., & Baer, R. A. (2015). How do mindfulness-based interventions work? Strategies for studying mechanisms of change in clinical research. In B. D. Ostafin, M. D. Robinson, & B. P. Meier (Eds.), *Handbook of mindfulness and self-regulation* (pp. 155–170). New York: Springer Science + Business Media.

Epstein, M. (1995). *Thoughts without a thinker: Psychotherapy from a Buddhist perspective.* New York: Basic Books.

Epstein, M. (2007). *Psychotherapy without the self: A Buddhist perspective.* New Haven, CT: Yale University Press.

Falb, M. D., & Pargament, K. I. (2012). Relational mindfulness, spirituality, and the therapeutic bond. *Asian Journal of Psychiatry, 5*(4), 351–354.

Farb, N. A. S., Anderson, A. K., & Segal, Z. V. (2012). The mindful brain and emotion regulation in mood disorders. *Canadian Journal of Psychiatry/La Revue Canadienne de Psychiatrie, 57*(2), 70–77.

Feldman, G. C., Hayes, A. M., Kumar, S. M., Greeson, J. M., & Larenceau, J. P. (2007). Development, factor structure, and initial validation of the Cognitive and Affective Mindfulness Scale—Revised (CAMS-R). *Journal of Psychopathology and Behavioral Assessment, 29,* 177–190.

Ferrari, M., Hunt, C., Harrysunker, A., Abbott, M. J., Beath, A. P., & Einstein, D. A. (2019). Self-compassion interventions and psychosocial outcomes: A meta-analysis of RCTs. *Mindfulness, 10,* 1455–1473.

Fleming, A. P., McMahon, R. J., Moran, L. R., Peterson, A. P., & Dreessen, A. (2015). Pilot randomized controlled trial of dialectical behavior therapy group skills training for ADHD among college students. *Journal of Attention Disorders, 19*(3), 260–271.

Flook, L., Goldberg, S., Pinger, L., & Davidson, R. (2015). Promoting prosocial behavior and self-regulatory skills in preschool children through a mindfulness-based kindness curriculum. *Developmental Psychology, 51,* 44–51.

Flook, L., Smalley, S. L., Kitil, M. J., Galla, B. M., Kaiser-Greenland, S., Locke, J., et al. (2010). Effects of mindful awareness practices on executive functions in elementary school children. *Journal of Applied School Psychology, 26*(1), 70–95.

Fox, K. C. R., Nijeboer, S., Dixon, M. L., Floman, J. L., Ellamil, M., Rumak, S. P., et al. (2014). Is meditation associated with altered brain structure?: A systematic review and meta-analysis of morphometric neuroimaging in meditation practitioners. *Neuroscience and Biobehavioral Reviews, 43,* 48–73.

Franklin, M. S., Mrazek, M. D., Anderson, C. L., Smallwood, J., Kingstone, A., & Schooler, J. W. (2013). The silver lining of a mind in the clouds: Interesting musings are associated with positive mood while mind-wandering. *Frontiers in Psychology, 4,* 583.

Frazier, S. N., & Vela, J. (2014). Dialectical behavior therapy for the treatment of anger and aggressive behavior: A review. *Aggression and Violent Behavior, 19*(2), 156–163.

Fromm, E. (1994). *The art of being.* London: Continuum International.

Fung, J., Kim, J. J., Jin, J., Chen, G., Bear, L., & Lau, A. S. (2018). A randomized trial evaluating school-based mindfulness intervention for ethnic minority youth: Exploring mediators and moderators of intervention effects. *Journal of Abnormal Child Psychology, 47*(1), 1–19.

Galante, J., Galante, I., Bekkers, M., & Gallacher, J. (2014). Effect of kindness-based meditation on health and well-being: A systematic review and meta-analysis. *Journal of Consulting and Clinical Psychology, 82,* 1101–1114.

Gambrel, L. E., & Piercy, F. P. (2015). Mindfulness-based relationship education for couples expecting their first child: Part 1. A randomized mixed-methods program evaluation. *Journal of Marital and Family Therapy, 41*(1), 5–24.

Garland, E. L., Howard, M. O., Zubieta, J.-K., & Froeliger, B. (2017). Restructuring hedonic dysregulation in chronic pain and prescription opioid misuse: Effects of mindfulness-oriented recovery enhancement on responsiveness to drug cues and natural rewards. *Psychotherapy and Psychosomatics, 86*(2), 111–112.

Germer, C. K., & Neff, K. D. (2013). Self-compassion in clinical practice. *Journal of Clinical Psychology, 69*(8), 856–867.

Germer, C., & Neff, K. (2019). *Teaching the Mindful Self-Compassion program: A guide for professionals.* New York: Guilford Press.

Germer, C. K., & Siegel, R. D. (Eds.). (2012). *Wisdom and compassion in psychotherapy: Deepening mindfulness in clinical practice.* New York: Guilford Press.

Germer, C. K., Siegel, R. D., & Fulton, P. R. (Eds.). (2013). *Mindfulness and psychotherapy* (2nd ed.). New York: Guilford Press.

Gerritsen, R. J. S., & Band, G. P. H. (2018). Breath of life: The respiratory vagal stimulation model of contemplative activity. *Frontiers in Human Neuroscience, 12,* e397. Available at *www.frontiersin.org/articles/10.3389/fnhum.2018.00397/full.*

Gilbert, P. (2009). *The compassionate mind.* London: Constable & Robinson.

Gilmartin, H., Goyal, A., Hamati, M. C., Mann, J., Saint, S., & Chopra, V. (2017). Brief mindfulness practices for healthcare providers: A systematic literature review. *American Journal of Medicine, 130*(10). Available at *www.amjmed.com/article/S0002-9343(17)30633-2/pdf.*

Godfrey, K. M., Gallo, L. C., & Afari, N. (2015). Mindfulness-based interventions for binge eating: A systematic review and meta-analysis. *Journal of Behavioral Medicine, 38*(2), 348–362.

Goldberg, S. B., Tucker, R. P., Greene, P. A., Davidson, R. J., Kearney, D. J., & Simpson, T. L. (2019). Mindfulness-based cognitive therapy for the treatment of current depressive symptoms: A meta-analysis. *Cognitive Behaviour Therapy, 48*(6), 445–462.

Goldberg, S. B., Tucker, R. P., Greene, P. A., Davidson, R. J., Wampold, B. E., Kearney, D. J., et al. (2018). Mindfulness-based interventions for psychiatric disorders: A systematic review and meta-analysis. *Clinical Psychology Review, 59,* 52–60.

Goldberg, S. B., Tucker, R. P., Greene, P. A., Simpson, T. L., Hoyt, W. T., Kearney, D. J., et al. (2018). What can we learn from randomized clinical trials about the construct validity of self-report measures of mindfulness? A meta-analysis. *Mindfulness, 10,* 775–785.

Goleman, D. (1988). *The meditative mind: The varieties of meditative experience.* New York: Putnam.

Goleman, D. (2003). *Destructive emotions: How can we overcome them? A scientific dialogue with the Dalai Lama.* New York: Bantam Books

Goleman, D., & Davidson, R. J. (2017). *Altered traits: Science reveals how meditation changes* *your mind, brain, and body*. New York: Penguin Random House.

Gotink, R. A., Chu, P., Busschbach, J. J. V., Benson, H., Fricchione, G. L., & Hunink, M. G. M. (2015). Standardised mindfulness-based interventions in healthcare: An overview of systematic reviews and meta-analyses of RCTs. *PLOS ONE, 10*(4), e0124344.

Grant, J. A., Duerden, E. G., Courtemanche, J., Cherkasova, M., Duncan, G. H., & Rainville, P. (2013). Cortical thickness, mental absorption and meditative practice: Possible implications for disorders of attention. *Biological Psychology, 92*(2), 275–281.

Greenberg, J., Datta, T., Shapero, B. G., Sevinc, G., Mischoulon, D., & Lazar, S. W. (2018). Compassionate hearts protect against wandering minds: Self-compassion moderates the effect of mind-wandering on depression. *Spirituality in Clinical Practice, 5*(3), 155–169.

Greeson, J., Smoski, M., Suarez, E., Brantley, J., Ekblad, A., Lynch, T., et al. (2015). Decreased symptoms of depression after mindfulness-based stress reduction: Potential moderating effects of religiosity, spirituality, trait mindfulness, sex, and age. *Journal of Alternative and Complementary Medicine, 21,* 166–174.

Greeson, J. M., Webber, D. M., Smoski, M. J., Brantley, J. G., Ekblad, A. G., Suarez, E. C., et al. (2011). Changes in spirituality partly explain health-related quality of life outcomes after mindfulness-based stress reduction. *Journal of Behavioral Medicine, 34*(6), 508–518.

Gu, J., Strauss, C., Bond, R., & Cavanagh, K. (2015). How do mindfulness-based cognitive therapy and mindfulness-based stress reduction improve mental health and wellbeing? A systematic review and meta-analysis of meditation studies. *Clinical Psychology Review, 37,* 1–12.

Gu, Y., Xu, G., & Zhu, Y. (2018). A randomized controlled trial of mindfulness-based cognitive therapy for college students with ADHD. *Journal of Attention Disorders, 22*(4), 388–399.

Gunaratana, H. (1991). *Mindfulness in plain English*. Boston: Wisdom.

Hanh, T. N. (1975). *The miracle of mindfulness*. Boston: Beacon Press.

Hanh, T. N. (1991). *Peace is every step*. Berkeley, CA: Parallax Press.

Hanh, T. N. (1996). *Breathe! You are alive*. New York: Bantam Books.

Hanh, T. N. (1997). *Teachings on love*. Berkeley, CA: Parallax Press.

Hanh, T. N. (2017). *The art of living: Peace and freedom in the here and now*. San Francisco: HarperOne.

Harp, D. (1996). *The three-minute meditator* (3rd ed.). Oakland, CA: New Harbinger.

Hart, W. (1987). *The art of living: Vipassana meditation as taught by S. N. Goenka*. San Francisco: HarperCollins.

Hayes, S. C., Follette, V. M., & Linehan, M. M. (Eds.) (2004). *Mindfulness and acceptance: Expanding the cognitive-behavioral tradition*. New York: Guilford Press.

Hazlett-Stevens, H., Singer, J., & Chong, A. (2018). Mindfulness-based stress reduction and mindfulness-based cognitive therapy with older adults: A qualitative review of randomized controlled outcome research. *Clinical Gerontologist, 42*(4), 347–358.

Hilton, L., Hempel, S., Ewing, B. A., Apaydin, E., Xenakis, L., Newberry, S., et al. (2016). Mindfulness meditation for chronic pain: Systematic review and meta-analysis. *Annals of Behavioral Medicine, 51*(2), 199–213.

Hofmann, S. G., Grossman, P., & Hinton, D. E. (2011). Loving-kindness and compassion meditation: Potential for psychological interventions. *Clinical Psychology Review, 31*(7), 1126–1132.

Hölzel, B. K., Hoge, E. A., Greve, D. N., Gard, T., Creswell, J. D., Brown, K. W., et al. (2013). Neural mechanisms of symptom improvements in generalized anxiety disorder following mindfulness training. *NeuroImage: Clinical, 2,* 448–458.

Hopwood, T. L., & Schutte, N. S. (2017). A meta-analytic investigation of the impact of mindfulness-based interventions on post traumatic stress. *Clinical Psychology Review, 57,* 12–20.

Horan, R. (2009). The neuropsychological connection between creativity and meditation. *Creativity Research Journal, 21*(2–3), 199–222.

Horney, K. (1945). *Our inner conflicts*. New York: Norton.

Horney, K. (1987). *Final lectures*. New York: Norton.

Hunt, M., Al-Braiki, F., Dailey, S., Russell, R., & Simon, K. (2018). Mindfulness training, yoga, or both?: Dismantling the active components of a mindfulness-based stress reduction intervention. *Mindfulness, 9*(2), 512–520.

Ivtzan, I., Young, T., Lee, H. C., Lomas, T., Daukantaitė, D., & Kjell, O. N. E. (2017). Mindfulness-based flourishing program: A cross-cultural study of Hong Kong Chinese and British participants. *Journal of Happiness Studies, 19,* 2205–2223.

Johns, S. A., Brown, L. F., Beck-Coon, K., Monahan, P. O., Tong, Y., & Kroenke, K. (2015). Randomized controlled pilot study of mindfulness-based stress reduction for persistently fatigued cancer survivors. *Psycho-Oncology, 24*(8), 885–893.

Jones, P., Blunda, M., Biegel, G., Carlson, L. E., Biel, M., & Wiener, L. (2013). Can mindfulness-based interventions help adolescents with cancer? *Psycho-Oncology, 22*(9), 2148–2151.

Kabat-Zinn, J. (2013). *Full catastrophe living* (rev. & updated). New York: Bantam Books. (Original work published 1990)

Kabat-Zinn, J. (2005). *Coming to our senses.* New York: Hyperion.

Kabat-Zinn, J., & Chapman-Waldrop, A. (1988). Compliance with an outpatient stress reduction program: Rates and predictors of program completion. *Journal of Behavioral Medicine, 11*(4), 333–352.

Kabat-Zinn, J., Lipworth, L., Burney, R., & Sellers, W. (1986). Four-year follow-up of a meditation-based program for the self-regulation of chronic pain: Treatment outcomes and compliance. *Clinical Journal of Pain, 2,* 159–173.

Kabat-Zinn, J., Massion, A. O., Kristeller, J., Peterson, L. G., Fletcher, K. E., Pbert, L., et al. (1992). Effectiveness of a meditation-based stress reduction program in the treatment of anxiety disorders. *American Journal of Psychiatry, 149*(7), 936–943.

Kabat-Zinn, J., Wheeler, E., Light, T., Skillings, A., Scharf, M. J., Cropley, T. G., et al. (1998). Influence of a mindfulness meditation–based stress reduction intervention on rates of skin clearing in patients with moderate to severe psoriasis undergoing phototherapy (uvb) and photochemotherapy (puva). *Psychosomatic Medicine, 60*(5), 625–632.

Kasamatsu, A., & Hirai, T. (1969). An electroencephalographic study on the Zen meditation (Zazen). *Psychologia, 12,* 205–225.

Keng, S. L., Smoski, M. J., & Robins, C. J. (2011). Effects of mindfulness on psychological health: A review of empirical studies. *Clinical Psychology Review, 31,* 1041–1056.

Keng, S. L., Smoski, M. J., Robins, C. J., Ekblad, A. G., & Brantley, J. G. (2012). Mechanisms of change in mindfulness-based stress reduction: Self-compassion and mindfulness as mediators of intervention outcomes. *Journal of Cognitive Psychotherapy, 26*(3), 270–280.

Kerr, C., Sacchet, M., Lazar, S., Moore, C., & Jones, S. (2013). Mindfulness starts with the body: Somatosensory attention and top-down modulation of cortical alpha rhythms in mindfulness meditation. *Frontiers In Human Neuroscience, 7.*

Khong, B. S. L., & Mruk, C. J. (2009). Editors' introduction to special issue on mindfulness. *Humanistic Psychologist, 37,* 109–116.

Khoury, B., Lecomte, T., Fortin, G., Masse, M., Therien, P., Bouchard, V., et al. (2013). Mindfulness-based therapy: A comprehensive meta-analysis. *Clinical Psychology Review, 33*(6), 763–771.

Khoury, B., Lecomte, T., Gaudiano, B. A., & Paquin, K. (2013). Mindfulness interventions for psychosis: A meta-analysis. *Schizophrenia Research, 150*(1), 176–184.

Khoury, B., Sharma, M., Rush, S. E., & Fournier, C. (2015). Mindfulness-based stress reduction for healthy individuals: A meta-analysis. *Journal of Psychosomatic Research, 78*(6), 519–528.

Kirby, J. N., Tellegen, C. L., & Steindl, S. R. (2017). A meta-analysis of compassion-based interventions: Current state of knowledge and future directions. *Behavior Therapy, 48*(6), 778–792.

Kornfield, J. (1993). *A path with heart.* New York: Bantam Books.

Kozasa, E. H., Lacerda, S. S., Menezes, C., Wallace, B. A., Radvany, J., Mello, L. E. A. M., et al. (2015). Effects of a 9-day Shamatha Buddhist meditation retreat on attention, mindfulness and self-compassion in participants with a broad range of meditation experience. *Mindfulness, 6*(6), 1235–1241.

Kristeller, J. (2011). Spirituality and meditation. In J. D. Aten, M. R. McMinn, & E. L. Worthington (Eds.), *Spiritually oriented interventions for counseling and psychotherapy.* (pp. 197–227). Washington, DC: American Psychological Association.

Kristeller, J. (2015). *The joy of half a cookie. Using mindfulness to lose weight and end the struggle with food.* New York: Penguin Random House.

Kristeller, J., & Epel, E. (2014). Mindful eating and mindless eating. In A. Ie, C. Ngnoumen, & E. Langer (Eds.), *The Wiley Blackwell handbook of mindfulness* (Vol. 2, pp. 913–933). Hoboken, NJ: Wiley.

Kristeller, J., & Johnson, T. (2005). Cultivating loving kindness: A two-stage model of the effects of meditation on empathy, compassion, and altruism. *Zygon, 40,* 391–407.

Kristeller, J., & Jordan, K. (2018). Mindful eating: Connecting with the wise self, the spiritual self. *Frontiers in Psychology, 9,* 1–11.

Kristeller, J. L., & Wolever, R. Q. (2014). Mindfulness-based eating awareness training: Treatment of overeating and obesity. In R. A. Baer (Ed.), *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications* (2nd ed., pp. 119–139). San Diego, CA: Elsevier Academic Press.

Kristeller, J., Wolever, R. Q., & Lieberstein, A. (in press). *Mindfulness-based eating awareness training (MB-EAT): A handbook of theory, research and practice.* New York: Guilford Press.

Kristeller, J., Wolever, R. Q., & Sheets, V. (2013). Mindfulness-based eating awareness treatment (MB-EAT) for binge eating disorder: A randomized clinical trial. *Mindfulness, 4,* 282–297.

Kutz, I., Leserman, J., Dorrington, C., Morrison, C. H., Borysenko, J. Z., & Benson, H. (1985). Meditation as an adjunct to psychotherapy: An outcome study. *Psychotherapy and Psychosomatics, 43*(4), 209–218.

Kuyken, W., & Evans, A. (2014). Mindfulness-based cognitive therapy for recurrent depression. In R. A. Baer (Ed.), *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications* (2nd ed., pp. 29–60). San Diego, CA: Elsevier Academic.

Kuyken, W., Warren, F. C., Taylor, R. S., Whalley, B., Crane, C., Bondolfi, G., et al. (2016). Efficacy of mindfulness-based cognitive therapy in prevention of depressive relapse: An individual patient data meta-analysis from randomized trials. *JAMA Psychiatry, 73*(6), 565–574.

Kwee, M., & Ellis, A. (1998). The interface between rational emotive behavior therapy (REBT) and Zen. *Journal of Rational-Emotive and Cognitive Behavior Therapy, 16*(1), 5–43.

Labelle, L. E., Campbell, T. S., Faris, P., & Carlson, L. E. (2015). Mediators of mindfulness-based stress reduction (MBSR): Assessing the timing and sequence of change in cancer patients. *Journal of Clinical Psychology, 71*(1), 21–40.

Labelle, L. E., Lawlor-Savage, L., Campbell, T. S., Faris, P., & Carlson, L. E. (2015). Does self-report mindfulness mediate the effect of mindfulness-based stress reduction (MBSR) on spirituality and posttraumatic growth in cancer patients? *Journal of Positive Psychology, 10*(2), 153–166.

Lauche, R., Cramer, H., Dobos, G., Langhorst, J., & Schmidt, S. (2013). A systematic review and meta-analysis of mindfulness-based stress reduction for the fibromyalgia syndrome. *Journal of Psychosomatic Research, 75,* 500–510.

Lazar, S., Kerr, C., Wasserman, R., Gray, J., Greve, D., Treadway, M., et al. (2005). Meditation experience is associated with increased cortical thickness. *NeuroReport, 16,* 1893–1897.

Lehrer, P. M. (1983). Progressive relaxation and meditation: A study of psychophysiological and therapeutic differences between two techniques. *Behaviour Research and Therapy, 21*(6), 651–662.

Lehrer, P. M., Sasaki, Y., & Saito, Y. (1999). Zazen and cardiac variability. *Psychosomatic Medicine, 61*(6), 812–821.

Levin, M. E., Luoma, J. B., & Haeger, J. A. (2015). Decoupling as a mechanism of change in mindfulness and acceptance: A literature review. *Behavior Modification, 39*(6), 870–911.

Li, W., Garland, E. L., & Howard, M. O. (2018). Therapeutic mechanisms of mindfulness-oriented recovery enhancement for internet gaming disorder: Reducing craving and addictive behavior by targeting cognitive processes. *Journal of Addictive Diseases, 37*(1–2), 5–13.

Li, W., Howard, M. O., Garland, E. L., McGovern, P., & Lazar, M. (2017). Mindfulness treatment for substance misuse: A systematic review and meta-analysis. *Journal of Substance Abuse Treatment, 75,* 62–96.

Linardon, J., Gleeson, J., Yap, K., Murphy, K., & Brennan, L. (2018). Meta-analysis of the effects of third-wave behavioural interventions on disordered eating and body image concerns: Implications for eating disorder prevention. *Cognitive Behaviour Therapy, 48*(1), 15–38.

Lindahl, J. R., Fisher, N. E., Cooper, D. J., Rosen, R. K., & Britton, W. B. (2017). The varieties of contemplative experience: A mixed-methods study of meditation-related challenges in Western Buddhists. *PLOS ONE, 12*(5), e0176239.

Lindsay, E. K., Chin, B., Greco, C. M., Young, S., Brown, K. W., Wright, A. G. C., et al. (2018). How mindfulness training promotes positive emotions: Dismantling acceptance skills training in two randomized controlled trials. *Journal of Personality and Social Psychology, 115*(6), 944–973.

Lindsay, E. K., Young, S., Smyth, J. M., Brown, K. W., & Creswell, J. D. (2018). Acceptance lowers stress reactivity: Dismantling mindfulness training in a randomized controlled trial. *Psychoneuroendocrinology, 87,* 63–73.

Linehan, M. M. (1993). *Skills training manual for treating borderline personality disorder.* New York: Guilford Press.

Linehan, M. M. (2015). *DBT® skills training manual* (2nd ed.). New York: Guilford Press.

Linehan, M. M., & Wilks, C. R. (2015). The course and evolution of dialectical behavior therapy. *American Journal of Psychotherapy, 69*(2), 97–110.

Lomas, T. (2017). Recontextualizing mindfulness: Theravada Buddhist perspectives on the ethical and spiritual dimensions of awareness. *Psychology of Religion and Spirituality, 9*(2), 209–219.

Lomas, T., Medina, J. C., Ivtzan, I., Rupprecht, S., & Eiroa-Orosa, F. J. (2018). Mindfulness-based interventions in the workplace: An inclusive systematic review and meta-analysis of their impact upon wellbeing. *Journal of Positive Psychology, 14*(5), 625–640.

Lord, S. (2017). Mindfulness and spirituality in couple therapy: The use of meditative dialogue to help couples develop compassion and empathy for themselves and each other. *Australian and New Zealand Journal of Family Therapy, 38,* 98–114.

Louise, S., Rossell, S. L., & Thomas, N. (2019). The acceptability, feasibility and potential outcomes of an individual mindfulness-based intervention for hearing voices. *Behavioural and Cognitive Psychotherapy, 47*(2), 200–216.

Luberto, C., Shinday, N., Song, R., Philpotts, L., Park, E., Fricchione, G., et al. (2018). A systematic review and meta-analysis of the effects of meditation on empathy, compassion, and prosocial behaviors. *Mindfulness, 9*(3), 708–724.

Lutz, A., Greischar, L. L., Rawlings, N. B., Ricard, M., & Davidson, R. J. (2004). Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. *Proceedings of the National Academy of Sciences of the USA, 101*(46), 16369–16373.

Lutz, A., Jha, A. P., Dunne, J. D., & Saron, C. D. (2015). Investigating the phenomenological matrix of mindfulness-related practices from a neurocognitive perspective. *American Psychologist, 70*(7), 632–658.

Lutz, A., Slagter, H., Dunne, J., & Davidson, R. J. (2008). Attention regulation and monitoring in meditation. *Trends in Cognitive Sciences, 12*(4), 163–169.

Lutz, A., Slagter, H., Rawlings, N. B., Francis, A. D., Greischar, L. L., & Davidson, R. J. (2009). Mental training enhances attentional stability: Neural and behavioral evidence. *Journal of Neuroscience, 29*(42), 13418–13427.

Mahoney, M. J. (2003). *Constructive psychotherapy: A practical guide.* New York: Guilford Press.

Mak, C., Whittingham, K., Cunnington, R., & Boyd, R. N. (2018). Efficacy of mindfulness-based interventions for attention and executive function in children and adolescents: A systematic review. *Mindfulness, 9*(1), 59–78.

Marlatt, G. A., & Gordon, J. R. (Ed.). (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors.* New York: Guilford Press.

Marlatt, G. A., & Kristeller, J. L. (1999). Mindfulness and meditation. In W. R. Miller (Ed.), *Integrating spirituality into treatment: Resources for practitioners* (pp. 67–84). Washington, DC: American Psychological Association.

Marlatt, G. A., Witkiewitz, K., Dillworth, T. M., Bowen, S. W., Parks, G. A., Macpherson, L. M., et al. (2004). Vipassana meditation as a treatment for alcohol and drug use disorders. In S. C. Hayes, V. M. Follette, & M. M. Linehan (Eds.), *Mindfulness and acceptance: Expanding the cognitive-behavioral tradition* (pp. 261–287). New York: Guilford Press.

Marsh, I. C., Chan, S. W. Y., & MacBeth, A. (2017). Self-compassion and psychological distress in adolescents: A meta-analysis. *Mindfulness, 9*(4), 1011–1027.

Mason, A. E., Epel, E. S., Kristeller, J., Moran, P. J., Dallman, M., Lustig, R. H., et al. (2016). Effects of a mindfulness-based intervention on mindful eating, sweets consumption, and fasting glucose levels in obese adults: Data from the SHINE randomized controlled trial. *Journal of Behavioral Medicine, 39*(2), 201–213.

Maynard, B. R., Wilson, A. N., Labuzienski, E., & Whiting, S. W. (2018). Mindfulness-based approaches in the treatment of disordered gambling: A systematic review and meta-analysis. *Research on Social Work Practice, 28,* 348–362.

McCown, D., Reibel, D., & Micozzi, M. S. (2011). *Teaching mindfulness: A practical guide for clinicians and educators.* New York: Springer Science.

McCown, D., Reibel, D., & Micozzi, M. S. (Eds.). (2016). *Resources for teaching mindfulness: An international handbook.* Cham, Switzerland: Springer International.

Menahemi, A., & Ariel, E. (Directors/Producers). (1997). *Doing time, doing vipassana* [DVD/online]. India: Karuna Films.

Michalak, J., Crane, C., Germer, C. K., Gold, E., Heidenreich, T., Mander, J., et al. (2019). Principles for a responsible integration of mindfulness in individual therapy. *Mindfulness, 10,* 799–811.

Mikulas, W. L. (2015). Cultivating mindfulness: A comprehensive approach. *Mindfulness, 6*(2), 398–401.

Miller, C. K., Kristeller, J. L., Headings, A., & Nagaraja, H. (2014). Comparison of a mindful eating intervention to a diabetes self-management intervention among adults with type 2 diabetes: A randomized controlled trial. *Health Education and Behavior, 41*(2), 145–154.

Miller, J. J., Fletcher, K., & Kabat-Zinn, J. (1995). Three-year follow-up and clinical implications of a mindfulness meditation–based stress reduction intervention in the treatment of anxiety disorders. *General Hospital Psychiatry, 17*(3), 192–200.

Mitchell, J. T., Zylowska, L., & Kollins, S. H. (2015). Mindfulness meditation training for attention-deficit/hyperactivity disorder in adulthood: Current empirical support, treatment overview, and future directions. *Cognitive and Behavioral Practice, 22*(2), 172–191.

Monod, S., Brennan, M., Rochat, E., Martin, E., Rochat, S., & Büla, C. J. (2011). Instruments measuring spirituality in clinical research: A systematic review. *Journal of General Internal Medicine, 26*(11), 1345–1357.

Moynihan, J. A., Chapman, B. P., Klorman, R., Krasner, M. S., Duberstein, P. R., Brown, K. W., et al. (2013). Mindfulness-based stress reduction for older adults: Effects on executive function, frontal alpha asymmetry and immune function. *Neuropsychobiology, 68*(1), 34–43.

Neff, K., & Germer, C. (2013). A pilot study and randomized controlled trial of the mindful self-compassion program. *Journal of Clinical Psychology, 69*(1), 28–44.

Newberg, A. B. (2014). The neuroscientific study of spiritual practices. *Frontiers in Psychology, 5,* 215.

Newberg, A., & Waldman, M. R. (2009). *How God changes your brain: Breakthrough findings from a leading neuroscientist.* New York: Ballantine Books.

Oikonomou, M. T., Arvanitis, M., & Sokolove, R. L. (2017). Mindfulness training for smoking cessation: A meta-analysis of randomized-controlled trials. *Journal of Health Psychology, 22*(14), 1841–1850.

Parsons, C. E., Crane, C., Parsons, L. J., Fjorback, L. O., & Kuyken, W. (2017). Home practice in mindfulness-based cognitive therapy and mindfulness-based stress reduction: A systematic review and meta-analysis of participants' mindfulness practice and its association with outcomes. *Behaviour Research and Therapy, 95,* 29–41.

Perlman, D. M., Salomons, T. V., Davidson, R. J., & Lutz, A. (2010). Differential effects on pain intensity and unpleasantness of two meditation practices. *Emotion, 10*(1), 65–71.

Peters, J. R., Smart, L. M., Eisenlohr-Moul, T. A., Geiger, P. J., Smith, G. T., & Baer, R. A. (2015). Anger rumination as a mediator of the relationship between mindfulness and aggression: The utility of a multidimensional mindfulness model. *Journal of Clinical Psychology, 71*(9), 871–884.

Piet, J., Fjorback, L., & Santorelli, S. (2016). What is required to teach mindfulness effectively in MBSR and MBCT? In E. Shonin, W. Van Gordon, & M. D. Griffiths (Eds.), *Mindfulness and Buddhist-derived approaches in mental health and addiction* (pp. 61–83). Cham, Switzerland: Springer International.

Plante, T. G. (2016). Principles of incorporating spirituality into professional clinical practice. *Practice Innovations, 1*(4), 276–281.

Quaglia, J. T., Brown, K. W., Lindsay, E. K., Creswell, J. D., & Goodman, R. J. (2015). From conceptualization to operationalization of mindfulness. In K. W. Brown, J. D. Creswell, & R. M. Ryan (Eds.), *Handbook of mindfulness: Theory, research, and practice* (pp. 151–170). New York: Guilford Press.

Radin, R. M., Epel, E., Daubenmier, J., Moran, P., Schleicher, S., Kristeller, J., et al. (2020). Do stress eating or compulsive eating influence metabolic health in a mindfulness-based weight loss intervention? *Health Psychology, 39*(2), 147–158.

Raffone, A., Srinivasan, N., & Barendregt, H. P. (2014). Attention, consciousness and mindfulness in meditation. In N. N. Singh (Ed.), *Psychology of meditation* (pp. 147–166). Hauppauge, NY: Nova Science.

Ricard, M. (2006). *Happiness: A guide to developing life's most important skill.* New York: Little, Brown.

Riley, K. E., & Kalichman, S. (2015). Mindfulness-based stress reduction for people living with HIV/AIDS: Preliminary review of

intervention trial methodologies and findings. *Health Psychology Review, 9*(2), 224–243.
Robins, C. J., Keng, S. L., Ekblad, A. G., & Brantley, J. G. (2012). Effects of mindfulness-based stress reduction on emotional experience and expression: A randomized controlled trial. *Journal of Clinical Psychology, 68*(1), 117–131.
Robinson, A. H., & Safer, D. L. (2012). Moderators of dialectical behavior therapy for binge eating disorder: Results from a randomized controlled trial. *International Journal of Eating Disorders, 45*(4), 597–602.
Roemer, L., Fuchs, C., & Orsillo, S. M. (2014). Incorporating mindfulness and acceptance-based strategies in the behavioral treatment of generalized anxiety disorder. In R. A. Baer (Ed.), *Mindfulness-based treatment approaches: Clinician's guide to evidence base and applications* (2nd ed., pp. 95–118). San Diego, CA: Elsevier Academic Press.
Roos, C. R., Bowen, S., & Witkiewitz, K. (2017). Baseline patterns of substance use disorder severity and depression and anxiety symptoms moderate the efficacy of mindfulness-based relapse prevention. *Journal of Consulting and Clinical Psychology, 85*(11), 1041–1051.
Rubin, J. B. (1996). *Psychotherapy and Buddhism: Toward an integration.* New York: Plenum Press.
Rubin, J. B. (2016). Meditative psychoanalysis. *American Journal of Psychoanalysis, 76*(1), 54–70.
Rush, S. E., & Sharma, M. (2017). Mindfulness-based stress reduction as a stress management intervention for cancer care: A systematic review. *Journal of Evidence-Based Complementary and Alternative Medicine, 22,* 348–359.
Sagula, D., & Rice, K. G. (2004). The effectiveness of mindfulness training on the grieving process and emotional well-being of chronic pain patients. *Journal of Clinical Psychology in Medical Settings, 11*(4), 333–342.
Sahdra, B. K., MacLean, K. A., Ferrer, E., Shaver, P. R., Rosenberg, E. L., Jacobs, T. L., et al. (2011). Enhanced response inhibition during intensive meditation training predicts improvements in self-reported adaptive socioemotional functioning. *Emotion, 11*(2), 299–312.
Salmon, P., Santorelli, S., Sephton, S., & Kabat-Zinn, J. (2009). Intervention elements promoting adherence to mindfulness-based stress reduction (MBSR) programs in a clinical behavioral medicine setting. In S. A. Shumaker, J. K. Ockene, & K. A. Riekert (Eds.), *Handbook of health and behavior change* (3rd ed., pp. 271–286). New York: Springer.
Salzberg, S. (1999). *Voices of insight.* Boston: Shambhala.
Sanada, K., Alda Díez, M., Salas Valero, M., Pérez-Yus, M. C., Demarzo, M. M. P., Montero-Marín, J., et al. (2017). Effects of mindfulness-based interventions on biomarkers in healthy and cancer populations: A systematic review. *BMC Complementary and Alternative Medicine, 17*(1), 125.
Schroevers, M. J., Tovote, K. A., Snippe, E., & Fleer, J. (2016). Group and individual mindfulness-based cognitive therapy (mbct) are both effective: A pilot randomized controlled trial in depressed people with a somatic disease. *Mindfulness, 7(*6), 1339–1346.
Schumer, M. C., Lindsay, E. K., & Creswell, J. D. (2018). Brief mindfulness training for negative affectivity: A systematic review and meta-analysis. *Journal of Consulting and Clinical Psychology, 86*(7), 569–583.
Schutte, N. S., & Malouff, J. M. (2014). A meta-analytic review of the effects of mindfulness meditation on telomerase activity. *Psychoneuroendocrinology, 42,* 45–48.
Segal, Z. V., Anderson, A. K., Gulamani, T., Dinh Williams, L.-A., Desormeau, P., Ferguson, A., et al. (2019). Practice of therapy acquired regulatory skills and depressive elapse/recurrence prophylaxis following cognitive therapy or mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 87*(2), 161–170.
Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2013). *Mindfulness-based cognitive therapy for depression* (2nd ed.). New York: Guilford Press. (Original work published 2002)
Segall, S. R. (2005). Mindfulness and self-development in psychotherapy. *Journal of Transpersonal Psychology, 37*(2), 143–163.
Shapiro, S. L., & Carlson, L. E. (2017). What is mindfulness? In S. L. Shapiro & L. E. Carlson (Eds.), *The art and science of mindfulness: Integrating mindfulness into psychology and the helping professions* (2nd ed., pp. 9–20). Washington, DC: American Psychological Association.
Shapiro, S. L., & Schwartz, G. E. (2000). The role of intention in self-regulation: Toward intentional systemic mindfulness. In M. Boekaerts, P. R. Pintrich, & M. Zeidner (Eds.), *Handbook of self-regulation* (pp. 253–273). San Diego, CA: Academic Press.

Shapiro, S. L., Schwartz, G. E., & Bonner, G. (1998). Effects of mindfulness-based stress reduction on medical and premedical students. *Journal of Behavioral Medicine, 21*(6), 581–599.

Shapiro, S., Siegel, R., & Neff, K. D. (2018). Paradoxes of mindfulness. *Mindfulness, 9,* 1693–1701.

Shi, Z., & MacBeth, A. (2017). The effectiveness of mindfulness-based interventions on maternal perinatal mental health outcomes: A systematic review. *Mindfulness, 8*(4), 823–847.

Shiyko, M. P., Hallinan, S., & Naito, T. (2017). Effects of mindfulness training on posttraumatic growth: A systematic review and meta-analysis. *Mindfulness, 8*(4), 848–858.

Shoham, A., Goldstein, P., Oren, R., Spivak, D., & Bernstein, A. (2017). Decentering in the process of cultivating mindfulness: An experience-sampling study in time and context. *Journal of Consulting and Clinical Psychology, 85*(2), 123–134.

Shonin, E., Van Gordon, W., & Griffiths, M. D. (2014). The emerging role of Buddhism in clinical psychology: Toward effective integration. *Psychology of Religion and Spirituality, 6*(2), 123–137.

Shonin, E., Van Gordon, W., & Singh, N. N. (2015). Mindfulness and Buddhist practice. In E. Shonin, W. Van Gordon, & N. N. Singh (Eds.), *Buddhist foundations of mindfulness* (pp. 1–6). Cham, Switzerland: Springer International.

Shonin, E., Van Gordon, W., Slade, K., & Griffiths, M. D. (2013). Mindfulness and other Buddhist-derived interventions in correctional settings: A systematic review. *Aggression and Violent Behavior, 18*(3), 365–372.

Siegel, R. D. (2010). *The mindfulness solution. Everyday practices for everyday solutions.* New York: Guilford Press.

Singh, S. K., & Gorey, K. M. (2018). Relative effectiveness of mindfulness and cognitive behavioral interventions for anxiety disorders: Meta-analytic review. *Social Work in Mental Health, 16*(2), 238–251.

Smallwood, J., & Schooler, J. W. (2015). The science of mind wandering: Empirically navigating the stream of consciousness. *Annual Review of Psychology, 66,* 487–518.

Spears, C. A., Hedeker, D., Li, L., Wu, C., Anderson, N. K., Houchins, S. C., et al. (2017). Mechanisms underlying mindfulness-based addiction treatment versus cognitive-behavioral therapy and usual care for smoking cessation. *Journal of Consulting and Clinical Psychology, 85,* 1029–1040.

Springer, J. M. (2012). Acceptance and commitment therapy: Part of the "third wave" in the behavioral tradition. *Journal of Mental Health Counseling, 34*(3), 205–212.

Tang, Y.-Y., Tang, R., & Posner, M. I. (2016). Mindfulness meditation improves emotion regulation and reduces drug abuse. *Drug and Alcohol Dependence, 163*(Suppl 1), S13–S18.

Tapper, K. (2018). Mindfulness and craving: Effects and mechanisms. *Clinical Psychology Review, 59,* 101–117.

Teasdale, J. D. (1999). Metacognition, mindfulness, and the modification of mood disorders. *Clinical Psychology and Psychotherapy, 6,* 146–155.

Teasdale, J. D., Segal, Z. V., Williams, J. M. G., Ridgeway, V. A., Soulsby, J. M., & Lau, M. A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 68*(4), 615–623.

Thompson, J. M., Waelde, L. C., Tisza, K., & Spiegel, D. (2016). Hypnosis and mindfulness: Experiential and neurophysiological relationships. In A. Raz & M. Lifshitz (Eds.), *Hypnosis and meditation: Towards an integrative science of conscious planes* (pp. 129–142). New York: Oxford University Press.

Trammel, R. C. (2017). Tracing the roots of mindfulness: Transcendence in Buddhism and Christianity. *Journal of Religion and Spirituality in Social Work: Social Thought, 36*(3), 367–383.

Vago, D. R., & Zeidan, F. (2016). The brain on silent: Mind wandering, mindful awareness, and states of mental tranquility. *Annals of the New York Academy of Sciences, 1373*(1), 96–113.

Van Dam, N. T., van Vugt, M. K., Vago, D. R., Schmalzl, L., Saron, C. D., Olendzki, A., et al. (2018). Mind the hype: A critical evaluation and prescriptive agenda for research on mindfulness and meditation. *Perspectives on Psychological Science, 13*(1), 36–61.

van de Weijer-Bergsma, E., Formsma, A. R., Bruin, E. I., & Bögels, S. M. (2012). The effectiveness of mindfulness training on behavioral problems and attentional functioning in adolescents with ADHD. *Journal of Child and Family Studies, 21*(5), 775–787.

Veehof, M. M., Trompetter, H. R., Bohlmeijer, E. T., & Schreurs, K. M. G. (2016). Acceptance- and mindfulness-based interventions for the treatment of chronic pain: A meta-analytic review. *Cognitive Behaviour Therapy, 45*(1), 5–31.

Vieten, C., Wahbeh, H., Cahn, B. R., MacLean, K., Estrada, M., Mills, P., et al. (2018). Future directions in meditation research: Recommendations for expanding the field of contemplative science. *PLOS ONE, 13*(11).

Visted, E., Vøllestad, J., Nielsen, M. B., & Nielsen, G. H. (2015). The impact of group-based mindfulness training on self-reported mindfulness: A systematic review and meta-analysis. *Mindfulness, 6*(3), 501–522.

Vøllestad, J., Nielsen, M. B., & Nielsen, G. H. (2012). Mindfulness- and acceptance-based interventions for anxiety disorders: A systematic review and meta-analysis. *British Journal of Clinical Psychology, 51,* 239–260.

Walach, H., Buchheld, N., Buttenmüller, V., Kleinknecht, N., & Schmidt, S. (2006). Measuring mindfulness—The Freiburg Mindfulness Inventory (FMI). *Personality and Individual Differences, 40*(8), 1543–1555.

Walach, H., Schmidt, S., & Jonas, W. B. (Eds.). (2013). *Neuroscience, consciousness and spirituality.* New York: Springer.

Wallace, B. A. (2006). *The attention revolution: Unlocking the power of the focused mind.* Ithaca, NY: Snow Lion.

Walsh, R. (1999). *Essential spirituality.* New York: Wiley.

Walsh, R., & Shapiro, S. (2006). The meeting of meditative disciplines and Western psychology. *American Psychologist, 61*(3), 1–13.

Wang, Y.-Y., Li, X.-H., Zheng, W., Xu, Z.-Y., Ng, C. H., Ungvari, G. S., et al. (2018). Mindfulness-based interventions for major depressive disorder: A comprehensive meta-analysis of randomized controlled trials. *Journal of Affective Disorders, 229,* 429–436.

Wilson, A. D., Roos, C. R., Robinson, C. S., Stein, E. R., Manuel, J. A., Enkema, M. C., et al. (2017). Mindfulness-based interventions for addictive behaviors: Implementation issues on the road ahead. *Psychology of Addictive Behaviors, 31*(8), 888–896.

Witkiewitz, K., Lustyk, M. K. B., & Bowen, S. (2013). Retraining the addicted brain: A review of hypothesized neurobiological mechanisms of mindfulness-based relapse prevention. *Psychology of Addictive Behaviors, 27*(2), 351–365.

Wnuk, S. M., Du, C. T., Exan, J., Wallwork, A., Warwick, K., Tremblay, L., et al. (2017). Mindfulness-based eating and awareness training for post-bariatric surgery patients: A feasibility pilot study. *Mindfulness, 9,* 949–960.

Wolever, R. Q., Bobinet, K. J., McCabe, K., Mackenzie, E. R., Fekete, E., Kusnick, C. A., et al. (2012). Effective and viable mind–body stress reduction in the workplace: A randomized controlled trial. *Journal of Occupational Health Psychology, 17*(2), 246–258.

Woolfolk, R. L. (1984). Self-control meditation and the treatment of chronic anger. In D. H. Shapiro & R. N. Walsh (Eds.), *Meditation: Classic and contemporary perspectives* (pp. 550–554). New York: Aldine.

Xu, J., Vik, A., Groote, I. R., Lagopoulos, J., Holen, A., Ellingsen, Ø., et al. (2014). Nondirective meditation activates default mode network and areas associated with memory retrieval and emotional processing. *Frontiers in Human Neuroscience, 8,* 86.

Zainal, N. Z., Booth, S., & Huppert, F. A. (2013). The efficacy of mindfulness-based stress reduction on mental health of breast cancer patients: A meta-analysis. *Psycho-Oncology, 22*(7), 1457–1465.

Zeidan, F., & Vago, D. R. (2016). Mindfulness meditation-based pain relief: A mechanistic account. *Annals of the New York Academy of Sciences, 1373*(1), 114–127.

Zhang, J., Xu, R., Wang, B., & Wang, J. (2016). Effects of mindfulness-based therapy for patients with breast cancer: A systematic review and meta-analysis. *Complementary Therapies In Medicine, 26,* 1–10.

CHAPTER 15

Qigong Therapy for Stress Management

Kevin W Chen

HISTORY OF THE METHOD

The Term

Qigong (pronounced "chi kung") is a general term for a variety of traditional Chinese mind–body exercises and healing practices. The word *Qigong* is a combination of two Chinese ideograms: *qi,* meaning "breathing," "vital energy," and *gong,* meaning "skill," "work," and "achievement." So Qigong is an integrated mind–body practice for mastering vital energy, or cultivation of vital energy. Only with the accumulation of time and effort can the skill of cultivating vital energy be developed.

Qigong, just like acupuncture, herbal medicine, massage, and cupping, forms an integral component of traditional Chinese medicine (TCM). TCM posits the existence of a subtle energy (*qi*) circulating throughout the entire human mind and body. When it is strengthened or balanced, it can improve health and ward off or slow down the progress of disease. The concept of bioenergy could also be found in other cultures, such as *ki* in Japan, *prana* in India, and *mana* in Hawaii and the Philippines.

There are more than 1,000 registered Qigong schools or forms in contemporary China, and many more have existed throughout history. As a result, there is no consistent definition for Qigong in the academy, not even within the Qigong community. According to the textbook used in colleges of Chinese medicine, *Qigong* refers to "the skill of mind-body exercises that integrate the body posture, breathing and mind adjustments into one" (Liu & Chen, 2010, p. 15).

As Qigong gains more and more popularity, it is very important for a beginner to learn how to determine the authenticity and applicability of a specific Qigong to meet their individual needs and goals. In addition, there are, unavoidably, less qualified Qigong practitioners who would pose as Qigong masters or qualified teachers but who, in reality, have not mastered the true arts of *qi* cultivation themselves. It is important to be able to differentiate those who are qualified to teach Qigong from those who are not.

A Long History

Qigong is the key component of TCM. It has a history longer than Chinese medicine itself. *Qigong* has existed as a term and practice for a long time, but it did not become popular until the 1950s, when scientists and health care professionals started studying its effectiveness in health care and healing. At that time, the term *Qigong* gained public acceptance over other traditional and more abstruse terms, such as *tuina* (exhalation and inhalation, 吐纳), *daoyin* (guiding and conducting exercise, 导引), *xiulian* (cultivating and practicing 修炼), *jingzuo* (sitting meditation 静坐), and *xingqi* (circulating *qi*, 行气). Reference to the Qigong-type practice exists in more than 3,000 years' worth of written records, which can be approximately divided into four main periods in development (Yang, 1991):

1. The period of *united heaven, earth, and man* (before 206 B.C.E.): This period was marked in history by the famous book *Yi Jing* ("Book of Changes") introduced in 1122 B.C.E. It presented *qi* as the concepts of natural energies and the integration of Heaven, Earth, and Man. The first step in Qigong development involved the study of relationship between these three natural powers. Ancient Chinese practiced Qigong to better understand the relationship between humans and nature. Some developed an inner vision through Qigong meditation that enabled them to view and draw accurately the body meridian system (the channel of *qi* flow) 3,000 years ago. Acupuncturists still use this system today.

During the Zhou dynasty (1122–934 B.C.E.), *Dao De Jing* mentioned certain breathing techniques and established the philosophical foundation for Qigong and Daoism. He stressed that the way to obtain health was "to concentrate on qi and achieve softness." Later, the historical record *Shi Ji* (770–221 B.C.E.) had a more complete description of methods of breath training.

The most detailed description of *qi* meditation was recorded in a jade pendant inscription (around 600 B.C.E.) (see Figure 15.1) that included 36 ancient Chinese words laying out the detailed process of *qi* meditation and *qi* movement in the body as follows:

> When you breathe deeply to a degree, internally refined qi will gather. When the refined qi is fully accumulated, it expands and descends downward (Dantian in the lower abdomen area). When the refined qi is reinforced to a certain level it begins to rise up (along the spinal cord) to the head, and then falls down (to the Dantian) again. The internal qi then circulates around the Ren-Du meridians during your daily practice. The universal qi becomes integrated. It comes in through the top of the head, with the earth qi, which enters the body through the feet. Thus, you will be healthier and more alive while you follow it, and more prone to the effects of aging and death when you don't.

2. The period of *Qigong practice mixed with religion* (206 B.C.E.–500 C.E.): During the Han dynasty (206 B.C.E.), Buddhism meditation methods were imported from India, which brought Qigong practice and meditation into the religious Qigong era.

Not long after Buddhism was introduced into China, Master Zhang Daoling combined the traditional Daoist (Taoism) principles with Buddhism, and created a religion called Dao Jiao. Many meditation methods were a combination of the principles and training methods from both Buddhism and Daoism. As the development and cultivation of Qigong required a lot of time commitment and spiritual guidance away from the secular society, religious temples provided such a physical and spiritual environment. Meanwhile, Qigong practice might actually have helped religious practitioners to more effectively reach higher spiritual levels and deeper understanding of the religious

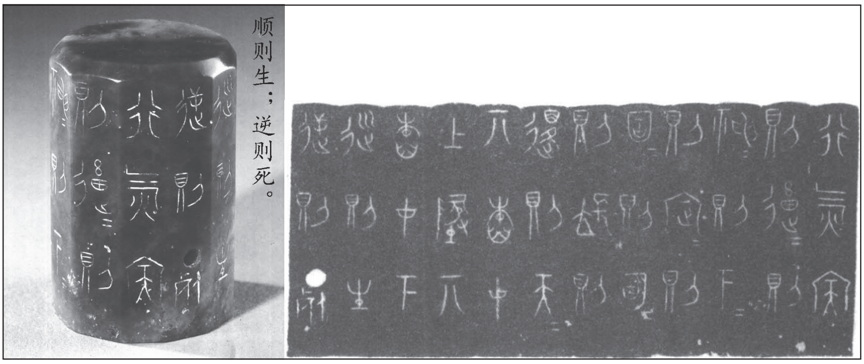


FIGURE 15.1. The script of a jade pendant (ca. 600 B.C.E.) described the process of Qigong exercises.

scriptures through deep meditation. These integrative results made Qigong a popular practice among religions. However, Qigong itself is not a religion.

At this stage, Qigong had shown its characteristics as an exercise of one's consciousness or intention. The founder of Zen (a Buddhist Qigong tradition), Bodhidharma (?–528 C.E.), spent 9 years in a deep meditation state, facing a rocky wall in a cave behind the Shaolin Temple. As a Chinese saying stated, "Bodhidharma brought no word with him from the west but the skill to work with mind or consciousness." Bodhidharma was also considered a significant contributor to the creation and development of martial-art Qigong, which is the Qigong style he taught in the Shaolin Temple.

3. The period of *development with appearance of martial-art Qigong* (500 C.E.–1950s): In the Liang dynasty (502–557 C.E.), people discovered that, in addition to improving health, Qigong training could also increase physical strength and be used for self-defense and fighting. The application of Qigong to martial arts made Qigong more popular in society. Many Qigong forms were created for this purpose. In parallel, both religious and medical Qigong also developed rapidly during this period.

With the modern convenience of travel and communications, Chinese Qigong training was mixed with Qigong practices from India, Japan, and many other countries from 1911 on. However, Qigong was still kept in the form of private transmission from generation to generation until very recently.

4. The period of *modern Qigong research and massive (large-scale promotion and practice) Qigong movement* (1950s–present): Qigong gradually emerged from the traditional secret transmission into a common practice by the public and a subject of scientific research when more and more Qigong practitioners came out (of traditional private practice) to openly show the public their amazing healing abilities and to teach the public to practice Qigong. During the Cultural Revolution (1966–1976), Qigong was considered a "pseudoscience" or "idealism," and its practice was forbidden in China. In 1978, many scientists and practitioners wished to rebuild Qigong as one of the effective healing methods in health care and tried to use advanced scientific measurements and technology to prove that Qigong is not purely "idealism" or psychological effects (placebo) but rather an objective life phenomenon with measurable processes. Since then, thousands of scientific research studies were conducted and published in China, and millions of people have started practicing Qigong for the purpose of health and healing. By this time, more and more people started Qigong practice not for the purpose of spirituality, but for better health.

Different Traditions with Different Focuses

There have been thousands of forms and schools of Qigong in Chinese history. Various methods can be used to classify these Qigong forms. Although Qigong was well known for its healing and health potential, most Qigong forms were not created for the purpose of healing but for the cultivation of mind and spirituality. Currently, the Chinese government classifies Qigong into two categories: health Qigong and medical Qigong, ignoring its tradition of spirituality.

Historically, Qigong can be divided into five major traditions: Confucianist, Buddhist, Taoist, medical, and martial arts. Each discipline has its own set of goals, methods, and forms. Following are brief descriptions of these five major traditions in Chinese Qigong development (Liu & Chen, 2010).

Confucian Qigong, developed by the followers of Confucius, is designed to attain a higher moral character and intelligence, focusing on education and moral cultivation. This form of meditation had relatively few followers historically, but it reflects one of the Qigong essences—an exercise of consciousness. Typical forms in this tradition are "listening to breathing" (a meditation form with a focus on slow breathing and attention to listening to the breathing) and the "sitting and forgetting self" exercise (a meditation form similar to mindfulness, with empty mind as the optimal goal; see Kristeller, Chapter 14, this volume).

Buddhist Qigong aims at liberating the mind, emphasizing the cultivation of virtue and enlightening wisdom, and it considers the human body to be just a "stinking bag" holding the honorable spirit. With this philosophy, a pure Buddhist Qigong form would not have much reason to build a healthy body; however, health and healing could be a side effect of developing a positive mind and relaxation state. The famous Buddhist Qigong traditions include Zen (Chan), Mi (Tibetan), and Tiantai (Vipassana).

Daoist (or Taoist) Qigong emphasizes the preservation of the physical body first, and then the higher level of virtue and spiritual cultivation. Most Daoist Qigong consisted of training of both the body (*qi,* or "Ming" = life) and the spirit (*yi,* or "Xing" = spirituality). Some encouraged body, or *qi,* cultivation before spiritual, or *yi,* cultivation, whereas others considered spiritual training more important than body training. Because Daoism put emphasis on the current life and explored the techniques of preserving long life, many Daoist Qigong masters lived extremely long lives. Biographies of Daoist Qigong masters in history show that most of them lived to be more than 90 years old at a time when the average life expectancy was around 40 (Zhang, Gu, Wang, Liu, & Song, 1994). The famous Daoist Qigong traditions include Taiji, Danding (including Nei Dan Qigong), Jianxian (Sward Qigong), Fulu (*fu* implies symbol), Xuanzeng, and others.

Medical Qigong refers to those Qigong forms created or practiced for healing of self or others. It emphasizes how to use human vital energy (*qi*) to help eliminate imbalances and disharmonies, which are considered the root causes for many illnesses and diseases, and how to prevent them. It was influenced greatly by Daoist philosophy but developed independently, mostly by TCM practitioners. Historically, the most famous TCM doctors were also good Qigong practitioners. Medical Qigong teaches medical practitioners how to use the inner *qi* in a dynamic way for diagnosis, healing and preventing diseases (discussed further later in the chapter). Today, medical Qigong is still a standard course in many schools of Chinese medicine. The typical medical Qigong forms include Five-Animal-Acts Qigong (by Hua Tuo), Six-Words-Knack Qigong (Liu & Chen, 2010), Brighten-Eye Qigong, and Taiji Five-Element Qigong (He, 2003).

Martial-arts Qigong tends to train the practitioner for self-defense, protecting and preparing the body to better endure sword cuts or sharp weapons or attacks by a powerful punch or kick. Such methods included Iron Shirt and Golden-Bell Qigong. It also trains the body to deliver powerful blows that were enhanced with *qi*, such as the Burning-Palm or Iron-Palm methods. We can see demonstrations of martial arts Qigong from time to time. However, many martial-art *Qigong* practitioners have died prematurely due to overexerting their body limits or to an imbalance of inner *qi* (Zhang et al., 1994). For example, Bruce Lee was a well-known contemporary martial-art Qigong practitioner, but he died in his mid-30s.

In short, all forms of Qigong are not the same. As Qigong gains popularity, it is very important for a beginner to know which Qigong form is most appropriate for him or her. The above classification may also help beginners learn how to determine the authenticity of a Qigong form, as there are many sham or fake Qigong "masters" in the market who don't know what Qigong really is and, therefore, tend to provide incomplete or misleading instruction.

Most Qigong practice may involve a combination of elements such as relaxation, breathing work, guided imagery, slow movement, biofeedback, tranquil state, mindfulness meditation (see Kristeller, Chapter 14, this volume), and mind–body integration. Qigong practice is said to help relax and develop a balance between the body, mind, and spirit, while relaxation and balance are the basic ingredients of good health (Zhang et al. 1994; Liu & Chen, 2010).

Various forms of Qigong have reported some health benefits, but not all forms were designed for the purpose of health care or healing; only medical Qigong makes treating illness or curing disease its major purpose.

DESCRIPTION OF THE METHOD

Basic Theory

According to TCM, good health is a result of a free-flowing, well-balanced *qi* (energy) system, whereas sickness or the experience of pain is the result of *qi* blockage or an unbalanced energy in the body. Acupuncture and Qigong share the same *qi*-blood flow and meridian theory. The meridian system charts the major channels of *qi* flow. The difference between acupuncture and Qigong is that acupuncture uses external force (needles or pressure stimulation) to help the *qi* flow and balance, whereas Qigong uses mostly internal force (*qi* cultivation and self-practice) to help smooth the *qi* flow and break the *qi* blockages.

In addition to the concept of *qi*, the concept of *yi* (mind or intention) also plays a key role in Qigong practice and Qigong healing. TCM believes that *qi* tends to follow the *yi* ("*qi shui yi xing*"). When a person is under stress or in a complex of random thoughts for a long time, his or her *qi* cannot flow smoothly or normally, and he or she will soon experience *qi* imbalance (emotional disturbance) or *qi* blockage (psychosomatic symptoms). Therefore, having the consciousness focus on one thing or nothing (mindfulness) is the key to training in Qigong practice. This focus is called the *empty mind without desire* state. Cultivating the mind can be practiced at any time without Qigong or meditation.

Medical Qigong or Qigong Therapy

Medical Qigong refers to the Qigong forms or therapies used by medical practitioners with an emphasis on using vital energy (*qi*) to assist with diagnosis and to control and prevent illness and disease. Medical Qigong, or Qigong therapy, consists of both internal Qigong exercise (self-practice) and external *qi* healing (through clinician's involvement).

Although Qigong is a self-training method, the emission of *qi* (or external Qigong therapy) has always been part of the medical Qigong practice in the attempt to help others regain their health, similar to the practice of *reiki* and of therapeutic touch in the United States. Therefore, the difference between internal training and external Qigong therapy in the history and development of medical Qigong must be examined.

Internal Qigong training is the major component of medical Qigong and refers to the self-practice of Qigong forms. There are three major categories of the Qigong forms: (1) movement (active) Qigong (*dong gong*), (2) "standing pole" (*zhuang gong*), and (3) static forms or meditation (*jing gong*). Movement Qigong uses guided physical movements or gestures to help practitioners concentrate and induce the *qi* energy flow in the body. Earlier forms of *taiji quan* may be considered as typical movement Qigong. Static Qigong is mostly meditation, which may include relaxation, breathing manipulation, mindful meditation, guided imagery, incantation, seal palm symbols, and mindfulness practice. The main purpose of static Qigong is the training of intention power or consciousness stability when cultivating *qi* energy. It was said that the intention or consciousness, once well trained, will lead *qi* flow in the body and direct it to where it is needed, breaking through the blocked area. Blocked *qi* is considered the origin of many illnesses and diseases. The standing pole is a form between movement and static Qigong, which usually starts with a standing position; as the *qi* is cultivated or moved, various spontaneous movements will follow. The magnitude or degree of the movement varies and sometimes may even be greater than during movement Qigong.

External Qigong therapy (EQT) refers to the process by which a *Qigong* practitioner directs or emits his or her *qi* to help break *qi* blockages in others and induce the sick *qi* out of the body so as to relieve pain or balance the *qi* flow in the body and get rid of disease. EQT can be practiced through the use of *qi* (emitting vital energy) or *yi* (the consciousness or intentional therapy) or a combination of the two techniques (most commonly used.) Most schools of medical Qigong will teach both techniques. Many Qigong clinics in Chinese hospitals provide EQT.

Although the physical nature of *qi* remains as yet unproven, there are intriguing reports that suggest the possibility of physical, biophysical, and/or biochemical alterations induced by external *qi* therapy or "*qi*-emission" (Chen, 2004). For example, *qi*-emission by Qigong masters has been reported to be associated with significant structural changes in aqueous solutions, to alter the phase behavior of dipalmitoyl phosphatidyl choline (DPPC) liposomes, to enable the growth of Fab protein crystals (Yan et al., 1999), to inhibit tumor growth in mice (Chen, Li, Liu, & He, 1997), to change the conformation of such biomolecules as polyglutamic acid, polylysine, and metallothionein (Chu, He, Zhou, & Chen, 1998), and to reduce phosphorylation of a cell-free preparation (Muehsami, Markow, & Muehsami, 1994). Thus there is a small but growing body of scientific evidence that suggests the physical existence of *qi*, as well as the healing power of Qigong therapy (Chen, 2004; C. W. Wang et al., 2014; Wayne et al., 2018; Zou et al., 2019).

Qigong Meditation versus Other Meditations

The term *meditation* refers to a mental state or the continued thought or reflection or contemplation on sacred or solemn subjects. For many, troubled by the stresses of modern life, it is an age-old method, particularly in the East, that is turned to for help in coping with anxiety and in finding a deeper meaning in life. Mindfulness meditation has become popular in the United States and has been integrated with cognitive therapy to help improve outcomes in the treatment of anxiety (Chen et al., 2012) and depression (Segal et al., 2002).

Meditation is one of the most important components in Qigong practice. In some sense, all meditations could be called Qigong in China, but practice of Qigong is more than meditation. In order to differentiate medical Qigong from other meditation forms, I discuss how medical Qigong is thought to work toward health and healing from a TCM perspective. Some of these explanations have not yet been scientifically verified.

1. In terms of practice, most meditations involve a single practice or focus—for example, concentration on one's breathing. Medical Qigong consists of three major components for stimulating a balanced *qi* flow and a tranquil state, as described above: movement Qigong, static Qigong (meditation), and standing pole. Standing meditation accompanied by spontaneous movement is actually a powerful means of *qi* generation and *qi* balance by nature.

2. *Qi* plays an important role in Qigong meditation, but not necessarily in most other meditations. Qigong uses the *qi*-flow meridian theory. One of the healing mechanisms of Qigong, from a TCM perspective, is the belief that motivated *qi* (vital energy moving more powerfully after Qigong practice) strikes against sick locations and removes destructive blocks to allow healthy *qi* to flow. There is experiential evidence that Qigong practitioners may clearly feel pain or soreness during practice as a result of this *qi*-striking process.

3. Although most meditation requires the role of mind or intention, Qigong meditation puts the role of *yi* (mind or intention) above anything else in two distinct ways: (1) the induced state of "empty mind without desire" and emphasis on tranquility helps a practitioner to calm conflicting emotions and resolve mental disturbances, and (2) the resulting tranquil state strengthens a practitioner's power of intention. The *qi*-meditation introduced here to treat stress engages these components through a guided imagery process.

4. In addition to the relaxation response common to other meditations, medical Qigong meditation generates healing results by rapidly uncovering the body's self-healing potential through modulated breathing, deep tranquility, intentional *qi*-induction, guided imagery, and an enhanced mind–body integration. The increased immune functions that are facilitated by these processes result in a strengthened self-repair and self-regeneration capability. Sancier (1996, p. 44) concluded, after reviewing the literature on medical applications of Qigong, that "Qigong enables the body to heal itself."

EFFECTS ON STRESS AND STRESS-RELATED PROBLEMS

Although most Qigong forms were not designed for healing nor for stress management, Qigong practice is said to be very effective in ameliorating the effects of many stress-related problems, such as hypertension, allergy, asthma, back or neck pain, premenstrual syndrome, insomnia, headache, depression, anxiety, and addiction. It is said that Qigong works by increasing the practitioners' self-healing capabilities, including immune functions, self-recovery capability, and self-regeneration capability (He, 2003; Liu & Chen, 2010; Sancier, 1996; C. W. Wang et al., 2014). The potential applications of Qigong in health and healing are too numerous to list, and to some extent, the potential of Qigong practice challenges our current understanding of life and health in general. Most practitioners credit their Qigong practice with improving daily life in many ways, including:

- A more relaxed, harmonious state of mind and body.
- A noticeable reduction in prior ailments and a reduction in feelings of stress.
- An increased resistance to illness.
- A heightened sensitivity to the body's internal organs, with a developed ability to regulate their own health and vitality.

There are quite a few clinical studies in the United States assessing the effectiveness of Qigong and tai chi (a movement form of Qigong) in stress-related problems, and many more studies published in China and Japan may be included in English literature. Table 15.1, later in the chapter, summarizes the main findings of Qigong effects on some major stress-related problems.

Qigong Exercise Directly Reduces Stress

Research has suggested that 80% of the patients in a primary care setting show evidence of significant psychosocial distress (Sobel, 1995). Many common health problems are related to stress, such as headache, hypertension, overweight, lower back pain, asthma, allergy, insomnia, and so on. Therefore, an effective strategy to manage stress will greatly reduce health problems and increase the quality of life. The relaxation and energetic response produced by Qigong exercise has been well documented in clinical studies of health benefits of Qigong (Liu et al. 2005; Sandlund & Norlander, 2000; Wall, 2005; C. W. Wang et al., 2014; Zou, Sasaki, Wei, et al., 2018; Zou, Yeung, Quan, Boyden, & Wang, 2018).

Sandlund and Norlander (2000) reviewed more than 20 studies published in the 1990s about the effects of tai chi chuan (a slow-movement form of Qigong) on stress response and well-being and concluded that, although the slow-movement tai chi may not achieve aerobic fitness, it could enhance flexibility and overall psychological well-being. Tai chi exercises lead to an improvement in mood. Sandlund and Norlander (2000) concluded that all studies on the benefits of tai chi have revealed positive results and that tai chi was an effective way to reduce stress. C. W. Wang et al. (2014) did a systematic review of stress-related outcomes of Qigong exercise. Among the seven randomized controlled trials (RCTs) they reviewed with stress and psychological measures, two RCTs suggested that Qigong exercise immediately relieved anxiety among healthy adults, compared with lecture attendance and structured movements only. Four RCTs suggested Qigong exercise relieved anxiety, and three RCTs suggested that Qigong exercise reduced stress among healthy participants following 1–3 months of Qigong practice, compared with wait-list controls.

Recently Zou, Sasaki, Wei, et al. (2018) reviewed 17 medium- to high-quality RCTs with a focus on the heart rate variability (HRV) parameters and found that tai chi showed significantly beneficial effects on HRV parameters and that stress reduction may be attributed to sympathetic–vagal balance modulated by mind–body exercises such as tai chi. Mind–body exercise such as tai chi could be an alternative method for stress management among people who live under high stress or with negative emotions.

We also found laboratory studies of animals with external *qi* therapy for stress reduction. Zhang, Wang, Yan, Ge, and Zhou (1993) applied external *qi* therapy in cold-stressed mice. The mice were divided into three groups: the normal control, the stress control (mice underwent 5 minutes of 1^{o} C per day cold-stress exposure for 8 days), and the Qigong groups (in addition to cold stress, mice received emitted *qi* 30 minutes/day for 8 days). Then, the thymuses, spleens, and brains were dissected, and T cell and B cell proliferation and the RDCC activities of K cells were used to investigate the immune adjusting effect of the emitted *qi* on cold-stressed mice. They found that the T cell and B cell proliferation rate and the activities of K cells of the stressed mice were significantly lower than those of the nonstressed controls; however, the T and B cell proliferation and the activities of K cell in the Qigong group were significantly higher than those of the stressed mice in the control group ($p < .01$). Although the immune organs (thymus and spleen) of the stressed mice were atrophied more significantly than in the nonstressed mice ($p < .01$), the external *qi* treated group had no thymus and spleen injuries, in comparison with the control group ($p < .01$).

Qigong Therapy for Stress-Related Symptoms

There are many studies focusing on the effectiveness of Qigong therapy for stress-related symptoms or problems. Table 15.1 lists the major health conditions for which Qigong therapy (including tai chi) has shown efficacy in controlled trials, supported mostly by systematic review with meta-analysis. Increased numbers of clinical studies suggest that sufficient evidence supports the conclusion that Qigong and tai chi practice have significant benefits for many stress-related health conditions, such as arthritis (Chen & Liu, 2004; Chen, Hunt, Campbell, Peill, & Reid, 2016), cancer care (Wayne et al., 2018), anxiety (Chen et al., 2012), cardiovascular problems (Lan, Chen, Wong, & Lai, 2013; T. Liu, Chan, Liu, & Taylor-Piliae, 2018), chronic obstructive pulmonary disease (COPD; Chen et al. 2016; S. J. Liu, Ren, Wang, Wei, & Zou, 2018), depressive symptoms (Guo, Kong, & Zhang, 2019), diabetes (Zhou, Zhou, et al., 2019), fibromyalgia (Lynch, Sawynok, Hiew, & Marcon, 2012; Sawynok & Lynch, 2014, 2017), heart failure (Chen et al., 2016), hypertension (Xiong, Wang, Li, & Zhang, 2015), chronic pain (Chen et al., 2016), stroke (Zou, Sasaki, Zeng, et al., 2018), and substance abuse (Zhu et al., 2016, Zhu, Dai, et al., 2018).

In the college textbook *Chinese Medical Qigong* (Liu & Chen, 2010), a total of 19 conditions with clinical applications of Qigong therapy are listed in Part 3 of the book, including: pulmonary tuberculosis, hypertension, coronary artery disease, peptic ulcer, chronic gastritis, chronic liver diseases, chronic nephritis, diabetes mellitus, obesity, menopause syndrome, impotence, chronic fatigue syndrome, depression, insomnia, tumors and cancer, lower back pain and leg pain, cervical spondylosis, myopia, and glaucoma. In each clinical section, there is an outline of how this condition is defined by TCM, what the ancient Qigong therapies for treating the condition were, what the modern Qigong therapies look like, the selected Qigong forms by TCM syndrome differentiation (more specific prescription), and the precaution in clinical application. Please keep in mind that this is actually what a TCM doctor learns in his or her regular medical training.

How Qigong Works for Stress-Related Problems

The mechanism by which Qigong works to achieve health benefits and reduce stress is still the subject of scientific exploration. Various Qigong healers may have different explanations of how Qigong works for healing. Qigong's main therapeutic properties may well lie in its regulation of the respiratory system, metabolism, activity of the cerebral cortex and the central nervous system, and the cardiovascular system, as well as its effect in correcting abnormal reactions of the organs, massaging effect on the organs of the abdominal cavity, and its effect on self-control over the physical functions of one's body. Recent studies suggest that Qigong practice may be associated with increased HRV (Zou Sasaki, Wei, et al., 2018), reduced cortisol level (Zeng, et al. 2014), change of electroencephalogram (EEG) pattern (Qin, Jin, Lin, & Hermanowicz, 2009), increased

TABLE 15.1. Conditions Showing Efficacy for Qigong Therapy (Including Tai-chi) in Controlled Trials

Condition	Studies	Note
Anxiety	Chen et al. 2012; Sharma & Harder (2015); Wang et al. (2013); Wang et al. (2014); Wang et al. (2013), Wang et al. (2014)	Reviews
Arthritis	Chen & Liu (2004); Chen, Hunt, Campbell, Peill, & Reid (2016); Lauche, Langhorst, Dobos, & Cramer (2013); Marks (2017); Wang et al. (2009); Ye et al. (2014)	Reviews
Asthma	Chang, Yang, Chen, & Chiang (2008); Lin et al. (2017)	
Cancer care	Chen & Yeung (2002); Chen et al. (2013); Klein (2017); Klein, Schneider, & Rhoads (2016); Pan et al. (2015); VanVu, Molassiotis, Ching, & Le (2017); Winters-Stone (2014); Wayne et al. (2018); Zeng et al. (2014); Zeng, Xie, & Cheng (2019)	Mostly reviews
Cardiovascular problems	Lan, Chen, Wong, & Lai (2013); T. Liu, Chan, Liu, & Taylor-Piliae (2018); Nery et al. (2014); Ng et al. (2012); Yeh, Wang, Wayne, & Phillips (2009); Wang, Collet, & Lau (2004) Wang, Pi, et al. (2016)	Mostly reviews
COPD	Chan, Lee, Suen, & Tam (2011); Chen et al. (2016); Liu, Ren, Wang, Wei, & Zou (2018); Ngai, Jones, & Tam (2016); Polkey et al. (2018); Wu et al. (2018)	Reviews
Depression	Chi, Jordan-Marsh, Guo, Xie, & Bai (2013); Guo, Kong, & Zhang 2019; Liu et al. (2015); Oh, Choi, Inamori, Rosenthal, & Yeung (2013); Wang et al. (2013); Wang et al. (2014); Zou, Yeung, Li, et al. (2018)	Reviews
Diabetes	Chao, Wang, Dong, & Ding (2018); Chen, Liu, Zhang, & Ling (2009); Putiri, Close, Lilly, Guillaume, & Sun (2017); Song et al. (2018); Zhou, Zhou, et al. 2019	Reviews
Fibromyalgia	Chan et al. (2012); Lauche, Cramer, Häuser, Dobos, & Langhorst (2013); Lynch, Sawynok, Hiew, & Marcon (2012); Sawynok & Lynch (2014, 2017); Wang et al. (2018)	Reviews
Heart failure	Chen et al. (2016); Gu et al. (2017); Lan et al. (2013); Pan, Choi, Inamori, Rosenthal, & Yeung (2013); Ren et al. (2017)	Mostly reviews
Hypertension	Lee, Lee, Kim, & Moon (2003); Lee, Pittler, Guo, & Ernst (2007); Mayer (1999); Xiong, Wang, Li, & Zhang (2015)	Reviews
Insomnia	Irwin, Olmstead, & Motivala (2008); Raman, Zhang, Minichiello, D'Ambrosio, & Wang (2013); Wang et al. (2016)	Reviews
Pain	Bai et al. (2015); Chen et al. (2016); Girard & Girard (2019); Hall et al. (2017); Kong et al. (2016); Zou, Yeung, Quan, et al. (2018); Zou et al. (2019); Zhang et al. (2019)	Reviews
PTSD	Kim, Schneider, Kravitz, Mermier, & Burge (2013); Niles et al. (2016)	Reviews
Stress	Sandlund & Norlander (2000); Wall (2005); Wang et al. (2014); Zou, Sasaki, Wei, et al. (2018)	Reviews
Stroke	Lauche et al. (2017); Lyu et al. (2018); Zou, Sasaki, Zeng, et al. (2018)	Reviews
Substance dependence	Li, Chen, & Mo (2002); Oh & Kim (2016); Zgierska et al. (2009); Zhu et al. (2016), Zhu, Dai, et al. (2018)	Reviews

melatonin production (Liou et al. 2010), and strengthening immune functions (Morgan, Irwin, Chung, & Wang, 2014; Zeng et al., 2014).

According to TCM and available scientific literature in Chinese, Qigong may work to benefit the practitioners through the following possible paths.

1. Motivated *qi* (vital energy flowing more strongly in meridian after Qigong practice) strikes against sick locations. According to TCM, good health is a result of a free-flowing and well-balanced *qi* (energy) system, whereas sickness and the experience of pain are the results of *qi* blockage or unbalanced energy in certain areas of the body. *Qi* imbalance is the precursor of any physical illness. One way to stay healthy and function well is to perform Qigong exercises in order to keep the *qi* flowing smoothly in the body so that each cell in the body gets a constant supply of vital energy. Once the supply of *qi* to the cells becomes blocked, blood flow to that area will change, and disease or pain may occur. One possible mechanism of Qigong therapy for pain relief and symptom reduction is through motivating *qi* and energy within the body, breaking the *qi* blockage and balancing the energy system. Therefore, it is common for Qigong practitioners to report more serious symptoms or pain on a temporary basis due to *qi* striking against sick locations. These pains or symptoms will go away completely with continued practice.

2. Cultivation of a mindfulness mindset and the emphasis on "empty mind without desire," "follow the float," and "let go" in Qigong practice may help the practitioners strengthen their consciousness and intention, release suppressed emotions, and resolve mental disturbances. It is said that Qigong training of the mind or intention helps the practitioner to release him- or herself from the socialized self or consciousness (the source of all stress) and return him or her to the original self without social pressure or natural consciousness. Many chronic diseases of unknown origin may well be related to mental disturbances, social pressures, or emotional twists. Qigong practice may lead to the release of these emotional disturbances, which may have been the sources of many chronic diseases. It is a common phenomenon for Qigong practitioners to tear up, cry, or laugh during Qigong practice and then feel complete relief after the practice.

In addition, the increased power of focused mind and energy level of intention could also work with the human body by reaching the sick locations (similar to the mechanism of guided imagery), changing the features of illness, and turning the abnormal into normal (He, 2003; Chen, 2004). There is scientific evidence in parapsychological studies to suggest such human consciousness or intention potential, even though we still do not know exactly how it works.

3. An individual can rapidly reveal or uncover the body's potential self-healing capability with quality Qigong practice. This includes increasing the immune functions, the self-repair capability, and the self-regeneration capability (Chen & Yeung, 2002; He, 2003). For example, the relaxation and tranquility status achieved during Qigong practice may relieve stress, build up vital energy, and rapidly increase the immune function (Lee et al., 2001). There is some scientific evidence to connect relaxation and guided imagery with increased immune function, while connecting stress and depression with malfunction of the immune system. We have also observed that many patients have completely recovered from multiple complicated diseases or symptoms with Qigong therapy in a short period of time without any medications, or have reproduced some lost physical parts, such as new hair growth and new teeth growth among middle-aged adults and seniors. In this sense, Qigong therapy has challenged current medical practice of depending on the use of symptom-relieving pharmaceutical drugs.

SIDE EFFECTS AND CONTRAINDICATIONS

Many people think there are no side effects at all for Qigong practice. However, strictly speaking, some side effects could occur from practicing Qigong if the practitioner is not prepared for it. If one practices Qigong a lot on a daily basis without appropriate understanding of the natural process of Qigong practice, or without the right instructor to assist him or her, one may run into some *qi* phenomena, or side effects. These effects occur more likely among those who learn Qigong from books or tapes without proper guidance or from an unqualified instructor. However, if one understands what kinds of effects may be experienced in intensive Qigong practice, there is no danger at all to practicing Qigong continuously and safely.

Generally speaking, there are three common types of response during intensive Qigong practice, which have been mislabeled as potential "side effects" or reported as "Qigong deviations" in the literature:

1. *Appearance of new symptoms or increased severity of old symptoms.* This is typical for medical Qigong practice when *qi* strikes against an illness location or blocked area. As stated previously, TCM believes that sickness or experience of pain is the result of *qi* blockage or unbalanced energy in the body. One mechanism of Qigong therapy for pain relief and symptom reduction is through motivating *qi* and energy within the body, breaking the *qi* blockage and balancing the energy system. Therefore, it is common for beginning Qigong practitioners to report more serious symptoms or pain temporarily when *qi* is striking against sick locations. These pains or symptoms will go away completely with continuous practice (He, 2003; Liu & Chen, 2010). For example, a person with a history of arthritis of the knee might feel increased pain at the knee for a period of time after intensive Qigong practice. If he or she stops practicing at that moment, the pain will continue for a while; if he or she continues practicing Qigong, the *qi* strikes against the blockage (arthritis), and the pain will completely disappear forever.

2. *Pain or soreness through the back and neck area, sometimes feeling like the pressure of a mountain over the head.* This occurs when strengthened *qi* starts flowing upward in the "Du" meridian (along the spinal cord) and strikes through the "Jiaji gateway" and "Yuzeng gateway" on the lower back and on the back of the neck, the common *qi* striking points. Under close supervision and with proper understanding, the practitioner can easily correct this problem by continuing practice and getting over the blockage to reach a higher level of cultivation. However, uninformed practitioners may consider that there is something wrong in their practice and stop the Qigong practice altogether at this stage. Many psychiatrists who did not practice Qigong would also consider this a psychosomatic symptom due to Qigong practice (Shan et al., 1989).

3. *Hallucination and Qigong psychosis.* It is quite common for the advanced Qigong practitioner to experience some hallucination or illusion during Qigong practice, such as photism or phonism (Ng, 1999; Lee, 2000). However, as long as the practitioners do not believe what they see or hear, do not sustain these hallucinations, and continue their practice, these hallucinations will eventually go away. There is no danger of becoming disoriented as long as the practitioner knows in advance that this might happen. Unfortunately, most Qigong instructors have not had such an experience themselves, and they do not tell their students about this potential. Therefore, incorrect practice with the misunderstanding of Qigong hallucinations, or practice with strong intention or inappropriate purposes (such as intention to communicate with higher being, develop supernatural

ability, or reach self-completion as some sham Qigong practitioners claim), may lead to various forms of psychosis or even schizophrenia or other types of abnormal behaviors (Shan et al., 1989; Lee, 2000).

As discussed earlier, most of the so-called side effects of Qigong practice are the natural pathways of *qi* cultivation and can be overcome by continuous practice. The key to dealing with this issue is for the practitioner to be informed in advance, to try not to believe that the hallucination in practice is real, and to continue practicing Qigong until the stage is over. However, the unfortunate end result for those who do not understand these phenomena would be for them to discontinue practice when it is most needed.

Qigong practice can be improved rapidly and successfully if one can avail him- or herself of the natural flow of *qi* and adhere to the notion of "a life of simplicity and empty mind without desires" during practice. It is crucial to understand the potential discomfort from *qi* striking the gateways and to increase practice when *qi* strikes against illness locations with more symptoms. When confronted with Qigong hallucinations during practice, one should simply ignore them all and continue to practice without interruptions. This way one will never deviate or lose control. Otherwise, the Qigong practice may become harmful to the practitioner or others if he or she practices with wants and desires, seeking a quick fix or immediate success, or practices under the misguided influence of a "sham" Qigong master.

THE MANUAL FOR TRAINING

There are a thousand different Qigong forms or methods, most of them having the basic elements of relaxation, tranquility, and breathing works, and, therefore, they may have some therapeutic effect for stress management. It will be difficult to introduce a specific method as a representative of Qigong without misleading the student. Although there are many different elements and components in different traditions of Qigong, there are some common methods— three basic adjustments before undertaking Qigong meditation and the key components during Qigong practice, which can be found in most Qigong traditions. I describe these, then briefly introduce the taiji five-element medical qigong forms as an example of the medical Qigong practice.

The Three Adjustments: Basics of Qigong

1. Adjust your body position by finding a comfortable position to sit, lie down, or stand (as instructed by the specific form) and relax completely in the position you choose. Sometimes, you can put a gentle smile on your face and keep your eyes and mouth lightly closed if it is a meditation form.
2. Adjust your breathing. Different breathing techniques may be used by different Qigong forms or traditions. In the Taoist Qigong that we teach at World Institute for Self-Healing, we ask the students to pay special attention to the area around the abdomen (lower Dantian), begin abdominal breathing, and breathe softly, evenly, deeply, and slowly.
3. Adjust your mind or mental state by clearing your mind of all thoughts and letting your awareness stay at the lower Dantian area, relaxed and peaceful. In the optimal Qigong state, your mind will always be on one thing (the instructed subject) or nothing (mindfulness state).

It is said that the advanced Qigong meditation is the integration of the three adjustments into one without any distinction. The oneness state is also called the *Qigong state,* a higher level consciousness state that needs to be verified or assessed by more reliable scientific measurements.

Key Components of Qigong Exercise

The essential points during Qigong practice include: (1) relaxation (completely relaxing both physically and psychologically, without falling asleep); (2) tranquility (a mind state of quiet and concentration, ignoring surroundings, focusing on one thing or nothing); and (3) naturalness (following the natural way both physically and emotionally, without wants and intents).

Most Qigong forms also emphasize a state of empty mind without desire during practice (these are mostly subjective reports and very difficult to measure). Only those who can minimize desire and intention during practice can achieve the optimal status for both mind and body. Practicing Qigong with strong purpose or desire is considered a deviation from the tradition of cultivating *qi* essence. Researchers and practitioners have noticed that there are many stages of achievement and development during the Qigong practice and cultivation, and there are significant differences among various schools or traditions on the designated stages. For example, the complete Qigong cultivation process for the Daoist tradition may include the following stages: cultivating sperm or blood (for men or women, respectively) into *qi,* cultivating *qi* into spirit (*shen*), cultivating spirit return to emptiness (*xu*), and cultivating emptiness up to Dao. However, medical Qigong for the purpose of healing focuses only on the first two stages of this process.

Example: Relaxation through Smiling Exercise

The following introduces a special Qigong form for relaxation. Note that instead of simply teaching the practitioner to relax physically, this Qigong exercise achieves the relaxation state by inducing internal smiling or psychological/spiritual relaxation, as the psychological relaxation is more important in the mind–body–spirit integration.

Here are the instructions for this internal smiling exercise:

> "This exercise is designed to help you quickly recover from fatigue, reduce stress, increase your energy and efficacy. You can do this at anytime and anywhere.
>
> "You can do this exercise by standing, sitting, or lying down. Whichever position you select, make sure you are comfortable and relaxed. Clear your mind of all thoughts.
>
> "Lightly close your eyes. Breathe naturally, paying special attention to the area around your abdomen. Let your breathing be soft, even, deep, and slow.
>
> "Now you are relaxed. As you breathe naturally and relax, focus your attention on the area between your two eyebrows, visualize a small smiling face in between your two eyebrows, smiling, and smiling happily at you. The smiling face begins to grow bigger and bigger until it covers your entire head.
>
> "The smiling face continues to expand; and it covers your entire chest. Your entire chest becomes a smiling face. It continues to expand. . . . Your entire body becomes a smiling face. Your organs, your nervous system, all your cells and pores are smiling . . . smiling . . . and smiling.
>
> "The smiling face continues to expand from your body outward to become a very large smiling face covering the entire room, and then, to the surrounding

environment. It continues to expand, smiling and expanding, smiling and expanding until it covers the earth. The entire earth and everything in it turns into a huge smiling face, smiling, smiling, and smiling!

"The huge smiling face continues to expand, reaching the entire universe and the entire universe becomes an endless smiling face.

"Everything is smiling.

"Everything becomes the smiling face.

"There is no one, not you, or anyone else—just smiles . . .

"There are no desires, no wants; no demands—just smiles . . .

"There is no disease, no illness, no disasters—just smiles . . .

"There is no heaven, no earth, just smiles, smiles, and smiles. . . .

"Continue to visualize and feel the smiling face for a few minutes, or as long as you want.

"Now, you can finish the exercise by letting the smiling face gradually become smaller and smaller and come back into your body, then reduce it to the size of an egg and send it to your lower Dantian. Visualize the small smiling face in your lower Dantian for a minute as you continue to breathe naturally.

"You feel completely relaxed, content, and worry-free.

"Briskly rub your palms together until they feel warm, and then use your palms to stroke your face downward a few times. Your smiling exercise is now completed."

How to Get Formal Qigong Training

There are a number of Qigong schools in China that offer formal training for health and healing with the possibility of becoming a certified instructor. For example, inner-nourishing Qigong (*nei-yang gong*) is offered at the Hebei Qigong Hospital (Beidaihe, Hebei); Guolin New Qigong (a.k.a. anticancer Qigong) is offered in Beijing, Shanghai, and other major cities of China for those who want to become Qigong instructors. A few universities of Chinese medicine offer the formal training of medical Qigong with the credential of master's degree. Beijing University of Chinese Medicine is the only institution that offers doctoral training (PhD degree) in medical Qigong. However, there is no unified national license system in China to certify practitioners to become Qigong instructors or healers.

Finding a knowledgeable and reliable Qigong teacher is the best way to start your Qigong training. However, it is still a great challenge to find a knowledgeable Qigong teacher today, either in China or in the United States, as there is no authentic and nationwide certification system for Qigong healer or instructor. There are a few Qigong schools in the United States that offer certification programs, or even master's or doctoral degrees in medical Qigong, but their true credential and marketing values need to be tested and recognized by the health care system. Even in China there are more self-named (fake?) Qigong masters who lack lineage or traditional training than there are true Qigong masters with reliable lineage. It is up to the students to differentiate the true masters from the fake ones.

CASE EXAMPLES

Following are a few cases of patients who benefited significantly from Taiji Five-Element Medical Qigong (TFMQ).

Case 1

A 58-year-old Caucasian male in New Jersey suffered from a series of chronic conditions, some of which might have been directly related to psychological distress. These conditions include a high prostate-specific antigen (PSA) level (but not a confirmed cancer), atrial septal defect, asthma, allergies, hypertension, multiple injuries from an auto accident, and edema in both legs. He started the practice of TFMQ and Qigong therapy due to his concern about his elevated PSA level (around 11) and the family history of cancer. The Qigong therapy was introduced to him through an intensive Qigong workshop, which involved the training and practice of gathering *qi*, magnifying *qi* energy and using it for self-healing with visualization and guided imagery, and supervised energetic fasting. The patient practiced Qigong more than 4 hours a day during intensive training and about 1 to 2 hours daily thereafter. About 10 sessions of external *qi* healing were performed by a Qigong master for his pain relief and systematic adjustment. After the intensive workshop and Qigong therapy, plus 36 days of *Bigu* (energetic fasting), the patient discontinued all medications (eight in total) and lost 35 pounds in 2 months. His blood pressure dropped from 220/110 with medication to 120/75 without medication in 2 weeks; his pulse rate dropped from 88 beats per minute (bpm) resting to 68 bpm in the mornings and 55 bpm in the evening; the edema in his legs went away; symptoms of asthma and allergies disappeared; PSA level dropped from 11 to 4 (normal), all without any medications (Chen & Turner, 2004).

Although the patient's original motive for participating in Qigong practice was to lower his elevated PSA level, the outcome of the intensive Qigong training was really much better than he had expected—a complete recovery from multiple chronic symptoms. One of the unique characteristics of this case is that most of his previous physical conditions were not curable by the known conventional medications or healing procedures, yet he achieved simultaneous recovery with Qigong therapy in a very short period and has stayed medication-free for more than 2 years. None of his doctors can offer an explanation about the source of the simultaneous recovery from the multiple symptoms in such a short term.

Case 2

A female patient from Japan in her 30s weighed 215 pounds and also suffered from heart disease, diabetes, high cholesterol, arthritis, and major depression. She had lost her job, her boyfriend, and her self-confidence at the time when she was introduced to Qigong therapy in 1999. She came to China to try the TFMQ system with traditional Bigu technique (energetic fasting). After she participated in Qigong intensively for 40 days (6+ hours a day), including 25 days of *Bigu,* not only did her weight drop to 143 pounds, but also her depression and other chronic conditions disappeared completely. Now she enjoys a renewed physical and emotional well-being, is back in the work force, and is living a normal life.

Case 3

An Asian male in his 40s, a practitioner of Chinese medicine and acupuncture in southern California, had suffered from diabetes, headache, and back pain for quite some time. He had not practiced Qigong previously. Within 2 weeks of attending the TFMQ training class, he reported regaining the energy level that he had had about 15 years previously.

TABLE 15.2. Patient Self-Reported Improvement in 2 Weeks of Qigong Training

Conditions	Before Qigong class	After Qigong class
Blood sugar	293	110 (normal)
Weight	192 pounds	172 pounds
Blood pressure	135/95	118/78
Arthritis pain	Finger joints	Disappeared after 3 days
Sleep time	8–9 hours	5 hours
Other pains I	Liver, stomach, and colon for over 2 years	Recovered after 2 days
Other pains II	Headache, backache, and pain in right waist area	Recovered after 3 days
Other	Bleeding teeth and baldness	Stopped bleeding and numerous new hair growth after class

With practicing only 1½ hours of Qigong and having deep sleep for 4 hours a day, he said, “The benefits for my health are unmistakable. “As a doctor himself, he kept very detailed records of his health conditions during the Qigong class period, which involved intensive Qigong meditation with group *qi* adjustment (a healer placed and monitored the *qi* field in the classroom). Table 15.2 presents some observations of his personal conditions before and after Qigong training.

SUMMARY AND CONCLUSIONS

Qigong, an ancient Chinese energy healing practice that combines the elements of relaxation, breathing work (see van Dixhoorn, Chapter 12, this volume), guided imagery, biofeedback (see Thompson and Thompson, Chapter 9, and Lehrer, Chapter 10, this volume), mindfulness meditation (see Kristeller, Chapter 14, this volume), mind manipulation, and mind–body integration, is a very effective option for stress management. As the case studies demonstrated, Qigong therapy is more than just for stress management but can be applied to a variety of health conditions to achieve simultaneous recovery. These results present a great challenge to current medicine and health care.

Qigong therapy might be an effective tool in stress management. What is more important is that, accompanying Qigong therapy, there is a complete self-healing-based medical system to promote self-care and to find the healer within. The TFMQ described in this chapter is simple and easy to learn, yet powerful and effective for helping to relieve symptoms, improve recovery, and gain general health resilience. The factors contributing to its high efficacy of healing include a drug-free approach to avoid the side effects of pharmaceuticals, use of innate self-healing power, following TCM philosophies, and a healthy lifestyle.

Although Qigong has a lot of common elements with other effective stress reduction methods, it encompasses more than other therapies in terms of its concept and application of human vital energy (*qi*) and intention power (*yi*), which manage stress both physically and psychologically, and achieves a result beyond just reduction of stress. We hope that more scientists and health care professionals will become interested in Qigong research and applications of Qigong therapy.

ACKNOWLEDGMENTS

I would like to thank the Qigong Institute (*www.Qigonginstitute.org*) in California for its support of my Chinese literature review with a small research grant. I also appreciate the help of Mrs. Denise Dehnbostel, Ms. Joy Staller, and Dr. Yinong Chong in editing and commenting on an earlier version of this chapter.

REFERENCES

Bai, Z., Guan, Z., Fan, Y., Liu, C., Yang, K., Ma, B., et al. (2015). The effects of Qigong for adults with chronic pain: Systematic review and meta-analysis. *American Journal of Chinese Medicine, 43*(8), 1525–1539.

Chan, A. W., Lee, A., Suen, L. K., & Tam, W. W. (2011). Tai chi Qigong improves lung functions and activity tolerance in COPD clients: A single blind, randomized controlled trial. *Complementary Therapies in Medicine, 19*(1), 3–11.

Chan, C. L., Wang, C. W., Ho, R. T., Ng, S. M., Ziea, E. T., Wong, V. T. (2012). Qigong exercise for the treatment of fibromyalgia: A systematic review of randomized controlled trials. *Journal of Alternative and Complementary Medicine, 8*(7), 641–646.

Chang, Y. F., Yang, Y. H., Chen, C. C., & Chiang, B. L. (2008). Tai Chi Chuan training improves the pulmonary function of asthmatic children. *Journal of Microbiology, Immunology, and Infection, 41,* 88–95.

Chao, M., Wang, C., Dong, X., & Ding, M. (2018, July 5). The effects of tai chi on type 2 diabetes mellitus: A meta-analysis. *Journal of Diabetes Research.* [Epub ahead of print]

Chen, K. (2004). An analytic review of studies on measuring effects of external qi in China. *Alternative Therapies in Health and Medicine, 10*(4), 38–50.

Chen, K. W., Beger, C. C., Manheimer, E., Forde, D., Magidson, J., Dachman, L., et al. (2012). Meditative therapies for reducing anxiety: A systematic review and meta-analysis of randomized controlled trials. *Depression and Anxiety, 29,* 545–562.

Chen, K., & Liu, T. J. (2004). Effects of Qigong therapy on arthritis: A review and report of a pilot trial. *Medical Paradigm, 1*(1), 36–48.

Chen, K. W., Liu, T. J., Zhang, H. B., & Ling, Z. P. (2009). An analytical review of the Chinese literature on Qigong therapy for diabetes mellitus. *American Journal of Chinese Medicine, 37*(3), 439–457.

Chen, K., & Turner, F. D. (2004). A case study of simultaneous recovery from multiple physical symptoms with medical Qigong therapy. *Journal of Alternative and Complementary Medicine, 10*(1), 159–162.

Chen, K., & Yeung, R. (2002). Exploratory studies of Qigong therapy for cancer in China. *Integrative Cancer Therapies, 1*(4), 345–370.

Chen, X., Li, Y., Liu, G., & He, B. (1997). The inhibitory effects of Chinese Taijing Five-Element Qigong on transplanted hepatocarcinoma in mice. *Asian Medicine, 11,* 36–38.

Chen, Y. W., Hunt, M. A., Campbell, K. L., Peill, K., & Reid, W. D. (2016). The effect of Tai Chi on four chronic conditions—cancer, osteoarthritis, heart failure and chronic obstructive pulmonary disease: A systematic review and meta-analyses. *British Journal of Sports Medicine, 50*(7), 397–407.

Chen, Z., Meng, Z., Milbury, K., Bei, W., Zhang, Y., Thornton, B., et al. (2013). Qigong improves quality of life in women undergoing radiotherapy for breast cancer: Results of a randomized controlled trial. *Cancer, 119,* 1690–1698.

Chi, I., Jordan-Marsh, M., Guo, M., Xie, B., & Bai, Z. (2013). Tai chi and reduction of depressive symptoms for older adults: A meta-analysis of randomized trials. *Geriatrics and Gerontology International, 13,* 3–12.

Chu, D. Y., He, W. G., Zhou, Y. F., & Chen, B. C. (1998). The effect of Chinese Qigong on the conformation of biomolecule. *Chinese Journal of Somatic Science, 8*(4), 155–159.

Girard, J., & Girard, A. (2019). The effects of qigong on neck pain: A systematic review. *Complementary Therapies in Clinical Practice, 34,* 23–29.

Gu, Q., Wu, S. J., Zheng, Y., Zhang, Y., Liu, C., Hou, J. C., et al. (2017). Tai Chi exercise for patients with chronic heart failure: A meta-analysis of randomized controlled trials. *American Journal of Physical Medicine and Rehabilitation, 96,* 706–716.

Guo, L., Kong, Z., & Zhang, Y. (2019). Qigong-based therapy for treating adults with major depressive disorder: A meta-analysis of randomized controlled trials. *International Journal of Environmental Research and Public Health, 16*(5), 826.

Hall, A., Copsey, B., Richmond, H., Thompson, J., Ferreira, M., Latimer, J., et al. (2017). Effectiveness of tai chi for chronic musculoskeletal pain conditions: Updated systematic review and meta-analysis. *Physical Therapy, 97,* 227–238.

He, B. H. (2003). *Taiji five-element self-recovery system: Comprehensive Qigong therapy.* Hong Kong: Qingwen.

Irwin, M. R., Olmstead, R., & Motivala, S. J. (2008). Improving sleep quality in older adults with moderate sleep complaints: A randomized controlled trial of Tai Chi Chih. *Sleep, 31*(7), 1001–1008.

Kim, S. H., Schneider, S. M., Kravitz, L., Mermier, C., & Burge, M. R. (2013). Mind–body practices for posttraumatic stress disorder. *Journal of Investigational Medicine, 61,* 827–834.

Klein, P. (2017). Qigong in cancer care: Theory, evidence-base, and practice. *Medicines (Basel), 4*(1), 2.

Klein, P. J., Schneider, R., & Rhoads, C. J. (2016). Qigong in cancer care: A systematic review and construct analysis of effective Qigong therapy. *Supportive Care in Cancer, 24*(7), 3209–3222.

Kong, L. J., Lauche, R., Klose, P., Bu, J. H., Yang, X. C., Guo, C. Q., et al. (2016). Tai Chi for chronic pain conditions: A systematic review and meta-analysis of randomized controlled trials. *Scientific Reports, 29*(6), 25325.

Lan, C., Chen, S. Y., Wong, M. K., & Lai, J. S. (2013). Tai chi chuan exercise for patients with cardiovascular disease. *Evidence-Based Complementary and Alternative Medicine,* 983208.

Lauche, R., Cramer, H., Häuser, W., Dobos, G., & Langhorst, J. (2013). A systematic review and meta-analysis of qigong for the fibromyalgia syndrome. *Evidence-Based Complementary and Alternative Medicine,* 635182.

Lauche, R., Langhorst, J., Dobos, G., & Cramer, H. (2013). A systematic review and meta-analysis of Tai Chi for osteoarthritis of the knee. *Complementary Therapies in Medicine, 21,* 396–406.

Lauche, R., Peng, W., Ferguson, C., Cramer, H., Frawley, J., Adams, J., et al. (2017). Efficacy of Tai Chi and qigong for the prevention of stroke and stroke risk factors: A systematic review with meta-analysis. *Medicine (Baltimore), 96*(45), 8517.

Lee, M. S., Huh, H. J., Hong, S. S., Jang, H. S., Ryu, H., Lee, H. S., et al. (2001). Psychoneuroimmunological effects of Qi-therapy: Preliminary study on the changes of level of anxiety, mood, cortisol and melatonin and cellular function of neutrophil and natural killer cells. *Stress and Health, 17,* 17–24.

Lee, M. S., Lee, M. S., Kim, H. J., & Moon, S. R. (2003). Qigong reduced blood pressure and catecholamine levels of patients with essential hypertension. *International Journal of Neuroscience, 113,* 1707–1717.

Lee, M. S., Pittler, M. H., Guo, R., & Ernst, E. (2007). Qigong for hypertension: A systematic review of randomized clinical trials. *Journal of Hypertension, 25,* 1525–1532.

Lee, S. (2000). Chinese hypnosis can cause Qigong induced mental disorders. *BMJ, 320*(7237), 803.

Li, M., Chen, K., & Mo, Z. X. (2002). Detoxification with qigong therapy for heroin addicts. *Alternative Therapies in Health and Medicine, 8*(1), 50–59.

Lin, H. C., Lin, H. P., Yu, H. H., Wang, L. C., Lee, J. H., Lin, Y. T., et al. (2017). Tai-Chi-Chuan exercise improves pulmonary function and decreases exhaled nitric oxide level in both asthmatic and nonasthmatic children and improves quality of life in children with asthma. *Evidence-Based Complementary and Alternative Medicine,* 6287642.

Liou, C. H., Hsieh, C. W., Hsieh, C. H., Chen, D. Y., Wang, C. H., Chen, J. H., et al. (2010). Detection of nighttime melatonin level in Chinese Original Quiet Sitting. *Journal of the Formosan Medical Association, 109*(10), 694–701.

Liu, S. J., Ren, Z., Wang, L., Wei, G. X., & Zou, L. (2018). Mind–body (Baduanjin) exercise prescription for chronic obstructive pulmonary disease: A systematic review with meta-analysis. *International Journal of Environmental Research and Public Health, 15*(9), 1830.

Liu, T., Chan, A. W., Liu, Y. H., & Taylor-Piliae, R. E. (2018). Effects of Tai Chi-based cardiac rehabilitation on aerobic endurance, psychosocial well-being, and cardiovascular risk reduction among patients with coronary heart disease: A systematic review and meta-analysis. *European Journal of Cardiovascular Nursing, 17*(4), 368–383.

Liu, T., & Chen, K. W. (Eds.). (2010). *Chinese medical Qigong*. London: Singing Dragon.

Liu, X., Clark, J., Siskind, D., Williams, G. M., Byrne, G., Yang, J. L., et al. (2015). A systematic review and meta-analysis of the effects of Qigong and Tai Chi for depressive symptoms. *Complementary Therapies in Medicine, 23,* 516–534.

Lynch, M., Sawynok, J., Hiew, C., & Marcon, D. (2012). A randomized controlled trial of qigong for fibromyalgia. *Arthritis Research and Therapy, 14*(4), R178.

Lyu, D., Lyu, X., Zhang, Y., Ren, Y., Yang, F., Zhou, L., et al. (2018). Tai Chi for stroke rehabilitation: A systematic review and meta-analysis of randomized controlled trials. *Frontiers in Physiology, 9,* 983.

Marks, R. (2017). Qigong exercise and arthritis. *Medicines (Basel), 4*(4).

Mayer, M. H. (1999). Qigong and hypertension: A critique of research. *Journal of Alternative and Complementary Medicine, 5*(4), 371–382.

Morgan, N., Irwin, M. R., Chung, M., & Wang, C. (2014). The effects of mind–body therapies on the immune system: Meta-analysis. *PLOS ONE, 9*(7), e100903.

Muehsami, D. J., Markow, M. S., & Muehsami, P. A. (1994). Effects of Qigong on cell-free myosin phosphorylation: Preliminary experiments. *Subtle Energies, 5,* 103–104.

Nery, R. M., Zanini, M., Ferrari, J. N., Silva, C. A., Farias, L. F., Comel, J. C., et al. (2014). Tai Chi Chuan for cardiac rehabilitation in patients with coronary arterial disease. *ABC Cardiology, 102*(6), 588–592.

Ng, B. Y. (1999). Qigong-induced mental disorders: A review. *Australian and New Zealand Journal of Psychiatry, 33*(2), 197–206.

Ng, S. M., Wang, C. W., Ho, R. T., Ziea, T. C., He, J., Wong, V. C., et al. (2012). Tai chi exercise for patients with heart disease: A systematic review of controlled clinical trials. *Alternative Therapies in Health and Medicine, 18*(3), 16–22.

Ngai, S. P., Jones, A. Y., & Tam, W. W. (2016). Tai Chi for chronic obstructive pulmonary disease (COPD). *Cochrane Database of Systematic Reviews*. Retrieved from *www.cochrane.org/CD009953/AIRWAYS_tai-chi-chronic-obstructive-pulmonary-disease-copd*.

Niles, B. L., Mori, D. L., Polizzi, C. P., Pless, K. A., Ledoux, A. M., & Wang, C. (2016). Feasibility, qualitative findings and satisfaction of a brief Tai Chi mind–body programme for veterans with post-traumatic stress symptoms. *BMJ Open, 6*(11), e012464.

Oh, B., Choi, S. M., Inamori, A., Rosenthal, D., & Yeung, A. (2013). Effects of qigong on depression: A systemic review. *Evidence-Based Complementary and Alternative Medicine,* 134737.

Oh, C. U., & Kim, N. C. (2016). Effects of T'ai Chi on serotonin, nicotine dependency, depression, and anger in hospitalized alcohol-dependent patients. *Journal of Alternative and Complementary Medicine, 22*(12), 957–963.

Pan, L. C. J. (2013). Effects of Tai Chi training on exercise capacity and quality of life in patients with chronic heart failure: A meta-analysis. *European Journal of Heart Failure, 15*(3), 316–323.

Pan, Y., Yang, K., Shi, X., Liang, H., Zhang, F., & Lv, Q. (2015). Tai chi chuan exercise for patients with breast cancer: A systematic review and meta-analysis. *Evidence-Based Complementary and Alternative Medicine,* 535237.

Polkey, M. I., Qiu, Z. H., Zhou, L., Zhu, M.-D., Wu, Y.-X., Chen, Y.-Y., et al. (2018). Tai Chi and pulmonary rehabilitation compared for treatment-naive patients with COPD: A randomized controlled trial. *Chest, 53*(5), 1116–1124.

Putiri, A. L., Close, J. R., Lilly, H. R., Guillaume, N., & Sun, G. C. (2017). Qigong exercises for the management of type 2 diabetes mellitus. *Medicines (Basel), 4*(3), E59.

Qin, Z., Jin, Y., Lin, S., & Hermanowicz, N. S. (2009). A forty-five year follow-up EEG study of Qigong practice. *International Journal of Neuroscience, 119*(4), 538–552.

Raman, G., Zhang, Y., Minichiello, V. J., D'Ambrosio, C. M., & Wang, C. (2013). Tai Chi improves sleep quality in healthy adults and patients with chronic conditions: A systematic review and meta-analysis. *Journal of Sleep Disorders and Therapy, 2*(6), 141.

Ren, X., Li, Y., Yang, X., Li, J., Li, H., Yuan, Z., et al. (2017). The effects of Tai Chi training in patients with heart failure: A systematic review and meta-analysis. *Frontiers in Physiology, 8,* 989.

Sancier, K. M. (1996). Medical applications of Qigong. *Alternative Therapies in Health and Medicine, 2*(1), 40–45.

Sandlund, E. S., & Norlander, T. (2000). The effects of Tai Chi Chuan relaxation and exercise on stress responses and well-being: An overview of research. *International Journal of Stress Management, 7*(2), 139–149.

Sawynok, J., & Lynch, M. (2014). Qigong and fibromyalgia: Randomized controlled trials and beyond. *Evidence-Based Complementary and Alternative Medicine,* 379715.

Sawynok, J., & Lynch, M. E. (2017). Qigong and fibromyalgia circa 2017. *Medicines (Basel), 4*(2), E37.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse.* New York: Guilford Press.

Shan, H. H., Yan, H. Q., Xu, S. H., Zhang, M. D., Yu, Y. P., Zhao, J. C., et al. (1989). Clinical phenomenology of mental disorders caused by Qigong exercise. *Chin Medicine Journal, 102*(6), 445–448.

Sharma, M., & Haider, T. (2015). Tai chi as an alternative and complimentary therapy for anxiety: A systematic review. *Evidence-Based Complementary and Alternative Medicine, 20*(2), 143–153.

Sobel, D. S. (1995). Rethinking medicine: Improving health outcomes with cost-effective psychosocial interventions. *Psychosomatic Medicine, 57*(3), 234–244.

Song, G., Chen, C., Zhang, J., Chang, L., Zhu, D., & Wang, X. (2018). Association of traditional Chinese exercises with glycemic responses in people with type 2 diabetes: A systematic review and meta-analysis of randomized controlled trials. *Journal of Sport and Health Science, 7*(4), 442–452.

VanVu, D., Molassiotis, A., Ching, S. S. Y., & Le, T. T. (2017). Effects of Qigong on symptom management in cancer patients: A systematic review. *Complementary Therapies in Clinical Practice, 29,* 111–121.

Wall, R. B. (2005). Tai Chi and mindfulness-based stress reduction in a Boston public middle school. *Journal of Pediatric Health Care, 19*(4), 230–237.

Wang, C., Schmid, C. H., Fielding, R. A., Harvey, W. F., Reid, K. F., Price, L. L., et al. (2018). Effect of tai chi versus aerobic exercise for fibromyalgia: Comparative effectiveness randomized controlled trial. *BMJ, 360,* k851.

Wang, C., Schmid, C. H., Hibberd, P. L., Kalish, R., Roubenoff, R., Rones, R., et al. (2009). Tai Chi is effective in treating knee osteoarthritis: A randomized controlled trial. *Arthritis Care and Research, 61*(11), 1545–1553.

Wang, C. C., Collet, J. P., & Lau, J. (2004). The effect of Tai Chi on health outcomes in patients with chronic conditions. *Archive of Internal Medicine, 164,* 493–501.

Wang, C. W., Chan, C. H., Ho, R. T., Chan, J. S., Ng, S. M., & Chan, C. L. (2014). Managing stress and anxiety through qigong exercise in healthy adults: A systematic review and meta-analysis of randomized controlled trials. *BMC Complementary and Alternative Medicine, 14,* 8.

Wang, C. W., Chan, C. L., Ho, R. T., Tsang, H. W., Chan, C. H., & Ng, S. M. (2013). The effect of qigong on depressive and anxiety symptoms: A systematic review and meta-analysis of randomized controlled trials. *Evidence-Based Complementary and Alternative Medicine,* 716094.

Wang, F., Eun-Kyoung, L. O., Feng, F., Vitiello, M. V., Wang, W., Benson, H., et al. (2016). The effect of meditative movement on sleep quality: A systematic review. *Sleep Medicine Reviews, 30,* 43–52.

Wang, F., Lee, E. K., Wu, T., Benson, H., Fricchione, G., Wang, W., et al. (2014). The effects of tai chi on depression, anxiety, and psychological well-being: A systematic review and meta-analysis. *International Journal of Behavioral Medicine, 21,* 605–617.

Wang, F., Man, J. K., Lee, E. K., Wu, T., Benson, H., Fricchione, G. L., et al. (2013). The effects of qigong on anxiety, depression, and psychological well-being: A systematic review and meta-analysis. *Evidence-Based Complementary and Alternative Medicine,* 152738.

Wang, J. (1993). Role of Qigong on mental health. *Proceedings of the Second World Conference of Academic Exchange on Medical Qigong,* Beijing, China.

Wang, X. Q., Pi, Y. L., Chen, P. J., Liu, Y., Wang, R., Li, X., et al. (2016). Traditional Chinese exercise for cardiovascular diseases: Systematic review and meta-analysis of randomized controlled trials. *Journal of the American Heart Association, 5*(3), e002562.

Wayne, P. M., Lee, M. S., Novakowski, J., Osypiuk, K., Ligibel, J., Carlson, L. E., et al. (2018). Tai Chi and Qigong for cancer-related symptoms and quality of life: A systematic review and meta-analysis. *Journal of Cancer Survivorship, 12*(2), 256–267.

Winters-Stone, K. (2014). Tai Ji Quan for the aging cancer survivor: Mitigating the accelerated development of disability, falls, and cardiovascular disease from cancer treatment. *Journal of Sport and Health Science, 3*(1), 52–57.

Wu, L. L., Lin, Z. K., Weng, H. D., Qi, Q. F., Lu, J., & Liu, K. X. (2018). Effectiveness of meditative movement on COPD: A systematic review and meta-analysis. *International Journal of Chronic Obstructive Pulmonary Disease, 13,* 1239–1250.

Xiong, X., Wang, P., Li, X., & Zhang, Y. (2015). Qigong for hypertension: A systematic review. *Medicine (Baltimore), 94*(1), e352.

Yan, X., Lin, H., Li, H., Traynor-Kaplan, A., Xia, Z. Q., Lu, F., et al. (1999). Structure and property changes in certain material influenced by the external Qi of Qigong. *Material Research Innovation, 2,* 349–359.

Yang, J. M. (1991). *Qigong for arthritis: The Chinese way of healing and prevention.* Jamaica Plain, MA: YMAA Publication Center.

Ye, J., Cai, S., Zhong, W., Cai, S., & Zheng, Q. (2014). Effects of tai chi for patients with knee osteoarthritis: A systematic review. *Journal of Physical Therapy Science, 26,* 1133–1137.

Yeh, G. Y., Wang, C., Wayne, P. M., & Phillips, R. (2009). Tai chi exercise for patients with cardiovascular conditions and risk factors: A systematic review. *Journal of Cardiopulmonary Rehabilitation and Prevention, 29,* 152–160.

Zeng, Y., Luo, T., Xie, H., Huang, M., & Cheng, A. S. (2014). Health benefits of qigong or tai chi for cancer patients: A systematic review and meta-analyses. *Complementary Therapies in Medicine, 22*(1), 173–186.

Zeng, Y., Xie, X., & Cheng, A. S. K. (2019). Qigong or Tai Chi in cancer care: An updated systematic review and meta-analysis. *Current Oncology Reports, 21*(6), 48.

Zgierska, A., Rabago, D., Chawla, N., Kushner, K., Koehler, R., & Marlatt, A. (2009). Mindfulness meditation for substance use disorders: A systematic review. *Substance Abuse, 30,* 266–294.

Zhang, L., Wang, L., Yan, Y. Z., Ge, D. Y., & Zhou, Y. (1993). Adjusting effect of emitted qi on the immune function of cold-stressed mice. *Proceedings of the Second World Conference of Academic Exchange of Medical Qigong*, Beijing, China.

Zhang, Y., Loprinzi, P. D., Yang, L., Liu, J., Liu, S., & Zou, L. (2019). The beneficial effects of traditional Chinese exercises for adults with low back pain: A meta-analysis of randomized controlled trials. *Medicina (Kaunas), 55*(5), E118.

Zhang, Z. H., Gu, P. D., Wang, S. L., Liu, Z. B., & Song, T. B. (Eds.). (1994). *The encyclopedia of Chinese Qigong.* Beijing: United Publisher.

Zhou, Z., Zhou, R., Zhu, Y., Li, K., Luo, Y., Zhang, Z., et al. (2019). Effects of tai chi on physiology, balance and quality of life in patients with type 2 diabetes: A systematic review and meta-analysis. *Journal of Rehabilitation Medicine, 51*(6), 405–417.

Zhu, D., Dai, G., Xu, D., Xu, X., Geng, J., Zhu, W., et al. (2018). Long-term effects of tai chi intervention on sleep and mental health of female individuals with dependence on amphetamine-type stimulants. *Frontiers in Psychology, 9,* 1476.

Zhu, D., Xu, D., Dai, G., Wang, F., Xu, X., & Zhou, D. (2016). Beneficial effects of Tai Chi for amphetamine-type stimulant dependence: A pilot study. *American Journal of Drug and Alcohol Abuse, 42*(4), 469–478.

Zou, L., Sasaki, J. E., Wei, G. X., Huang, T., Yeung, A. S., Neto, O. B., et al. (2018). Effects of mind-body exercises (Tai Chi/Yoga) on heart rate variability parameters and perceived stress: A systematic review with meta-analysis of randomized controlled trials. *Journal of Clinical Medicine, 7*(11), E404.

Zou, L., Sasaki, J. E., Zeng, N., Wang, C., & Sun, L. (2018). A systematic review with meta-analysis of mindful exercises on rehabilitative outcomes among poststroke patients. *Archives of Physical Medicine and Rehabilitation, 99*(11), 2355–2364.

Zou, L., Yeung, A., Li, C., Wei, G. X., Chen, K. W., Kinser, P. A., et al. (2018). Effects of meditative movements on major depressive disorder: A systematic review and meta-analysis of randomized controlled trials. *Journal of Clinical Medicine, 7*(8), E195.

Zou, L., Yeung, A., Quan, X., Boyden, S. D., & Wang, H. (2018). A systematic review and meta-analysis of mindfulness-based (Baduanjin) exercise for alleviating musculoskeletal pain and improving sleep quality in people with chronic diseases. *International Journal of Environmental Research and Public Health, 15*(2), E206.

Zou, L., Zhang, Y., Yang, L., Loprinzi, P. D., Yeung, A. S., Kong, J., et al. (2019). Are mindful exercises safe and beneficial for treating chronic lower back pain?: A systematic review and meta-analysis of randomized controlled trials. *Journal of Clinical Medicine, 8*(5), E628.

CHAPTER 16

Yoga for Stress Management

History, Research, and Practical Details

Shirley Telles
Niranjan Kala
Ram Kumar Gupta
Acharya Balkrishna

HISTORY OF YOGA

Yoga is believed to have started with the dawn of civilization. At that time, the teachings of yoga were transmitted orally from the teacher (*guru*) to the disciple (*shishya*). The development and dissemination of yoga is discussed here from its origin in the Indus Valley civilization up to the present time.

Yoga in the Indus Valley Civilization

The Indus Valley, or Harappan, civilization is one of the oldest known civilizations extending from what today is northeast Afghanistan to Pakistan and northwest India. It is believed to be more than 9,000 years old (Sarkar et al., 2016). Archaeological excavations undertaken in the 1920s and 1930s provide strong evidence of the presence of yoga in ancient India (Dhyansky, 1987). Seals such as those that picture Lord Shiva as *Pashupati* in the lotus posture are suggestive of the presence of yoga at that time. Also, fossil remains, phallic symbols resembling the *shiva-lingam,* and seals of idols of the mother Goddess are suggestive of practice of *tantra* yoga, an esoteric form of yoga.

Yoga in the Vedas

The *Vedas* (which means “knowledge” in Sanskrit) are among the oldest written texts. They are composed in Sanskrit and are believed to be what is called *apaurusheya,* meaning authorless. Though the *Vedas* were not in the written form originally, the compilations today date from ca. 10,000 B.C.E. (Bhavanani, 2012). There are four *Vedas:* the *Rigveda,* the *Yajurveda,* the *Samaveda,* and the *Atharvaveda* (Flood, 1996). The *Rigveda* contains a number of verses describing yoga as a practice to control the mind (Bhavanani, 2012). The *Atharvaveda* talks about *prana,* the vital energy, and the *chakras,* the energy centers in the *pranic* body: "There are eight energy centers and nine gates in the body. Yogis search the divine treasure in the body itself" (*Atharvaveda* 10.2.31; Bloomfield, 1897).

The *Upanishads,* which are considered the essence of the *Vedas,* contain references to yoga, its philosophical foundations, life energy, special powers attained by a yogi, and the importance of mind control. The *Kathopanishad* narrates, "The gates of the senses are opened to the outside world and not within, therefore external senses perceive the external phenomena and not the soul (*atman)* within. The wise yogis turn their consciousness inwards with the desire for immortality" (*Kathopanishad* 2.1.1; Prem & Monod-Herzen, 1982). *Kathopanishad* further defines yoga as a "higher state of mental stability attained by controlling the senses" (*Kathopanishad* 2.3.11; Prem & Monod-Herzen, 1982). The *Maitri Upanishad* mentions the sixfold yoga, consisting of breath control (*pranayama*), withdrawal of the senses (*pratyahara*), meditation (*dhyana*), concentration, contemplative inquiry (*tarka*), and self-absorption (*samadhi*; *Maitri Upanishad* 6.18; Hume, 1971). The *Mandukya Upanishad* describes four states of consciousness, viz., wakefulness (*jagrat*), dreaming (*svapna*), deep sleep (*suśupti*), and ultimate consciousness (*turiya*; Chinmayayananda, 1984). The *Mandukya Upanishad* also emphasizes control of the mind for eternal peace (Chinmayayananda, 1984).

Yoga in the Epics

Following the era of the *Vedas,* descriptions of yoga can be found in different epics, such as the *Bhagwad Gita, Mahabharata, Ramayana,* and *Yoga Vashishtha.* The period of these writings would be approximately between 5000 B.C.E. and 3000 B.C.E. (Bhavanani, 2012). The *Bhagwad Gita* describes yoga as equilibrium of the mind in every situation, whether failure or success, misery or happiness, gain or loss (*Bhagwad Gita* 2.48; Prabhupada, 1972). Further, it describes a yogi as a person who rises above the duality of action and nonaction and does not get attached to the outcome of action (*Bhagwad Gita* 4.12; Prabhupada, 1972). The qualities of a yogi, the nature of the soul (*atman),* the mind and methods of controlling it, voluntary breathing, the fire sacrifice (*havan*), and many other details about yoga are described in the 700 verses in the 18 chapters of the *Bhagwad Gita.* These teachings could be helpful for coping with stress these days.

Yoga in the Time of Patanjali

The sage Patanjali (ca. 900 B.C.E.) organized yoga teachings in the form of aphorisms or *yoga sutras* (Bhavanani, 2012). There are four chapters and 196 aphorisms compiled in a simple and nonrhetorical style. Patanjali propagated the eightfold path of yoga, which consists of the ethical precepts (*yamas* and *niyamas*), physical postures (*asanas*), breathing techniques (*pranayamas*), control of the senses (*pratyahara*), deep concentration (*dharana*), meditation (*dhyana*), and the highest state of consciousness (*samadhi*). The teachings mainly emphasize the mind and its control (Yoga Sutra 1.2; Miller, 1996).

There are five *yamas,* or moral imperatives (lack of greed, nonviolence, truthfulness, nonstealing, and abstinence) and five *niyamas* (cleanliness, external and internal; contentment; discipline; study of spiritual books; and introspection about the Self and contemplation of the God/Supreme Being), which are considered the basics of yoga practice. These are interrelated in such a way that dedication to one practice would lead to other qualities in a practitioner. Lack of greed, for example, results in nonviolence, lack of need to steal, and other qualities. These principles also help a person cope with stress.

Yoga in the Last 200 Years

In the last 200 years, yoga spread to the West, and several new ways of yoga practice were derived. This began when Swami Vivekananda (1863–1902) gave his historic speech at the World Parliament of Religions in Chicago on September 11, 1893 (Vivekananda, 1965).

A renowned yogi, Paramhansa Yogananda, founded the Self-Realization Fellowship in the United States in 1920 and drew the attention of the world to yoga and a specific meditation technique called *kriya yoga* (Goldberg, 2018). He wrote his autobiography, *Autobiography of a Yogi,* which is considered one of the best spiritual books of the 20th century and is known as "the book that changed the lives of millions" (Goldberg, 2018). Several other great masters followed in spreading yoga worldwide.

Yoga in the Current Time

In the 21st century, yoga has gained popularity worldwide for fitness and as an add-on treatment strategy. The increased public awareness about yoga may be attributed to scientific research on yoga, along with yoga movements led by prominent yoga gurus and spiritual teachers. A survey in the United States in 2008 was conducted to determine the characteristics of yoga users in 31,044 respondents (Birdee et al., 2008). The survey reported that yoga practitioners were more likely to be white, female, young, and college educated, and the majority of them (61%) felt that yoga was important to maintain general health (Birdee et al., 2008). Another study in the United States surveyed 34,524 people, aiming at determining the prevalence, patterns, and predictors of meditation (Cramer et al., 2016). The results reported that most people adopted meditation for mental health problems. An online survey in Australia reported that the most common reason for yoga practice was physical well-being (Penman, Cohen, Stevens, & Jackson, 2012). A survey in India on 5,157 respondents investigated the characteristics of yoga practitioners, motivators, and yoga techniques of choice (Telles, Sharma, Singh, & Balkrishna, 2017). These findings indicated that yoga practitioners were more likely to be males, between 21 and 44 years of age, high school educated, and students (Telles et al., 2017). Again, yoga was reported to be chiefly practiced for physical fitness (Telles et al., 2017).

DESCRIPTION OF THE METHOD

Yoga is practiced as a mind–body intervention. The word *yoga* comes from the root *yuj* (i.e., "to unite"), meaning the union of the individual Self with the Supreme Self (Taimni, 1965). Swami Vivekananda (1863–1902) described four paths of yoga (Vivekananda, 1965):

1. The path of unselfish action (*karmayoga*)
2. The path of knowledge (*jnana yoga*)
3. The path of devotion (*bhakti yoga*)
4. The path of yoga practices and meditation (*raja yoga*)

The teachings of *raja* yoga (also called *ashtanga* yoga) were first systematically compiled by the sage Patanjali (ca. 900 B.C.E.), described as a method of controlling fluctuations of the mind and consisting of eight limbs (Miller, 1996). These include moral vows or code of conduct (*yamas*); self-purification, discipline, and introspection (*niyamas*); yoga postures (*asanas*); yoga breathing exercises (*pranayamas*); sensory withdrawal (*pratyaharas*); concentration (*dharana*); meditation (*dhyana*), and transcendence of the Self to the Supreme (*samadhi*).

As the needs of yoga aspirants differ widely, several new methods have been derived in the last 200 years (see previous discussion).

What Yoga Emphasizes

As a means of physical, mental, and spiritual well-being, yoga emphasizes various aspects of life. The most popular and well-known part of yoga is the postures (*asanas*), which vary in number in different texts (Muktibodhananda, 2002; Saraswati, 2012) but are described to be practiced with steadiness, comfort, and ease (see Patanjali's Yoga Sutras, Chapter 2, Verse 46, in Miller, 1996). The importance is given to the approach of surrendering oneself to the Supreme with minimal physical exertion (see Patanjali's Yoga Sutras, Chapter 2, Verse 47, in Miller, 1996). Another component that yoga places definite emphasis upon is the synchronization of breath and mind. Importance is given to the harmonious functioning of the mind, as it is considered to influence every aspect of existence. Patanjali's Yoga Sutras describe manifestations of a fluctuating mental state as mental distress, physical tremors, difficult and erratic breathing, as well as nervousness (Miller, 1996). The *Hatha Yoga Pradipika* says: "when the breath is irregular the mind is irregular; when the breath is steady the mind is steady as well. The yogi attains stability by the regulation of breath" (Chapter 2, Verse 2, in Muktibodhananda, 2002).

How Yoga Works in Stress Reduction

The studies done by Wallace and colleagues (Wallace, 1970; Wallace, Benson, & Wilson, 1971) showed that transcendental meditation, which is derived from the yoga tradition, reduces blood lactate levels, oxygen consumed, and heart and breath rates. There have also been early reports of reduced skeletal muscle tone, especially of the frontalis muscle (Padamashree, 2007). A study by Lang, Dehof, Meurer, and Kaufmann (1979) showed changes in norepinephrine levels at baseline in long-term practitioners of transcendental meditation. There are peripheral indicators suggesting that yoga is useful in improving immunity, possibly through psychoneuroimmunology (Pennebaker, Kiecolt-Glaser, & Glaser, 1988). The studies by Kiecolt-Glaser and her team have demonstrated the benefits of yoga to improve the function of the immune system. An increasing number of measures have demonstrated that yoga practice causes a shift in the autonomic balance toward vagal dominance (Bernardi et al., 2001). The recent polyvagal theory (Porges, 2007) has demonstrated that there are two divisions of the vagus (dorsal and ventral); it appears that the shift occurring in vagal dominance is toward increased activity of the ventral, myelinated vagus. There are also references that suggest benefits in the dynamic cortical function. Two studies by Chris Streeter of Boston University demonstrated that the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) increased in the neocortex using nuclear magnetic resonance spectroscopy (NMRS; Streeter et al., 2007; Streeter et al., 2010). The increased cortical inhibition would reduce subjective feelings of anxiety.

Because yoga is essentially an experiential science, it is equally important to mention qualitative changes suggestive of stress reduction and a better overall quality of life. There are reports that, with yoga, the levels of anxiety and depression are significantly reduced in patients with chronic disease (Telles, Pathak, Kumar, Mishra, & Balkrishna, 2015). Stress reduction has been demonstrated in a wide spectrum of persons, ranging from normal healthy volunteers (evidenced by reduced serum cortisol; Bershadsky, Trumpfheller, Kimble, Pipaloff, & Yim, 2014) to persons with considerable workplace stress (Telles, Gupta, Bhardwaj, et al., 2018; Telles, Gupta, Verma, Kala, & Balkrishna, 2018) and even to those who faced extreme stress such as natural calamities and experienced posttraumatic stress disorder (PTSD; Telles, Singh, Joshi, & Balkrishna, 2010; van der Kolk et al., 2014). It has been proven that an understanding of the yoga texts is also important (Telles, Gaur, & Balkrishna, 2009). The yoga text that is especially relevant to stress reduction in yoga practitioners is the Yoga Sutras of Patanjali; it includes a description (Chapter 2, Verse 3) of the stress-inducing factors (*kleshas* in Sanskrit; Miller, 1996). The stress-producing factors and how they can contribute to physical/somatic abnormalities have been described in detail in an article titled "A Theory of Disease from Ancient Yoga Texts" (Telles, 2010). To use yoga to reduce stress, one must understand the various psychophysiological and neurochemical factors involved, as well as descriptions of stress-related factors in yoga texts.

How Yoga Practices Are Taught

Yoga is ideally taught by a person who has completed a minimum of a 1-year course that includes a knowledge of yoga practice and yoga theory. Various organizations worldwide offer year-long training programs (see *www.iayt.org*).

Under ideal conditions, yoga is taught one-on-one or with a minimal teacher-to-yoga student ratio. This would ensure that the yoga student receives adequate instruction with sufficient detail so that she or he would be able to impart adequate practical knowledge of yoga postures (*asanas*), yoga breathing (*pranayamas*), and yoga meditation (*dharana* and *dhyana*). The yoga instructions would emphasize basic rules to ensure that yoga practice was safe. For example, instructions would include that yoga should be practiced in a well-ventilated, quiet, and protected environment (Muktibodhananda, 2002). The other important instructions would include practicing yoga (ideally) in the early morning, with adequate instructions about safety, and reporting adverse events.

Reporting adverse events is very important for any yoga class and should be a part of yoga instruction. Every yoga instructor should know how to report an adverse event in a clinically acceptable way, how to seek emergency care, and how to administer first aid after receiving adequate training from an authorized institution (see *www.indianredcross.org*).

To avoid confusion among the several yoga schools that exist nowadays, it is considered best for an instructor to use descriptions from traditional yoga texts such as the Hatha texts (e.g., *Gheranda Samhita*; Saraswati, 2012; and *Hatha Yoga Pradipika;* Muktibodhananda, 2002).

How Yoga Is Used to Manage Stress

Yoga is used to manage stress in normal healthy individuals over a wide age range, from children to people age 60 years and above. Nowadays, children are under increased stress compared with earlier times. The causes of stress are related to peer pressure, academic performance, body image, concerns about how their peers view them, and whether they are accepted and welcomed within their peer group. Preteens and teens face the expected problems of adolescence, as well as problems associated with an increasing desire for freedom that could be justifiably opposed by their parents and guardians. Studies have shown that yoga is beneficial for children by increasing their self-esteem (Telles, Singh, Bhardwaj, Kumar, & Balkrishna, 2013). Apart from this there are published reports of the use of yoga in schools (Butzer et al., 2015).

Hence stress is experienced throughout life and in different situations. Adults often face stress at the workplace (Telles, Gupta, Bhardwaj, et al., 2018; Telles, Gupta, Verma, et al., 2018), and stress occurs in older persons as well (Krishnamurthy & Telles, 2007). In all the cases mentioned above, yoga was found to be beneficial.

Overall Effects of Yoga

Apart from its usefulness in the management of stress, yoga practice has other proven benefits for health. A single yoga breathing practice was shown to reduce blood pressure, and improvement was evidenced in a vigilance task performance in normal healthy adults (Telles, Verma, Sharma, Gupta, & Balkrishna, 2017). This was suggestive of increased vagal activity and a relaxed state while being vigilant. Practicing yoga for 3 months was helpful in managing weight-related outcomes in 26 females with central obesity (Telles, Sharma, et al., 2018). The authors reported a significant decrease in anthropometric measures and cholesterol levels and an improvement in the quality of life following 12 weeks of yoga, and the results were almost comparable when analyzed for different age ranges (Telles, Sharma, et al., 2018).

The examples given in Table 16.1 and in the references cited above suggest the benefits of yoga in stress management and the mechanisms that may underlie these benefits. Some of the studies mentioned above have been detailed in Table 16.1.

CONTRAINDICATION AND CAVEATS OF YOGA PRACTICE

Nowadays there is increased awareness that adverse events can occur if yoga techniques are practiced wrongly. Some important instructions apply to all yoga techniques. These include:

1. Receiving instructions from an adequately trained yoga teacher. Instruction should not be received from those with dubious qualifications.
2. The instructions to practice yoga should be clear, based on a particular line of teaching that is supported by written material and preferably has its roots in traditional texts.
3. All esoteric and cult-driven practices should be avoided.
4. Yoga is best practiced when an individual is energetic and relaxed. It is generally considered that the most suitable time for this is the early morning. However, yoga can be practiced at other times as well. However, at least 3 hours should elapse after the preceding meal.
5. For the practices mentioned in Table 16.2, which are specific for stress reduction, there are a few contraindications. All forward-bending practices are to be strictly avoided in persons with back pain, irrespective of the cause. Certain practices, such as unilateral knee and ankle rotation, the triangle pose, or the half-wheel pose, which require balancing, should be practiced with adequate support in persons who are elderly or whose balance is disturbed due to stress. Contraindications also exist for many other yoga practices not mentioned in Table 16.2. Thus any yoga teacher or yoga therapist should consult a medical practitioner if prescribing therapeutic yoga.
6. A final point is that yoga practice should never cause pain. Any practice that involves pain should be avoided. Also, yoga practices should not lead to fatigue.

TABLE 16.1. Yoga for Stress Management in Different Age Groups: Methods and Mechanisms

Categories	Studies	Methods	Reported outcomes
Stress management through yoga in children and youth	Noggle, Steiner, Minami, & Khalsa (2012)	51 students were randomized for yoga or physical education. Psychosocial well-being was measured through Profile of Mood States—Short Form (POMS-SF) and Positive and Negative Affect Schedule for Children scales.	Total Mood Disturbance and Tension-Anxiety subscales improved in yoga students and worsened in controls. Negative affect significantly worsened in controls but improved in yoga students.
	Malathi & Damodaran (1999)	50 students of medicine were given yoga before examination. Levels of anxiety were measured through Spielberger's Anxiety Scale.	The anxiety scores, which rose prior to the exams, were reduced on the day of exam after yoga.
Stress management through yoga in adults	Telles, Gupta, Bhardwaj, et al. (2018)	Mental well-being and state anxiety were assessed in 118 schoolteachers following a 15-day yoga program and compared with an equal number of controls.	An increase in overall mental well-being and lower state anxiety was found in the yoga group.
	Telles, Gupta, Verma, Kala, & Balkrishna (2018)	722 military personnel were given yoga for 9 days; their state anxiety, self-rated sleep and performance in a vigilance test were measured before and after the intervention.	There was a significant increase in scores in the vigilance test, a decrease in state anxiety, and improved self-rated sleep after 9 days of yoga.
	Hartfiel & Edwards (2017)	Eight RCTs were reviewed to determine the effectiveness of yoga in the workplace.	Yoga's effectiveness was strong for musculoskeletal conditions, moderate for perceived stress, limited for sleep quality, and conflicting for heart rate variability.

Stress management through yoga in seniors	Bhatnagar, Tripathi, & Kumar (2016)	74 elderly subjects were given a lifestyle program for 3 months, which included a morning walk, *nadi shodan pranayama*, dietary restrictions, and increased intake of water. Blood pressure and oxidative stress markers, glutathione (GSH-r), super oxide dismutase (SOD), and malondialdehyde (MDA), were measured before and after the intervention.	An increase in GSH-r and SOD and reduction in MDA levels was reported after the intervention.
Mechanisms of how yoga works	Chaya, Kurpad, Nagendra, & Nagarathna (2006)	The basal metabolic rate (BMR) was compared between yoga practitioners and non–yoga practitioners.	The BMR was lower by about 13% in the yoga practitioners compared with the non–yoga practitioners.
	Streeter et al. (2010)	34 adults were randomized to either yoga or a metabolically matched exercise group. Mood, anxiety, and brain GABA levels were measured before and after the interventions.	The yoga group showed comparably increased GABA levels, along with improved mood and anxiety.
	Telles, Gupta, Yadav, Pathak, & Balkrishna (2017)	13 male participants practiced alternative nostril yoga breathing, breath awareness, and quiet sitting on different days. The EEG was measured before, during, and after the three interventions.	The relative power in the theta band decreased during alternative-nostril yoga breathing, and the beta amplitude was lower after this practice. The relative power in the beta band increased, and amplitude of the alpha band was reduced during quiet sitting.

A MANUAL FOR TRAINING IN YOGA

Yoga should be practiced in a safe, comfortable, and well-ventilated place. Ideally, there should be no external noise that could disturb the practice. Yoga practice should always be undertaken at least 3 hours after eating a meal. Also, the practices should be performed under the supervision of a trained yoga teacher. Some of the practices that are most useful to reduce stress and the effects of stress on the body are listed in Table 16.2.

CASE EXAMPLE: THE BENEFITS OF YOGA AS AN ADD-ON THERAPY

Stress can cause various changes in the normal functioning of the body, leading to a wide range of symptoms and, in certain cases, to disease.

The following case history is of a patient who presented with symptoms of difficulty in breathing at the emergency room. The patient was later admitted to the internal medicine department and was referred for stress management strategies to an integrative medicine center that manages stress-related disorders with yoga, various natural remedies, and ayurveda. The patient, ML, was a 35-year-old South Asian female; ML was from an "upper-middle-class" family (based on known criteria for categorizing families based on income per month and the number of non-wage-earning members in the family; Telles, Sharma, et al., 2017). ML lived in a cosmopolitan city, had been married for 8 years, and had a 4-year-old daughter. ML had a full-term pregnancy and normal delivery. ML was employed as a professor in a private university and worked for approximately 8 hours a day, 5 days a week. ML came from an urban background, and had two siblings and a widowed mother living in the same city.

Presenting Symptoms

After the acute episode of breathlessness was managed in the emergency services, ML was examined as an inpatient in a private room under the respiratory medicine division of the hospital. ML appeared at ease and interested in getting well as soon as she could. On examination, ML had a normal body mass index (BMI) and no evidence of nasal septal deviation, enlarged tonsils, or other signs of an upper respiratory tract infection. With a pulse of 80 beats per minute (bpm), a normal body temperature, and a respiratory rate (on observation) of approximately 22 breaths per minute, ML appeared to be in normal health, although slightly anxious. A further examination showed that ML had no evidence of pallor, cyanosis, chubbing of the fingers, stridor, or using the accessory muscles of respiration to breathe. However, ML's hands were clasped tightly, with clammy and cold palms. Her respiration was shallow, not rhythmic, and irregular. Her heart sounds were normal. There was no evidence of hepato/splenomegaly or any other abdomino-pelvic disorder.

History of the Present Illness

ML had developed acute respiratory distress at 3 A.M. 5 days prior to the present episode. She used an inhaler, which was a β2 adrenergic receptor agonist along with corticosteroids. She had been advised to use the inhaler as soon as she felt any difficulty in breathing. When ML did not feel any relief with the inhaler, she asked a neighbor (as her husband was away) to take her to the emergency room of a hospital nearby, which she

TABLE 16.2. Details of the Yoga Practices Suggested for Stress Management

Serial No.		Yoga practices	Description	Duration
1		Universal prayer[a]		3 minutes
2	Loosening exercises	Neck movements	Flexion, extension, side bending, and rotation	3 minutes
		Knee and ankle rotation	Practiced while standing	3 minutes
		Shoulder rotation	Both anterior–posterior and lateral	3 minutes
		Butterfly pose	Practiced seated with leg and hips flexed apart and knees flexed, feet in contact	1 minute
3	Standing postures	Half-wheel pose (*ardhachakrasana*)	A backward-bending posture	3 minutes
		Hand-to-foot pose (*padahastasana*)	A forward-bending posture	3 minutes
		Triangle pose (*trikonasana*)	A side-bending posture	2 minutes
4	Prone postures	Crocodile posture (*makarasana*)	A prone relaxation posture	5 minutes
		Cobra posture (*bhujangasana*)	A prone posture with raised upper body and extended neck	3 minutes
		Locust posture (*salabhasana*)	A posture with raised legs, extended and straight at the knee	3 minutes
5	Supine postures	Half-plough posture (*ardhhalasana*)	Legs raised to 90 degrees, straight at the knee	3 minutes
		Monkey pose (*markatasana*)	A spinal twist	3 minutes
6	Yoga breathing series *(pranayamas)*	Alternate-nostril yoga breathing (*anulom-vilom*)	Breathing through left and right nostril alternately	6 minutes
		Bumblebee breathing practice (*bhramari*)	Exhalation with a humming sound like a bumble bee	6 minutes
		Om chanting (*udgeeth*)	Exhalation with repetitive chanting of "om"	3 minutes
7	Relaxation	Supine relaxed posture (*Shavasana)* with breath awareness		5 minutes
8		Universal prayer[a]		2 minutes
		Total duration		60 minutes

[a]*Om Asato Maa Sad-Gamaya; Tamaso Maa Jyotir-Gamaya; Mrityor-Maa Amritam Gamaya; Om Shaantih Shaantih Shaantih* (Keep me not in Unreality but lead me towards Reality; Keep me not in Darkness but lead me towards Light; Keep me not in Death but lead me towards Immortality; Peace, Peace, Peace).

had visited before. In the emergency room, ML was noted as pale, mildly cyanosed, with noisy, difficult breathing. She was administrated oxygen to inhale and an intravenous bronchodilator. As her breathing eased, her heart rate decreased from 104 bpm on admission to 88 bpm. Her respiratory rate also decreased from 30 breaths per minute, and her arterial oxygen levels, which were low on admission, returned to normal levels, with 5 minutes of inhaled oxygen and intravenous medication.

While the patient appeared to breathe with greater ease, the emergency room notes recorded wheezing, use of the accessory muscles of respiration, and extreme anxiety. On auscultation, ronchi were present, with no other cardiorespiratory abnormality. No investigations were carried out until the patient was transferred to a private room in the respiratory medicine division the next morning. The notes the next morning recorded that ML had an uneventful sleep with normal arterial oxygen saturation, breathing, body temperature, and pulse rate. Despite oral bronchodilators and the use of a nebulizer with a β2 adrenergic agonist, ML continued to have ronchi but no difficulty in breathing 5 days after the acute episode.

Past History

ML said that she was diagnosed with bronchial asthma at the age of 8. She also had flexural eczema affecting the ventral surfaces of her elbows and knees. Both the respiratory and the dermatological symptoms were worsened by stress, cold air, strong smells (irrespective of whether they were pleasant or unpleasant), and certain foods, especially kidney beans and certain lentils. At menarche, her eczema was no longer a problem. However, ML continued to have episodes of breathing difficulty and continued to use an inhaler as required. The precipitating factors continued to be either cold or very warm humid weather, stress, and frequent respiratory tract infections, which usually required a dose of antibiotics. In the 5 years prior to the present episode, ML had four emergency room treatments. Three of them appeared to be related to upper respiratory tract infections and one of them to work-related stress, as ML had to complete a particular project within a short time interval at work.

Family History

Characteristically, ML's mother had a history of allergic rhinitis ("hay fever"). Neither of her siblings had a history of bronchial asthma, eczema, or allergic rhinitis.

Investigations

ML had a normal hemoglobin level, normal total white-blood-cell and platelet counts, but high levels of eosinophils, which was seen on earlier tests as well. Pulmonary function tests showed lower levels of the forced expiratory flow (FEF) at 25, 50, and 75% of the vital capacity. The tidal volume and vital capacity were lower than the expected value for ML, taking age, gender, and race into account. There were no abnormalities in the EKG.

Details about Stress as a Precipitating Factor

During childhood, ML was the eldest of three children, having a brother 3 years younger and a sister 6 years younger than she. ML remembered her childhood as happy and carefree. However, she also mentioned that she used to be excessively anxious to perform well at school, as her parents expected her to be a good role model for her younger siblings. Her episodes of eczema and bronchial asthma were worse just before examinations and also when the extended family gathered together. At these times, ML felt that she had to take care of her younger siblings. She also felt responsible for their behavior. Apart from this, ML was very academically successful and participated in various extracurricular activities. However, ML did not enjoy sports or organized physical training in school, as she was apprehensive that it would bring on an attack of bronchial asthma. ML was a very conscientious child. Despite her first description of her childhood as happy and carefree, further discussions with her showed that she was often anxious, especially when there appeared to be a disagreement between the adults, when anyone was ill, or when she had to stay away from home, either for the holidays or for a school trip. Most often, anxiety worsened her eczema and episodes of bronchial asthma.

Stress Reported during Adolescence

ML attained menarche at the age of 13. Her transition into adolescence was not marked by any episodes of anger or rebellion. During this time, she stopped experiencing eczema. However, her episodes of bronchial asthma worsened in frequency and severity, with a minimum of two emergency room admissions in a year with status asthmaticus. ML had few close friends. She continued to perform well academically. As an adolescent, her stress was precipitated by having to participate in social events. ML states that at this stage in her life she felt acutely self-conscious. She also remembers that her mother used to be critical of her posture, and this increased her anxiety and self-consciousness. She also experienced academic stress as she approached her school-ending examinations. At this stage, she also developed tension headaches, which continued while at the university.

Stress Related to the Transition from Student Life to Holding a Job

ML was married at the age of 28 years to a man who was 4 years older than she. Both of them were from an academic background and encouraged each other to do well. They had a single child. ML's anxiety and stress continued to precipitate episodes of bronchial asthma and even status asthmaticus.

Management through Yoga

ML was admitted at an integrative medicine center. She knew that she would continue with the conventional treatment (inhaled bronchodilators with corticosteroids) and could access emergency treatment. To begin with, a detailed history of stressful life events and ML's coping strategies was recorded.

ML's routine was planned. Apart from yoga practice, this included "yoga-based counseling" and a diet plan that was based on clinical experience, as well as principles of natural medicine (Cordingley, 1996) and ayurveda (Balkrishna, 2013). The diet was a normal, high-fiber plant-based diet. Milk products were avoided, with the exception of whey/buttermilk, which ML was allowed twice a week. ML had a usual breakfast, a usual lunch, and at night she was encouraged to eat early and eat light (boiled vegetables and a warm, clear soup) at least 2 hours before bedtime.

The yoga practice began with an observation of ML'S breathing. Her breathing was rapid, shallow, thoracic, and jerky. She was encouraged to try diaphragmatic breathing, placing her hand on her anterior abdominal wall, feeling it push outward with an in-breath and inward with each out-breath.

ML found this difficult to do. She felt anxious initially, but when she realized that she was not being evaluated or judged, she relaxed, and gradually her breathing became slower and deeper. During this time, ML was given "loosening exercises," which help in chest expansion (e.g., spreading the arms outward while inhaling and bringing them together while exhaling). Because ML often contracted her neck muscles when breathing with difficulty, she was given loosening exercises to relax her neck muscles (these included flexion, extension, side bending, and rotation). The postures were initially selected to promote chest expansion, such as backward bending in the half-wheel posture. Forward bending produced compression of the chest, and ML was told to remain in the posture for only as long as she felt comfortable. Although she was initially very anxious with any posture that involved pressure on the chest, at the end of the 15-day course she reported that she felt confident that she was able to remain in the posture and not feel anxious. Other postures that were included specially to reduce ML's anxiety associated with breathing were the cobra pose (for chest expansion), the locust pose (to strengthen the back muscles), the fish pose (for chest expansion), and the mountain pose (for chest compression). ML was also taught sun salutations and yoga breathing exercises. The yoga counseling was carried out by a trained psychologist counselor who was also trained to use yoga as a therapy. The sessions consisted of understanding ML, the causes of her getting anxious at different stages in her life (described earlier), and how these things influenced her episodes of bronchial asthma. After listening to ML, the therapist counselor asked ML to observe her reactions to situations (or places and people) that she was uncomfortable with and notice whether her breathing changed. This also encouraged ML to analyze and understand causes of her anxiety and discomfort. As the sessions progressed, ML reported that she was convinced that her mental state and thoughts affected her breathing and that she could modify this if she were aware of it.

At the end of the 15-day intensive lifestyle-change program, ML felt more confident that she could cope with stress and anxiety and prevent stress-related episodes of bronchial asthma. It was made clear to ML that this was not a cure. She needed to remain in contact with her respiratory medicine physician and use allopathic/conventional medicine when required. In conclusion, it could be said that yoga practice, yoga counseling, and a change in diet helped ML reach normal values of FEF 25–75%, as well as a normal tidal volume and vital capacity values. Her dependence on inhaled medication was reduced. On the whole, it was a positive outcome.

REFERENCES

Balkrishna, A. (2013). *A practical approach to the science of ayurveda: A comprehensive guide for healthy living.* Haridwar, India: Divya Prakashan.

Bernardi, L., Sleight, P., Bandinelli, G., Cencetti, S., Fattorini, L., Wdowczyc-Szulc, J., et al. (2001). Effect of rosary prayer and yoga mantras on autonomic cardiovascular rhythms: Comparative study. *British Medical Journal, 323*(7327), 1446.

Bershadsky, S., Trumpfheller, L., Kimble, H. B., Pipaloff, D., & Yim, I. S. (2014). The effect of prenatal Hatha yoga on affect, cortisol and depressive symptoms. *Complementary Therapies in Clinical Practice, 20*(2), 106–113.

Bhatnagar, A., Tripathi, Y., & Kumar, A. (2016). Change in oxidative stress of normotensive elderly subjects following lifestyle modifications. *Journal of Clinical and Diagnostic Research, 10*(9), CC09–CC13.

Bhavanani, M. D. (2012). *The history of yoga from ancient to modern times.* Pondicherry, India: Satya Press.

Birdee, G. S., Legedza, A. T., Saper, R. B., Bertisch, S. M., Eisenberg, D. M., & Phillips, R. S. (2008). Characteristics of yoga users: Results of a national survey. *Journal of General Internal Medicine, 23*(10), 1653–1658.

Bloomfield, M. (Ed.). (1897). *Hymns of the Atharva-veda: Together with extracts from the ritual books and the commentaries* (Vol. 1). London, UK: Clarendon Press.

Butzer, B., Day, D., Potts, A., Ryan, C., Coulombe, S., Davies, B., et al. (2015). Effects of a classroom-based yoga intervention on cortisol and behavior in second- and third-grade students: A pilot study. *Journal of Evidence-Based Complementary and Alternative Medicine, 20*(1), 41–49.

Chaya, M. S., Kurpad, A. V., Nagendra, H. R., & Nagarathna, R. (2006). The effect of long-term combined yoga practice on the basal metabolic rate of healthy adults. *BMC Complementary and Alternative Medicine, 6*(1), 28.

Chinmayayananda, S. (1984). *Mandukya Upanishad*. Mumbai, India: Sachin.

Cordingley, E. W. (1996). *Principles and practice of naturopathy*. Pomeroy, WA: Health Research Books.

Cramer, H., Hall, H., Leach, M., Frawley, J., Zhang, Y., Leung, B., et al. (2016). Prevalence, patterns, and predictors of meditation use among US adults: A nationally representative survey. *Scientific Reports, 6,* 36760.

Dhyansky, Y. Y. (1987). The Indus Valley origin of a yoga practice. *Artibus Asiae, 48*(1/2), 89–108.

Flood, G. D. (1996). *An introduction to Hinduism*. Cambridge, UK: Cambridge University Press.

Goldberg, P. (2018). *The life of Yogananda: The story of the yogi who became the first modern guru*. Carlsbad, CA: Hay House.

Hartfiel, N., & Edwards, R. D. (2017). Research-based applied psychophysiology: Yoga for occupational stress and health. In S. Telles & N. Singh (Eds.), *Research based perspectives on the psychophysiology of yoga* (pp. 175–183). Hershey, PA: IGI Global.

Hume, R. E. (1971). *The thirteen principal Upanishads: Translated from the Sanskrit with an outline of the philosophy of the Upanishads*. Oxford, UK: Oxford University Press.

Krishnamurthy, M., & Telles, S. (2007). Effects of yoga and an Ayurveda preparation on gait, balance and mobility in older persons. *Medical Science Monitor, 13*(12), LE19–LE20.

Lang, R., Dehof, K., Meurer, K. A., & Kaufmann, W. (1979). Sympathetic activity and transcendental meditation. *Journal of Neural Transmission, 44*(1–2), 117–135.

Malathi, A., & Damodaran, A. (1999). Stress due to exams in medical students: A role of yoga. *Indian Journal of Physiology and Pharmacology, 43,* 218–224.

Miller, B. S. (1996). *Yoga: Discipline of freedom: The yoga sutra attributed to Patanjali*. Cambridge, UK: University of California Press.

Muktibodhananda, S. (2002). *Hatha Yoga Pradipika*. Munger, India: Bihar School of Yoga.

Noggle, J. J., Steiner, N. J., Minami, T., & Khalsa, S. B. S. (2012). Benefits of yoga for psychosocial well-being in a US high school curriculum: A preliminary randomized controlled trial. *Journal of Developmental and Behavioral Pediatrics, 33*(3), 193–201.

Padamashree, G. (2007). *Immediate effect of yogic relaxation on frontalis surface EMG*. Unpublished master's thesis, Swami Vivekananda Yoga University, Bengaluru, India.

Penman, S., Cohen, M., Stevens, P., & Jackson, S. (2012). Yoga in Australia: Results of a national survey. *International Journal of Yoga, 5*(2), 92.

Pennebaker, J. W., Kiecolt-Glaser, J. K., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology, 56*(2), 239.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology, 74*(2), 116–143.

Prabhupada, A. C. B. S. (1972). *Bhagavad-Gita as it is*. Mumbai, India: Bhaktivedanta Book Trust.

Prem, K., & Monod-Herzen, G. E. (1982). *The yoga of the Kathopanishad*. Monaco, France: Editions du Rocher.

Saraswati, N. (2012). *Gheranda Samhita: Commentary on the yoga teachings of Maharshi Gheranda*. Munger, India: Yoga Publications Trust.

Sarkar, A., Mukherjee, A. D., Bera, M. K., Das, B., Juyal, N., Morthekai, P., et al. (2016). Oxygen isotope in archaeological bioapatites from India: Implications to climate change and decline of Bronze Age Harappan civilization. *Scientific Reports, 6,* 26555.

Streeter, C. C., Jensen, J. E., Perlmutter, R. M., Cabral, H. J., Tian, H., Terhune, D. B., et al. (2007). Yoga Asana sessions increase brain GABA levels: A pilot study. *Journal of Alternative and Complementary Medicine, 13*(4), 419–426.

Streeter, C. C., Whitfield, T. H., Owen, L., Rein, T., Karri, S. K., Yakhkind, A., et al. (2010).

Effects of yoga versus walking on mood, anxiety, and brain GABA levels: A randomized controlled MRS study. *Journal of Alternative and Complementary Medicine, 16*(11), 1145–1152.

Taimni, I. K. (1965). *The science of yoga: A commentary on the Yoga-sūtras of Patañjali in the light of modern thought*. Madras, India: Theosophical Publishing House.

Telles, S. (2010). A theory of disease from ancient yoga texts. *Medical Science Monitor, 16*(6), LE9.

Telles, S., Gaur, V., & Balkrishna, A. (2009). Effect of a yoga practice session and a yoga theory session on state anxiety. *Perceptual and Motor Skills, 109*(3), 924–930.

Telles, S., Gupta, R. K., Bhardwaj, A. K., Singh, N., Mishra, P., Pal, D. K., et al. (2018). Increased mental well-being and reduced state anxiety in teachers after participation in a residential yoga program. *Medical Science Monitor Basic Research, 24,* 105.

Telles, S., Gupta, R. K., Verma, S., Kala, N., & Balkrishna, A. (2018). Changes in vigilance, self-rated sleep and state anxiety in military personnel in India following yoga. *BMC Research Notes, 11*(1), 518.

Telles, S., Gupta, R. K., Yadav, A., Pathak, S., & Balkrishna, A. (2017). Hemisphere specific EEG related to alternate nostril yoga breathing. *BMC Research Notes, 10*(1), 306.

Telles, S., Pathak, S., Kumar, A., Mishra, P., & Balkrishna, A. (2015). Influence of intensity and duration of yoga on anxiety and depression scores associated with chronic illness. *Annals of Medical and Health Sciences Research, 5*(4), 260–265.

Telles, S., Sharma, S. K., Kala, N., Pal, S., Gupta, R. K., & Balkrishna, A. (2018). Twelve weeks of yoga or nutritional advice for centrally obese adult females. *Frontiers in Endocrinology, 9,* 466.

Telles, S., Sharma, S. K., Singh, N., & Balkrishna, A. (2017). Characteristics of yoga practitioners, motivators, and yoga techniques of choice: A cross-sectional study. *Frontiers in Public Health, 5,* 184.

Telles, S., Singh, N., Bhardwaj, A. K., Kumar, A., & Balkrishna, A. (2013). Effect of yoga or physical exercise on physical, cognitive and emotional measures in children: A randomized controlled trial. *Child and Adolescent Psychiatry and Mental Health, 7,* 37.

Telles, S., Singh, N., Joshi, M., & Balkrishna, A. (2010). Posttraumatic stress symptoms and heart rate variability in Bihar flood survivors following yoga: A randomized controlled study. *BMC Psychiatry, 10,* 18.

Telles, S., Verma, S., Sharma, S. K., Gupta, R. K., & Balkrishna, A. (2017). Alternate-nostril yoga breathing reduced blood pressure while increasing performance in a vigilance test. *Medical Science Monitor Basic Research, 23,* 392–398.

van der Kolk, B. A., Stone, L., West, J., Rhodes, A., Emerson, D., Suvak, M., et al. (2014). Yoga as an adjunctive treatment for posttraumatic stress disorder: A randomized controlled trial. *Journal of Clinical Psychiatry, 75*(6), e559–e565.

Vivekananda, S. (1965). *The complete works of Swami Vivekananda*. Kolkata, India: Advaita Ashrama.

Wallace, R. K. (1970). Physiological effects of transcendental meditation. *Science, 167*(3926), 1751–1754.

Wallace, R. K., Benson, H., & Wilson, A. F. (1971). A wakeful hypometabolic physiologic state. *American Journal of Physiology, 221*(3), 795–799.

SECTION C

PSYCHOTHERAPEUTIC METHODS

CHAPTER 17

Cognitive Therapy for Stress

Robert L. Leahy
Scott Woodruff

In this chapter, we describe a cognitive therapy model of stress, a conceptualization of stress, and descriptions of helpful and unhelpful strategies for coping with stress. Rather than limit ourselves to the traditional cognitive model advanced by Aaron T. Beck, we propose an expanded, modern cognitive model that is necessarily integrative, reflecting advances in modern cognitive and behavioral therapy, including more traditional cognitive therapy, schema therapy, metacognitive therapy, mindfulness, emotional schema therapy, and acceptance approaches. We begin with a general description of stress, stressors, and coping. We elaborate a more complex model of stress beginning with the appraisal model first advanced by Lazarus. We examine how stress can be conceptualized by different contemporary cognitive-behavioral therapy (CBT) models. We identify problematic coping strategies for stress and propose more helpful models for intervention. Finally, we describe how mindfulness-based cognitive therapy (MBCT) can be integrated into a more comprehensive approach to stress. We complete this chapter with a brief review of the research on the effectiveness of CBT in coping with stress. Throughout this chapter, we describe cases of work and relationship stress and how this integrative modern cognitive model can be useful.

DEFINING STRESS AND STRESSOR

Stress Appraisal

It is common for people to refer to "stress" as the tasks or challenges they face, the experience of frustration when coping with the challenge, or the resulting sense of exhaustion after addressing the challenge. In this chapter, we refer to several facets of the "stress response": (1) the *stressor*—the challenge or task that is being addressed (e.g., "I have to get this project completed by Monday morning"); (2) the *meaning* of the stressor to the individual ("If I don't do a good job, I could get fired"); (3) the *coping evaluation*—the individual's evaluation as to his or her ability to cope with this challenge, including the entire range of automatic thoughts, conditional rules, and assumptions and personal schemas that are activated ("I am really lazy and incompetent and I don't know how to do a lot of these things"); (4) the *resulting experience*—the physiological correlates, frustrations, emotions, and experiences when confronting the challenge ("I feel tension in my stomach, sweating, I feel frustrated and defeated and hopeless"); (5) the *emotional schemas*—the appraisal of one's emotions and experience (e.g., that the experience of stress will go on indefinitely and overwhelm the individual); and (6) the *problematic and the helpful coping strategies*—the resulting strategies to cope with these phenomena ("I should wait until I am ready to cope with this"; "I need to avoid"; "I worry about how it will turn out"). We discuss the appraisal and response to stress as a sequence of evaluations and strategies.

Identifying the Stressor

Many clients will say that they are stressed by tasks or challenges that they are facing: "I have a lot of stress in my life." It may be unclear whether they are referring to specific tasks, to their frustrating experience, or to their appraisals of their ability to cope. However, in identifying the stressor, the clinician can attend to the manner in which the stressor is described. For example, is the stressor described in amorphous and overgeneralized terms ("I have so many things to do that I feel overwhelmed"), or in specific terms ("I need to get this report in by Friday morning"), or is there no specific or general task or challenge ("I just feel stressed")? In the case of overgeneralized descriptions (e.g., "I have so many things to do" or "Everything is coming at me all at once"), the client is likely to feel overwhelmed, confused, and helpless, as it is difficult to imagine how one copes with "everything" at once. Research on overgeneralized thinking indicates that this style of thinking is associated with vulnerability to recurrent depression, because the individual believes that he or she is unable to specify a coping style for amorphous challenges (Williams, Teasdale, Segal, & Soulsby, 2000; Segal, Williams, & Teasdale, 2002). A clinician using the cognitive therapy approach assists the client in narrowing the focus to the specific task or aspects of a task that are believed to elicit stress—for example, "Can you tell me which specific part of this project seems the most stressful?" In addition, it can be helpful to identify this style of overgeneralized thinking as a source of stress, because amorphous and overgeneralized "stressors" are difficult to identify and difficult to evaluate rationally. For example:

> "I notice that when you describe what is frustrating you that you talk about it in very general terms, like 'My work is getting me down.' Now, one thing to think about is that we are going to be much better at coping with something if we can describe a specific task—for example, 'I need to get the outline done before Monday.' The reason that a specific description is better than something more general is that your description about 'my work' is hard to define. It could be anything. Do you find that you often think about things that frustrate you in very general and vague terms?"

We have also found it helpful to indicate to clients that the most valuable descriptions of a stressor would be something that we could describe as a behavior ("Get the outline done") or something that we could imagine taking a picture of ("Drive to the office and get the files"; Leahy, 2017). We discuss the value of mindfulness interventions later in this chapter.

Interpretations of Stressors

The client's interpretation of the stressful event includes a number of dimensions: the meaning, the magnitude, the duration, the approaching or looming process, and the possibility of escalation of stressors. The "meaning" of the stressor includes the interpretations that the stressor is personally important, that it reflects on the competence or value of the individual, and that one "must" cope successfully. For example, the stressor at work—"This report needs to be done by Monday"—may be viewed as personally important simply because it was assigned to the individual. But the *personal relevance* of the stressor may be magnified in the individual's mind because coping successfully with the stressor is viewed as a reflection of whether one is a failure or a success in life. Concurrent with this interpretation is the view that one "must do well on this." The clinician can address these personal meanings of the stressor and coping by asking the following questions:

1. "How can a simple task at work be so personally relevant to you when you have completed countless other tasks in the past and will in the future?"
2. "Was this task 'designed for you' or is it an arbitrarily determined task in this job?"
3. "How can one task lead to the conclusion that someone is a failure if you succeed on other tasks? Would it follow that succeeding on one task means you are a success?"

In addition, the demand that "I must do well on this" can be explored directly with the following inquiries:

1. "Why must you do well on this? Is there a universal moral rule or law that says this?"
2. "Would it be more accurate to say, 'I would prefer to do well, but it is not a requirement for living to do well on this task'?"
3. "Would everyone who is presented with this task be obligated by some moral rule to do well?"

Examining these demanding expectations may help the client put in perspective that this is just a task—it is not the meaning of life, the value of who they are, or a burdensome moral prescription.

Another set of interpretations of the stressor includes the magnitude and duration of the stressor. The magnitude of the stressor reflects how large and significant the particular task is. For example, performing open-heart surgery would be a task with large magnitude, as people can die, but getting a report in next week is not of equivalent magnitude. The duration of the stressor is also significant. For example, being under a time constraint that this needs to be done by Tuesday implies that one may undergo a couple of days (duration) of the stressful event but not a lifetime of demands from this task. Some stressors are viewed as approaching rapidly, although in reality they may approach gradually in small degrees. The individual may imagine the stressor as moving slowly so that, at each point in the movement of the stressor as it approaches, the individual may have coping strategies (Riskind, Black, & Shahar, 2010). For example, rather than thinking of the Tuesday deadline as approaching at a rapid speed, one can think of each hour of the day and of each day as involving smaller tasks that can be completed. In addition, to reduce the sense of rapid approach and collision with the stressor, the individual can imagine and plan stepping aside for stress breaks to recognize that one is not on an imminent collision path (Riskind, Williams, Gessner, Chrosniak, & Cortina, 2000; Riskind, 1997).

EXPECTATIONS OF COPING

Clients differ considerably in terms of the expectations of performance that they hold for themselves. These expectations can include the standard of outcome that is acceptable, the time frame that is tolerated, the comfort level that is anticipated, and how their performance is evaluated.

Maximizing Beliefs

Individuals who expect close to 100% levels in performance are far more likely to experience stress than those with more realistic expectations of performance. Clients holding perfectionistic beliefs may also endorse a motivational theory that "the highest standards are necessary to preserve motivation." For example, one client commented, "If I accept less than perfection, I will lose my edge, I won't be motivated, I just want to be able to do as well as I could. If I let my standards relax, I am afraid that I will become lazy and mediocre." Research comparing "maximizers" and "satisfiers" indicates that maximizers who uniformly seek the best possible outcome are more dissatisfied, more indecisive, and have more regrets (Schwartz et al., 2002). The research on perfectionism also supports this observation. The clinician can illustrate three levels of evaluation: maximizers, satisfiers, and those who are apathetic. The classic Yerkes–Dodson law (Yerkes & Dodson, 1908) indicates that with almost no arousal, there is almost no motivation; that with very high arousal, motivation also drops out due to feelings of helplessness and frustration; and that the best levels of motivation are the optimal (intermediate) levels of arousal. Maximizers have a punitive and fear-based theory of motivation: "I have to scare myself to get things done." The clinician can examine the negative consequences of this fear—such as procrastination, avoidance, rumination, worry, and depression—that all interfere with performance. For example, one client thought that he had to treat each assignment as critical or he would become lazy, but then he observed that his catastrophic thoughts about assignments contributed to his procrastination, avoidance, and depression.

Fixed versus Growth Beliefs

One source of stress at work or study is the belief that one's abilities are stable and cannot improve or change. Dweck (2006) has described differences in mind-sets that individuals utilize in approaching challenging tasks. Drawing on decades of research on cognitive factors that underlie helplessness versus persistence or resilience, Dweck describes a mind-set that some individuals have that abilities are fixed as opposed to the belief or mind-set that abilities can grow. In early studies, Dweck (1975) divided children into two groups—100% success versus partial success with "effort" attribution. In the first group, children were given easily solvable puzzles on which they experienced 100% success, whereas in the second group children were given solvable puzzles along with impossible puzzles on which the children would fail. After their failure on the task, the experimenter told them that they should try harder (effort attribution). In the second phase of the study, both groups were given solvable and unsolvable puzzles, along with challenging but solvable puzzles.

The 100% success group were more likely to give up following failure, whereas the effort attribution group was more likely to persist (Dweck, 1975). Dweck found that children who persist following a failure are more likely to spontaneously use "growth" terms in self-talk, such as "I can try harder" or "That's interesting, let me look at it again." These beliefs in flexible growth concepts of ability are often implicated in the stress response.

In addressing stress, the individual can consider the possibility that increasing and prolonged effort might increase ability over time. This longer term "investment" approach, with effort as the investment and ability or skill as the payoff, can reduce the sense of helplessness and decrease the self-critical labeling that often exacerbates stress. For example, a young man on a new job that required learning technical skills initially felt undermined, demoralized, and frustrated because he believed he would never learn what was necessary and that this predicted that his future would be bleak. Moreover, because he attributed his difficulty to a fixed trait of "incompetence," he felt discouraged in even trying: "Why bother? I will never learn this." The clinician and the client reviewed all the other skills that he had acquired in the past and how this often took time, trial and error, and an investment in some discomfort along the way. "Perhaps this is another skill that takes time to acquire," the clinician observed.

Attribution Style

As the foregoing discussion implies, individuals who confront a challenging task utilize explanatory styles that reflect how they explain success and failure (Weiner, 1985; Alloy, 1988; Abramson, Seligman, & Teasdale, 1978). For example, one can explain her or his success or failure on a task as due to causes that are internal (effort, ability) or external (luck, task difficulty). These causal attributions also vary on the dimension of whether they are stable (ability, task difficulty) or unstable (effort, luck). People prone to depression are more likely to attribute failure to internal, stable qualities (lack of ability), and they sometimes attribute their success to external, variable qualities (luck). When individuals are encouraged to attribute their failure on a specific task to lack of effort and encouraged to try harder next time, the quality of their performance increases (Dweck, 1975). Moreover, explanatory style also reflects whether the focus is on a specific task or is generalized to all tasks—for example, "I didn't do well on this quiz in organic chemistry, but I did well on other quizzes in the course." In addition, explanatory style can also reflect the degree to which the task may be one that others would have done well on or whether only the individual him- or herself does poorly. This is the dimension of "consensus" in the more general attribution model advanced by Weiner (1985) and others. Research on these dimensions of attribution show that depression, self-criticism, and helplessness are linked to the tendency to attribute failure to internal and stable causes and that those attributions for failure generalize to other tasks; furthermore, the individual tends to discount information about how others would respond.

In the preceding case of the frustrated young man, he has immediately jumped to claiming that he lacks the ability, that effort will be of no avail, that others would have done better, and that the task should not be that difficult for him. The clinician was able to draw on his past history to illustrate that he had not even taken on the task yet, that he had overcome obstacles before, that he had received good feedback from former clients on his work, that others in the circumstance of having this dumped on them at the last minute would also be challenged, and that in the past, when he focused and disciplined himself to put in effort, he had succeeded.

Helpful questions that address attributional style include the following, which we have divided into "anticipatory thoughts" and "after-the-fact thoughts":

Anticipatory Thoughts

- "If you begin by believing that you lack the ability or that it is too difficult, would that mean you have already defeated yourself?"
- "Have you been successful on tasks before where you put in a lot of effort?"
- "Could you consider trying to exert more effort first on this task?"

After-the-Fact Thoughts

- "How would you explain not doing well on this task? Would it be due to lack of effort, ability, bad luck, or that the task was just too hard for almost anyone?"
- "Is there something that you learned from this experience that can help you cope better in the future?"

Beliefs about Persistence

A fundamental element of resilience is the willingness to persist on a frustrating and difficult task without giving up. Learned helplessness models view this as "persistence" rather than "helplessness," but we can also view this as involving longer term perspective, delay of gratification, and taking an investment approach (Read & Read, 2004). The ability to replicate a behavior (or an investment) is a good predictor of eventual success in achieving an outcome. For example, consider the difficult task of losing weight and keeping it off. This involves practicing the habit of exercise and proper diet over and over rather than relying on an inspirational "quick and easy" approach. Persistence involves keeping your eye on the goal—whether it is a daily, weekly, monthly, or yearly goal—and practicing the advantageous habits regardless of momentary fluctuations in motivation. Interestingly, an overwhelming majority of people who purchase annual health club memberships only go a few times right after purchasing the membership, believing that their initial motivation while visiting the club will continue to motivate them to go on a regular basis. Stress is exacerbated when the individual takes a short-term view, expecting immediate gratification on a task. Encouraging persistence and replication—and taking a longer term view of rewards that are delayed—may help build both patience and resilience and reduce the experience of stress.

Accepting Limitation

A doctor working in an emergency room is confronted with life-and-death challenges on a regular basis. That is part of the job. Although objectively stressful in some ways, some clinicians are able to cope better than others. The therapist with a severely depressed patient who has experienced multiple hospitalizations, who is often noncompliant with medication, and who has a difficult family environment will recognize that his or her best efforts may have limited effectiveness. Just as the emergency-room doctor recognizes that there is just so much that can be done to help a patient during a medical crisis, the clinician working with psychiatric patients will realize that his or her abilities to "cure" the patient will be limited and that one needs to adjust expectations to be consistent with reality. One difficulty in appraising stressors that one faces is to have a realistic expectation as to what can be accomplished and to accept these limitations of imperfect outcomes. The clinician can assist the client in evaluating the value of accepting some limitation of control and outcome while committing to action toward the stressful event. Clients may be more amenable to accepting limitations when they can recognize the other areas of life in which they currently accept limitations.

PUTTING THINGS IN PERSPECTIVE

A key element in stress is the belief that success at a task is absolutely essential and that failure is catastrophic. For example, a woman studying for an exam indicated that she had fallen behind in the preparation, that she would not do well, and that her failure on this would condemn her to oblivion and she would never get a good job after graduation. Moreover, she defined "success" as getting an "A" and failure as anything less than an "A." The cognitive therapist can utilize a wide range of techniques to help a client put things in perspective (Leahy, 2017). We review several of these techniques next, including the continuum technique, the pie chart, "what I can still do," "compare yourself to a zero point," "observe how others cope," "affirm the negative," and "travel to the future."

Continuum Technique

The individual who is stressed often views performance on a task as having considerable magnitude or importance and failure on the task as being catastrophic or unbearable. Realistically, few things in life are unbearable or catastrophic, but when we are in the grips of anxiety, it often seems that our survival is at stake. The clinician can suggest the following:

THERAPIST: It seems that this task has great importance for you and that you are feeling very stressed right now. So, let's start with rating the degree of stress that you are feeling from 0 to 100, where 100 reflects the most unbearable stress imaginable and 0 is the absence of stress. Where would you put your stress on this task?

CLIENT: It feels like 90%.

THERAPIST: OK. I am going to draw a line from left to right, and we are going to look at different experiences that you can have and rate each one for the stress that you would imagine that you would have. So, let's label the very right end as 100% stress and put this task at 90%. What could happen in your life that would be 100% stress?

CLIENT: Losing my family.

THERAPIST: OK, what would be 80% stress?

CLIENT: (*Pauses.*) I don't know, getting really sick but recovering.

THERAPIST: What would be 70% stress?

CLIENT: A friend getting angry at me.

The therapist then laboriously goes down each 10 points in the scale and notices that the client has difficulty identifying points below 70%.

THERAPIST: I notice that you ranked this at 90%, which is 10% below losing your family. Does that seem reasonable, or would you want to reconsider?

CLIENT: I guess I am viewing it too extremely. Maybe rate it at 50%.

THERAPIST: Why is it not as bad as you initially thought it was?

CLIENT: Well, I am not losing my family, I still have my health and my job and I know that in a while I won't feel as stressed.

Pie Chart Technique

Stress often involves focusing exclusively on a single negative event or factor to the exclusion of other neutral or positive factors. The pie chart technique allows the client to view the current event in the context of other experiences, opportunities, relationships, and sources of reward.

THERAPIST: So, it seems that this one event is really absorbing you and seems to occupy your mind and control how you feel. We often lose sight of other things in our lives when we are upset. Let's do this. I will make a circle that represents a "pie chart" in which sources of reward or meaning in your life get different pieces of the pie. So this piece here represents the current task. What are some sources of reward or meaning other than this task?

CLIENT: My relationship with my wife and my kids.

THERAPIST: OK. So let's give each person a piece of the pie. (*Draws three pieces—wife, daughter, and son.*) Now what other sources of meaning do you have?

CLIENT: Well, I have friends, but I haven't been seeing them as much since I have been feeling depressed.

THERAPIST: OK, so let's add a piece for friends. What other sources of reward do you have?

CLIENT: Well, I like sports. It's kind of an escape watching football and baseball, but I like it.

THERAPIST: OK, so we can add that as well. (Continues to add other pieces—exercise, learning, being part of a community, siblings, travel, sleep, dining out.) As we can see, there are lots of sources of reward in your life other than this one task. What do you make of that?

CLIENT: I guess I have lost my perspective and haven't been seeing that there are many things in life that have meaning for me.

"What I Can Still Do"

With this technique—which can follow the pie chart technique—the clinician can explore all the activities and sources of reward and meaning still available, even if this task is not completed to the standard that the client demands. The clinician can ask the client, "What activities or things that you did in the past month gave you some meaning or reward?" The client can then list as many as she or he can recall—for example, time with friends, partner, children, leisure activities, travel, learning, talking with people at work, and so forth. Then the clinician can ask, "Which of these things will you still be able to do?" followed by "Which will you never be able to do again?" These questions often dramatically change the perspective about the stressful event, as, in most cases, the client is able to do everything she or he ever did before the stressful event. The clinician can ask, "If you can do almost everything you did before, then how essential is this event in your real life?"

"Compare Yourself to a Zero Point"

One source of stress is comparing oneself to an unrealistic standard—often of perfection. As we discussed earlier in comparing maximizers with satisfiers, attempting to achieve close to 100% is often a guarantee of stress, frustration, regret, and self-criticism. The continuum technique helps clients see the range of possible sources of stress, from 0 to 100, and the zero-point technique takes this process a step further.

THERAPIST: We often focus on what we don't have and overlook what we do have. For example, no matter how successful, rich, famous, or attractive someone is, there are always many people doing better on these dimensions. So let's take this a step further. Let's imagine that you are comparing yourself to someone who has nothing—no friends, no relationships, no money, no home, bad health, no intelligence, no personal skills—nothing. Do you see homeless people in the city?

CLIENT: Yes, but I don't really pay attention to them, I have to confess.

THERAPIST: OK, that happens. We tune out what is uncomfortable. So how do you compare to someone who has nothing?

CLIENT: I feel a little ashamed of myself to admit that it never occurs to me. I guess I am fortunate. I do have a job, I have some income and some savings, and I am healthy. I seem to have forgotten about all of this.

THERAPIST: Perhaps we can make a point of remembering each one of these things when we see someone who is less fortunate. Think of that person as a reminder so that you can begin to appreciate what you do have.

Observe How Others Cope

It often happens when we are stressed that we fail to see how other people have coped with frustration, disappointment, and even failure on a task. Normalizing coping with frustration and stress takes away some of the self-imposed stigma and sense of helplessness and can also provide clients with role models of people coping successfully with stress. We suggest looking for models who are coping rather than mastery models. Mastery models are people who easily handle a task without any difficulty and may reinforce unrealistic expectations about stress, whereas coping models normalize the ups and downs of coping but also provide examples of how people are able to ultimately solve problems that initially may seem insurmountable (Bandura, 1982). The problem with using a mastery model that depicts ideal behavior is that it may add to the demoralization of the client, as he or she may believe that imitating this ideal behavior is an impossible task. The coping model, in contrast, displays some of the initial frustration and even negative thinking of the client but also illustrates how this can be overcome with persistence, problem solving, and planning.

THERAPIST: Do you know some people at your work who had some frustration initially but were able to cope with it and eventually make progress?

CLIENT: Yeah, my friend's older sister started in a job like this and felt really frustrated for the first few months—even the first year, I think. But she eventually got the hang of it and learned how to do the work.

THERAPIST: How did she cope with it—how did she get the hang of it?

CLIENT: She put a lot of time into learning things and got some help from someone from work and I think she also took a training that helped her.

THERAPIST: What if you took this view—coping over a period of time, ups and downs, using resources, putting time in. Have you ever coped before with difficult tasks?

CLIENT: Yeah, in college I thought I would flunk the economics course because there was a lot of math. But I studied and got help and I did fairly well in the long run.

AUTOMATIC THOUGHTS THAT ADD TO STRESS

Automatic thoughts are thoughts that occur spontaneously and that are often accompanied by sadness, anger, or anxiety (Beck, Rush, Shaw, & Emery, 1979). In the case of mania, automatic thoughts may be accompanied by euphoric mood and excessive risk taking (Leahy & Beck, 1988; Leahy, 1999; Newman et al., 2002). Automatic thoughts may be categorized into a range of dimensions—for example, mind reading, fortune telling, discounting positives, personalizing, and other dimensions (see Table 17.1). These habitual styles of thinking are then linked to conditional rules or maladaptive assumptions that often take the form of "should" statements, "if–then" statements, or rules. For example, the automatic thought that "He thinks I am a loser" may be linked to a conditional belief—"If people don't like me, then it means I am not worthwhile" or "I should get the approval of everyone."

Thus it is possible that one might incorrectly (or correctly) engage in mind reading—for instance, that someone thinks he or she is a loser—but if the individual does not endorse the conditional belief or "should" statement that gives rise to the automatic thought, then he or she may not care what the other person thinks. In addition, conditional beliefs and "should" statements may be linked to core beliefs about oneself and others—that is, "schemas"—and these more general, deeper, and more lasting beliefs give added credibility and weight to the automatic thoughts and conditional beliefs. For example, the individual with the schema that he or she is incompetent may have the automatic thought that the boss thinks that he or she is a loser, and this triggers the conditional belief that "If someone thinks I am a loser, then there must be something wrong with me." This then triggers the underlying schema of being a loser that precipitates stress—that is, anxiety, sadness, and humiliation. We discuss conditional beliefs and schemas later.

When confronting a challenging task, individuals may activate a wide range of stressful thoughts that augment their experience of stress and their belief in their helplessness in coping. For example, consider the following train of thought by an attorney in considering a legal case that he was working on:

> "I just feel overwhelmed. I have this new client and it is a complicated deal, and I just don't know how I am ever going to get a handle on this. There are so many details, and if I don't get this right, they're going to be really pissed off at me. I haven't worked on something like this for a long time, and it's due next month, and I don't know where to start. You know this other [attorney] in the firm just seems to have it so easy, he just seems to know how to do everything right, but I seem to fumble along and never get anything right. You know if I screw this up they could fire me."

Implicit in this train of thinking is a string of "what-ifs," such as "What if I don't get this done the right way, on time, or in the manner that the client likes?"; "What if the senior partners in the firm are really angry with me?"; "What if they decide that I am not up to the job?"; "What if they fire me?"; "What if I can't get another job?"; "What if I can't pay my mortgage?"; "What if I end up impoverished and a total failure?" These multiple contingencies of bad outcomes—one after another, like dominos falling in a rapid sequence—we refer to as "cost cascades"; that is, one negative after another in a chain reaction. This string of stressful thoughts illustrates many of the typical automatic thought biases that often accompany stress.

TABLE 17.1. Categories of Automatic Thoughts

1. *Mind reading:* You assume that you know what people think without having sufficient evidence of their thoughts. "He thinks I'm a loser."
2. *Fortune telling:* You predict the future negatively: Things will get worse, or there is danger ahead. "I'll fail that exam" or "I won't get the job."
3. *Catastrophizing:* You believe that what has happened or will happen will be so awful and unbearable that you won't be able to stand it. "It would be terrible if I failed."
4. *Labeling:* You assign global negative traits to yourself and others. "I'm undesirable" or "He's a rotten person."
5. *Discounting positives:* You claim that the positive things you or others do are trivial. "That's what wives are supposed to do—so it doesn't count when she's nice to me" or "Those successes were easy, so they don't matter."
6. *Negative filtering:* You focus almost exclusively on the negatives and seldom notice the positives. "Look at all of the people who don't like me."
7. *Overgeneralizing:* You perceive a global pattern of negatives on the basis of a single incident. "This generally happens to me. I seem to fail at a lot of things."
8. *Dichotomous thinking:* You view events or people in all-or-nothing terms. "I get rejected by everyone" or "It was a complete waste of time."
9. *Shoulds:* You interpret events in terms of how things should be, rather than simply focusing on what is. "I should do well. If I don't, then I'm a failure."
10. *Personalizing:* You attribute a disproportionate amount of the blame to yourself for negative events, and you fail to see that certain events are also caused by others. "The marriage ended because I failed."
11. *Blaming:* You focus on the other person as the source of your negative feelings, and you refuse to take responsibility for changing yourself. "She's to blame for the way I feel now" or "My parents caused all my problems."
12. *Unfair comparisons:* You interpret events in terms of standards that are unrealistic—for example, you focus primarily on others who do better than you and find yourself inferior in the comparison. "She's more successful than I am" or "Others did better than I did on the test."
13. *Regret orientation:* You focus on the idea that you could have done better in the past, rather than on what you can do better now. "I could have had a better job if I had tried" or "I shouldn't have said that."
14. *What if?:* You keep asking a series of questions about "what if" something happens, and you fail to be satisfied with any of the answers. "Yeah, but what if I get anxious?" or "What if I can't catch my breath?"
15. *Emotional reasoning:* You let your feelings guide your interpretation of reality. "I feel depressed, therefore, my marriage is not working out."
16. *Inability to disconfirm:* You reject any evidence or arguments that might contradict your negative thoughts. For example, when you have the thought "I'm unlovable," you reject as irrelevant any evidence that people like you. Consequently, your thought cannot be refuted. "That's not the real issue. There are deeper problems. There are other factors."
17. *Judgment focus:* You view yourself, others, and events in terms of evaluations as good–bad or superior–inferior, rather than simply describing, accepting, or understanding. You are continually measuring yourself and others according to arbitrary standards and finding that you and others fall short. You are focused on the judgments of others as well as your own judgments of yourself.

Note. From Leahy (2009). Reprinted by permission.

Let us return to the attorney experiencing stress at work. In the case described, he views any possibility of failure on this task as catastrophic. Moreover, his catastrophic thinking is embedded in his fortune telling—that is, predicting that a series of "terrible" events will unravel. He personalizes any negatives on this task, believing that it is entirely up to him. He does not acknowledge that the client may have unrealistic expectations and that the facts of the case may not entirely support his interests. Thus it is entirely his responsibility. He also shows all-or-nothing thinking, believing that everything has to go well or nothing will work—he is either a success or a failure—rather than being a vulnerable, hard-working but limited human being. He overgeneralizes this difficult task to claim that this is happening in all areas of his life—his work, his colleagues, his marriage, and even his health. He discounts any positives that he has going for him, including praiseworthy work he has done on other cases, clients who have been pleased with him, and even laudatory comments from partners in the firm. He labels himself as a failure by focusing on the anticipated difficulties of this task—and he views any failure as unsuccessful. He compares himself with a fictional ideal lawyer who breezes through complicated cases without any effort. And, finally, he engages in mind reading, believing that he already knows what the client thinks of him, what the senior partners think of him, and what his coworkers think of him.

Similarly, we can see how automatic thoughts function in an intimate relationship. Consider the following negative thought biases in a conflicted couple:

1. *Mind reading:* "He thinks I am boring. He doesn't care about my feelings."
2. *Fortune telling:* "We will have a boring time this weekend. I won't be able to get him to listen to me when I talk about my work."
3. *Catastrophizing:* "It's really awful that we haven't had sex in the past few weeks. This probably means that we will end up getting divorced. If we got divorced my life wouldn't be worth living."
4. *Labeling:* "He's a narcissist with no feelings for anyone else."
5. *Discounting positives:* "Even though he spent time talking to me about my work, I know he only did it because I told him I needed him to listen."
6. *Negative filtering:* "Look at how he just sits there reading his tablet and not showing any interest in me."
7. *Overgeneralizing:* "This seems to be going on over and over without any break. Just one boring time after another because he is wrapped up in himself."
8. *Dichotomous thinking:* "We are either going to have a good time or a lousy time. He's either going to show interest or not show interest."
9. *Shoulds:* "We should be close all the time. After all, we are married. We should spontaneously always want to have sex."
10. *Personalizing:* "His sitting there reading his tablet shows how little interest he has in me."
11. *Blaming:* "He's making me feel worthless, unattractive, and boring."
12. *Unfair comparisons:* "I was watching this romantic movie and I realized that we haven't had that kind of romance since we first dated."
13. *Regret orientation:* "I shouldn't have asked him to watch that movie with me because now it just reminds me of how bad things are."
14. *What if?:* "What if this keeps happening? What if he finds someone else? What if we never have sex again? What if I am miserable for the rest of my life?"
15. *Emotional reasoning:* "I feel really sad and lonely and that must mean that we are drifting apart and he has lost all interest in me."

16. *Inability to disconfirm:* "I know he shows interest and he tells me I am attractive, but that doesn't prove that he really cares."
17. *Judgment focus:* "I don't like the way he sounds, his tone of voice. He is conceited and condescending and he is intolerant of anything I say. He seems selfish to me, narcissistic, unfeeling."

The cognitive therapist can assist clients in reducing their stress by evaluating the validity of each thought—or cognitive distortion. Some helpful techniques for addressing each category of thought are shown in Table 17.2.

TABLE 17.2. Techniques for Reversing Negative Thoughts

Category of biased thinking	Helpful techniques
1. Mind reading	"How do you know what they are thinking? Could they be thinking something else?"
2. Fortune telling	"You are predicting negatives for the future, but is it possible that there could be neutral or positive outcomes? What would be the best, worst, and most likely outcome? How often have you been wrong in the past in your predictions?"
3. Catastrophizing	"You seem to think that what is happening or could happen is awful. Let's take what is currently going on. How could you convince a neutral person that this is truly terrible? What can you still do even if this event has happened? How will you feel about this in a week, month, year, 10 years? Rather than view things as catastrophes, what if you were to view them as frustrating at times or unpleasant at times?"
4. Labeling	"When we label people, we often miss some of their good or neutral qualities. Does your labeling add to your stress? What if you didn't label and simply described the behavior that you are thinking about?"
5. Discounting positives	"You seem to not count some of the positives in the situation or positives about yourself and other people. Does this add to your stress? What is the rationale for not counting positives? What if you took a more balanced view and considered those positives? What would they be?"
6. Negative filter	"You seem to focus at times exclusively on negatives. Let's imagine someone were looking at the situation and they had a different filter and they could only see positives. What positives would they see? How would someone else who is less stressed see things?"
7. Overgeneralizing	"Sometimes you seem to take one incident and then generalize to the entire situation or person. Is this rational or fair? Does over-generalizing make you more discouraged? What if you simply stayed with the one incident?"
8. Dichotomous thinking	"Sometimes you view things as either–or, black or white. Either all good or all bad, for example. What if you thought of things along a continuum, from 0 to 100, and filled in each of the points in between the extremes?"
9. Shoulds	"You often attach moral 'shoulds' to things, or you think that things must be a certain way. This may add to your stress because you have a moralistic and demanding way of viewing things. You might consider replacing your 'should' statements with statements like 'I would prefer this or that,' rather than 'It must be this way' or 'I should do this.'"

(continued)

TABLE 17.2. *(continued)*

Category of biased thinking	Helpful techniques
10. Personalizing	"You add to your stress by taking things personally—whether it is at work or in relationship issues. Could it be that what other people say or do is not personally directed at you, but may be due to other things? Like, at work, perhaps there are demands and expectations that other people have, or they may have their own issues and problems. Or in relationships, it may be that other people are experiencing things unrelated to you or that their response has more to do with their personality than it is about you."
11. Blaming	"When we get frustrated, we often blame other people, but could blaming others or blaming yourself add to your stress? What if you simply described what is happening rather than blame? What if you thought of problem solving rather than blaming?"
12. Unfair comparisons	"We often add to our stress by comparing ourselves with other people whom we idealize and sometimes see as almost perfect. You might consider experimenting with not comparing but simply describing or stating a preference. You might consider even comparing yourself with other people who are doing a lot worse—in other words, rather than compare upward, compare downward."
13. Regret orientation	"We often make decisions that don't have perfect outcomes. But when you focus on regrets, doesn't this add to your stress? What if you thought that you made a past decision with the information you had at the time—that you didn't know everything? What good will regret do? Rather than regret, you might consider how you can make the best of what there is at the present time. Focus on present and future positive action rather than getting stuck in the past."
14. What if?	"You add to your stress by continually focusing on what might happen in the future. We generally don't know what will happen until it actually happens. Just because you are uncertain about the future, it doesn't mean that it will be bad. Rather than get stuck with 'what if?' thoughts, try to focus on what is going on in the present moment. What are some productive actions that you can take today?"
15. Emotional reasoning	"You add to your stress by using your emotions to evaluate things. If you feel anxious, you think bad things will happen. If you feel sad, you think you are helpless and things are hopeless. But using your stressful emotions to judge things or predict the future is irrational and adds to your stress. Stick with the facts, focus on what you can control, and realize that your emotions are not a predictor of facts."
16. Inability to disconfirm	"You often stress yourself out when you come up with thoughts that are not open to facts. For example, when you say, 'Yes, I know those facts, but you don't understand what it is really like,' it gets you stuck in a closed loop of negativity. What facts could be relevant to challenging your thoughts? If you can't evaluate your thoughts with facts, then won't that make your stress worse?"
17. Judgment focus	"A lot of our stress is due to judgments we make about ourselves and others or how we feel judged. Again, this is a choice that you have. You can simply describe in neutral terms what is going on without judging it with 'good' or 'bad.' Think about what 'is' as given and then think about ways to cope effectively. If you judge other people, then you will have more stress. Simply describe their behavior and then describe a preference of what you would like them to do."

EXAMINING AND MODIFYING PROBLEMATIC AUTOMATIC THOUGHTS

The clinician can assist the client in examining her or his habitual, stress-related thoughts and find more adaptive ways of thinking of the situation. In this section, we consider several categories of negative thoughts and provide several techniques to gain a more adaptive perspective.

1. Catastrophizing: Believing it will be terrible if something doesn't work out.
   - "What, exactly, will happen?"
   - "How often have you predicted catastrophes that have not come true?"
   - "What is the worst outcome, the best outcome, and the most likely outcome?"
   - "What could you still do if the most likely outcome did occur?"
   - "How do you think you will feel about this in a month, a year, five years?"
2. Fortune telling: Predicting the future in negative terms without sufficient evidence.
   - "What is the evidence that this outcome will occur?"
   - "How often have your predictions been wrong?"
   - "Exactly what would have to happen for this to occur?"
   - "Are there positive events that could occur?"
3. Personalizing: Believing that negative outcomes are entirely one's own fault or that negative events uniquely happen to oneself.
   - "Are you viewing this as something that happens to you personally rather than something that could happen to anyone?"
   - "What could be some reasons why this is not directed at you personally?"
4. All-or-nothing thinking: Viewing events in dichotomous terms—either all good or all bad—without considering variations or shades of gray.
   - "What is the consequence of viewing events in all-or-nothing terms?"
   - "How would you think and feel if you viewed this in shades of gray or different degrees at different times?"
5. Overgeneralization: Generalizing from one event to many other events without recognizing the wide range of differences of events and experiences.
   - "When you generalize from one event to many other events, does it add to your stress?"
   - "How could you look at this as simply one event?"
   - "If you focused on one event, how could you approach this with realistic problem solving?"
   - "Would it be realistic to take one positive event and then generalize to think everything is positive?"
6. Discounting positives: Discounting any positive things one might think of about oneself as irrelevant or inaccurate.
   - "What is your rationale or reason for not counting some of your positives?"
   - "What would happen if you gave yourself credit for some positives or recognized some positives currently in your life?"
7. Labeling: Labeling oneself, others, or tasks in global negative terms without recognizing the variability that exists.
   - "When you label yourself in these global negative terms, what are the costs and benefits to you?"
   - "You seem rather harsh with yourself. What advice would you give a friend? Would you be as negative and judgmental with them? Why not?"

- "If your best friend were looking at this, what would they think of you? Why would their judgment be less negative than yours?"
- "Rather than label 'the whole person,' what if you were to look at specific behaviors that you could try and see what you could accomplish?"

8. Unfair comparisons: Comparing oneself to others or to standards that are unrealistic and then falling short.
   - "Why do you need to compare yourself to others?"
   - "What are the costs and benefits of comparing?"
   - "You often seem to compare yourself to people who are excelling at something. What if you compared yourself to the entire range of humanity?"
9. Mind reading: Inferring that others are having negative thoughts and feelings about oneself without sufficient evidence that this is true.
   - "You often seem to think that you know what others are thinking. Could it be that your mind reading is not accurate?"
   - "What is the evidence for and against your view that others think negatively of you?"

Conditional Beliefs and Stress

We have described how automatic thoughts may trigger conditional beliefs or assumptions that add to stress. For example, the individual who personalizes the moods of his partner is more likely to have stress in their intimate relationship, but this stress is magnified if he believes that he needs to get approval for everything that he does. Similarly, the person who labels herself as a failure if her work is not perfect may hold the assumption, "If I am not perfect, then I am a failure." We can access the conditional beliefs by using the "downward-arrow technique," which begins with the situation and the automatic thought and keeps inquiring "What would that mean to you?" For example, the worker who is not on top of his or her work might have the thought, "I will never get this done" (fortune telling), which leads to the thought, "If I don't get this done and do a great job, then my boss will be angry" (mind reading, fortune telling), which leads to the next thought, "If my boss is angry with me, then I am a failure" (conditional belief) and "I must always get the approval of everyone" (conditional belief).

Table 17.3 lists examples of conditional beliefs that contribute to stress at work and in relationships.

Challenging Conditional Beliefs

Because conditional beliefs are often a major source of stress, the cognitive therapist will use a wide variety of techniques to test out and modify these beliefs. For example, the belief that "I must get the approval of everyone" is a common source of stress at work and in a range of relationships. The therapist can ask the client the following questions to modify this belief: "How much do you believe this, from 0 to 100? What emotions do you have when you have this belief? What are the costs and benefits of this belief? How would your stress be less if you did not hold this belief? Would you apply this rule to everyone? Does everyone hold this belief—and, if not, why not? Are there people that you admire who do not hold this belief? Are they more or less stressed?" In addition, the clinician can challenge the idea that these situations call for "moralistic" statements that include "should" statements. For example, rather than use moralistic and absolute statements such as "I should get the approval of everyone," the clinician can suggest that a

TABLE 17.3. Conditional Beliefs at Work and in Intimate Relationships

Stressful beliefs at work	Stressful beliefs in intimate relationships
"I must do everything perfectly."	"Our relationship should be perfect."
"It's terrible if I am not the best."	"I should make my partner happy all the time."
"I should always get it right the first time."	"We should never have arguments."
"My work should be exciting and interesting."	"We should have exciting sex all the time."
"I should never be bored."	"My partner should understand my needs without my telling him/her."
"If I don't do a perfect job, I will lose my job."	"Relationships should be easy—it should always come naturally."
"I should criticize myself if I don't do a good job."	"I should tell my partner about every negative thought and feeling I have."

more flexible and realistic thought may reduce stress: "What if you had a more flexible thought such as, 'It would be pleasurable if I got their approval, but it is not necessary'?" The clinician can introduce beliefs that normalize imperfection, disapproval, and even frustration: "What if you considered it normal or inevitable to be imperfect or not get approval all the time? Would that reduce stress?"

STRESS AND PERSONAL SCHEMAS

A key element of the cognitive approach is the recognition that negative automatic thoughts and conditional beliefs gain considerable importance when they are linked to underlying personal schemas about oneself and others. For example, the individual who predicts that she or he will fail to meet the approaching deadline (fortune telling) may also believe that it would be catastrophic if this occurs. Her or his conditional belief might be, "Other people will think I am a failure if I don't do things perfectly" and "I am a failure if I don't live up to expectations." The core belief or schema about the self might be "incompetent." This may then trigger other negative beliefs related to incompetence, such as focusing on disappointments in school, work history, or relationships. Alternatively, another individual might have the same automatic thought ("I will fail to meet the deadline") and a different conditional belief ("If I fail, then others will disown me and abandon me"). This may trigger a core belief, "I am unlovable," which then leads to worry that other people will see the fundamental flaw and reject the person.

According to Beck and colleagues, each of the personality disorders is characterized by core beliefs or schemas about the self. For example, avoidant personality disorder is characterized by feelings of ineptness and incompetence; dependent personality disorder by feelings of weakness and helplessness; obsessive–compulsive personality disorder by feelings of responsibility and fastidiousness; and narcissistic personality disorder by feelings of being special and unique (Beck, Freeman, & Davis, 2014; Leahy, Beck, & Beck, 2005). Thus, for the narcissistic individual, stress may be activated by thoughts that one's special and unique status will be jeopardized and that one will be humiliated. In addition, the narcissistic individual may believe that he or she is entitled to special treatment and should not have to do things that he or she finds unpleasant or boring and that others should defer to him or her. The obsessive–compulsive individual may experience stress due to exaggerated beliefs about control, perfectionism, and performance and may concurrently view others as irresponsible and unreliable. This individual often complains about how unreliable and incompetent coworkers are. The dependent personality has core beliefs about his or her helplessness and the supposed superior competence of other people and will often turn to others to help, reassure, or rescue him or her. The avoidant personality may wish to avoid unpleasant experiences and, therefore, may procrastinate, delay, or escape into more pleasant distractions and fantasies, acting as if the challenging task temporarily does not exist.

As an example, Ken has been working at his company for 12 years and has received mostly good reviews from his supervisors. He now has a challenging project. The deadline is approaching, and Ken now thinks, "I won't get this done on time," "My boss will reject me," "I will get fired," and "I am a failure." His core belief is that he is incompetent and a failure. Ken has attempted to cope with these core beliefs by working long hours, doing more than is asked of him. He believes that if he doesn't do a perfect job he will be exposed as the incompetent that he has always thought himself to be. He activates worry as a strategy to avoid letting his guard down, becoming complacent, and becoming the "lazy, incompetent person I know myself to be."

The link between automatic thoughts, conditional beliefs, and core beliefs or schemas is illustrated in Figure 17.1. We can see how the individual may begin with a schema

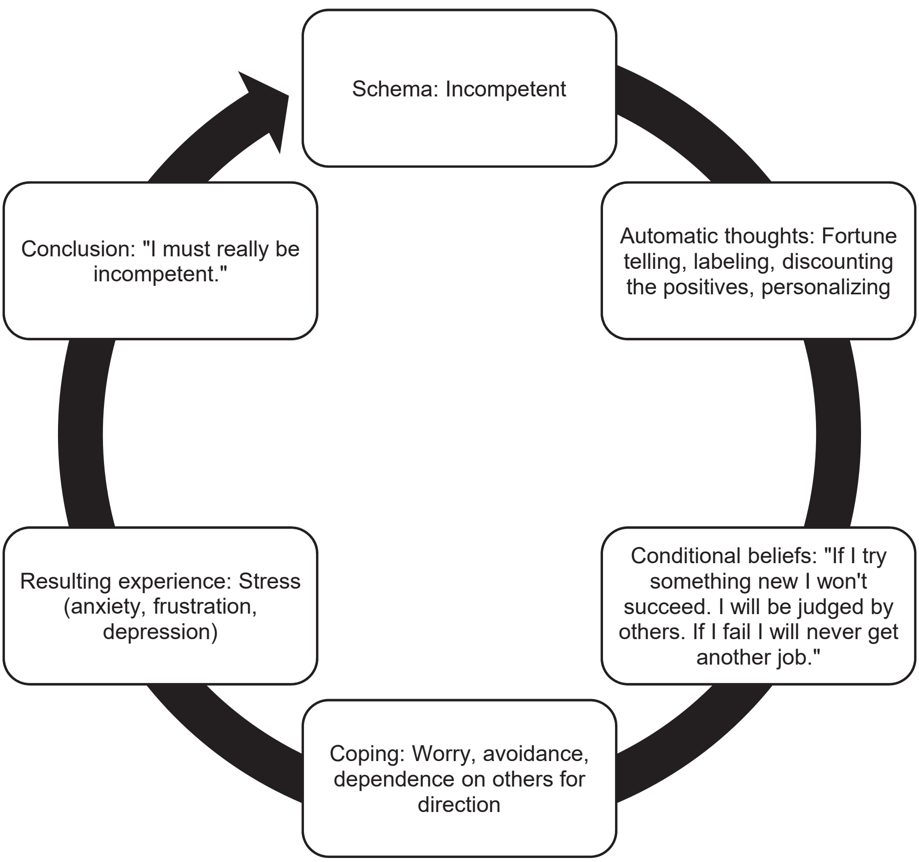


FIGURE 17.1. Cycle of stress for core belief of incompetence.

that she is incompetent and cannot do the job. She then generates a number of negative automatic thoughts: fortune telling ("I will fail"), labeling ("I am helpless"), discounting the positives ("nothing I have done amounts to anything"), and personalizing ("other people must find this easy, why don't I?"). These automatic thoughts then activate the conditional beliefs: "If I try something new, I won't succeed," "I will be judged by others," and "if I fail, I will never get another job." Then the maladaptive coping strategies are activated—worry, avoidance, procrastination, and reassurance seeking. The foregoing augments the stress level, which, in turn, "confirms" the belief that one is incompetent, reinforcing the underlying personal schema of incompetence.

Similarly, the core belief that one is unlovable is illustrated in Figure 17.2. For example, the husband who believes that his wife is not interested in what he is talking about may begin with the core belief or schema that he is unlovable and boring. This then leads to his automatic thoughts of mind reading ("She has no interest"), personalizing ("She is reading, so she doesn't want to talk to me"), and discounting the positives ("Even though we had a nice time last night at dinner, she seems not to be interested in me"). These automatic thoughts are linked to his conditional beliefs that his wife has to be interested in him all the time and that he needs her approval to feel good about himself. He then tries to cope by worrying, seeking reassurance, and avoiding conflict, which only adds to his stress and frustration, which then leads him to confirm his core belief that he is unlovable and boring.

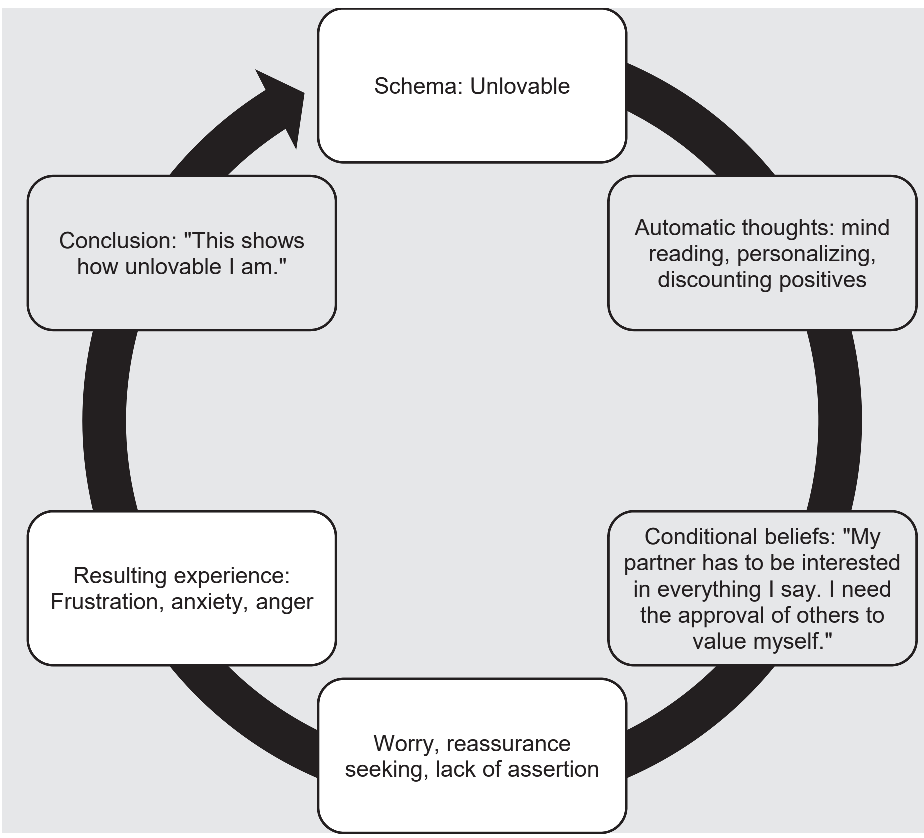


FIGURE 17.2. Cycle of stress for core belief of unlovable.

As indicated above, each level of cognition and coping may be addressed in cognitive therapy. The automatic thoughts and conditional beliefs may be challenged and often replaced with more flexible, less stress-inducing thoughts. The personal schemas can be modified by using a wide variety of cognitive therapy techniques. These include identifying the schemas using the downward-arrow technique and by noticing a pattern of problematic functioning—for example, often getting upset when work activates perfectionistic beliefs or relationships become conflicted over one's need for approval. In addition, personal schemas may be examined by using the cost–benefit analysis, examining the evidence, asking how others view the person, reviewing how the schema was established in the family of origin, and considering more flexible and reasonable beliefs about the self. For example, the individual who was concerned about constant approval from his wife recalled feeling unattractive and awkward as a child and humiliated by other children in school. The clinician can trace the origins of these schemas and then engage the client in role plays in which he is assertive, challenging these "voices from the past." Additionally, he can be encouraged to write out an assertive letter (unsent) to the originators of this negative schema.

EMOTIONAL SCHEMAS AND STRESS

According to the stress-appraisal model advanced by Lazarus, individuals evaluate an expected demand in terms of their capability to cope effectively with the demand (Lazarus & Folkman, 1984; Lazarus, 1999). The demand is referred to as a *stressor,* and the stress is the *experience* that results from the interaction of the stressor and the appraisal. Thus, if I believe that I can cope effectively with a task, I will experience minimal stress, but if I believe that the task is important and that I will not be able to cope, then I will experience greater stress. The emotional schema model advanced by Leahy extends the appraisal model to include cognitive appraisals of emotion or experience and the strategies for coping with the experience of stress (Leahy, 2002, 2011, 2015). The emotional schema model draws on Beck's cognitive model and the metacognitive model advanced by Wells to view emotions (or experience) as an object of experience (Wells, 2009; Beck et al., 1979). Just as the metacognitive model is cognition about cognition, the emotional schema model is cognition about emotion. In addition, the emotional schema therapy (EST) model proposes that strategies of control, expression, and coping result from these interpretations of one's emotions (Leahy, 2015).

The extended emotional schema model is illustrated in Figure 17.3. When the individual experiences an emotion in anticipating or responding to a stressful event, cognitive appraisals of emotion are activated. These include recognition, labeling, and differentiating the emotional experience—for example, "I notice that I am feeling frustrated, anxious and angry." Correspondingly, the individual may believe that these emotions will last a long time (duration), will escalate, and will go out of control. The individual may believe that she or he must eliminate these unpleasant experiences immediately and that they cannot be accepted. Other evaluations of the experience include beliefs about whether the emotions make sense, whether others would feel the same way, feeling guilt and shame over the emotion, and being intolerant of mixed feelings. Alternatively, the individual may label his or her emotions (or frustration) as normal, lasting a limited period of time, controllable, part of the learning experience, and similar to the emotions that others might have. Normalizing unpleasant emotions will lead to less stress about the emotion and less likelihood of recruiting maladaptive coping strategies (Leahy, 2015, 2017).

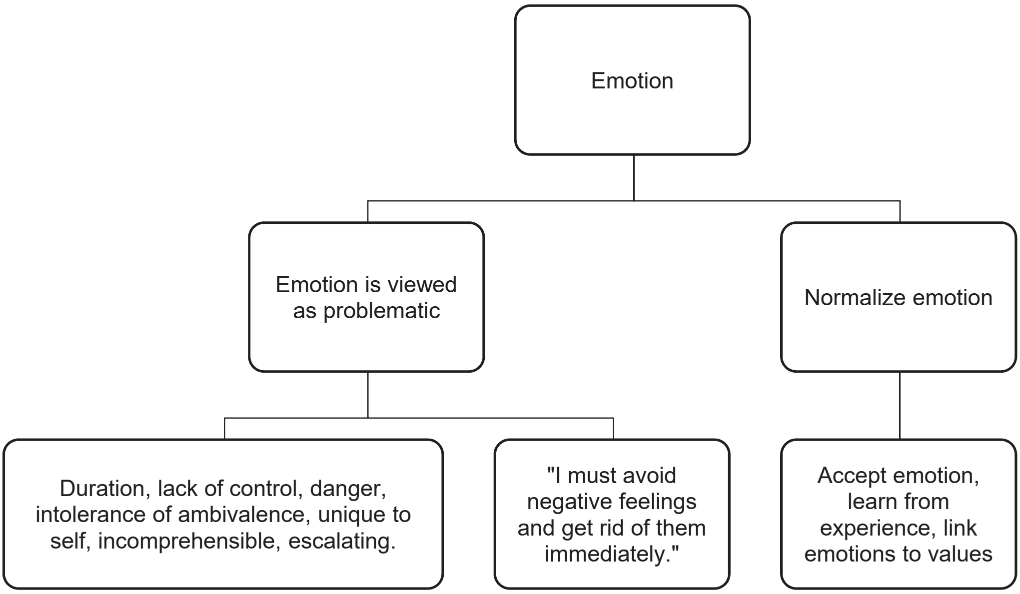


FIGURE 17.3. Emotional schemas and stress.

The clinician can help the client evaluate and test out his or her beliefs about his or her stressful emotions. For example, a young man beginning a job believed that his stress would last indefinitely, that it would escalate out of control, and that this would impair him significantly. He also believed that other people on the job did not experience stress and that he could not understand why he was having such difficulty. These beliefs were tested by charting his stress levels over the course of a week, noting that the level of stress abated once he was actually focused on the work and that it was negligible when he was away from work, with friends, with his partner, or at the health club. He was able to normalize his stress by asking other people at work how stressed they were, whereby he also learned that the most stressful times were in the first few months when the learning curve was steep. His belief that he should be strong enough not to be stressed was related to his shame about his difficulty and his belief that "real men" can handle anything. He was able to make sense of his excessive level of stress given his fear of negative emotions, which was then traced to the fact that his mother had suffered from bipolar disorder and had been hospitalized a year earlier.

EST recognizes the importance of heuristics or rules of thumb that often guide people in inferences, predictions, and decision making (Kahneman, 2011; Leahy, 2015; Wilson, Gilbert, & Centerbar, 2003). One aspect of emotional schemas is affective forecasting, which entails the degree to which the individual predicts the nature, magnitude, and duration of his or her future emotions (Wilson & Gilbert, 2003). For example, in the case described above, this individual predicted that his future negative emotions would escalate, would last indefinitely, and would impair him significantly to the point where suicide seemed an option for him. Predictions about future emotions are often anchored to current emotions and focused on a single element (focalism), and they often ignore future mitigating or adaptive coping factors (immune neglect; Wilson & Gilbert, 2003; Wilson, Wheatley, Meyers, Gilbert, & Axsom, 2000; Gilbert, 1998). The clinician was able to help this individual review his tendency to predict future emotions based on current emotions (emotion heuristic) and to ignore the fact that he would likely learn new skills that he could apply to future work (overcoming immune neglect), as well as the fact that there were other aspects of the work on which he had done well (overcoming focalism). In addition, stress is often exacerbated by time discounting—that is, preference and demands for immediate rewards while discounting the value of higher future rewards (Frederick, Loewenstein, & O'Donoghue, 2002). A consequence of time discounting is a sense of urgency that one must have the reward or the answer immediately. The clinician was able to illustrate this demand for immediate results for this client by suggesting the following analogy: "Let's imagine you wanted to lose 20 pounds. You know you need to reduce calories and increase exercise. How long would it take you to do this?" By using an analogy to incremental progressive gain with replication of behavior, the clinician was able to help the client understand that learning skills at work would require some investment in time and in having the patience to see outcomes that occur later.

PROBLEMATIC STRATEGIES FOR COPING WITH STRESS

Following these negative appraisals of emotional experience, the individual may activate problematic coping strategies to "handle" or "avoid" these emotions. These problematic strategies include avoidance (e.g., procrastination), rumination, worry, reassurance seeking, substance misuse, bingeing, and other unhelpful means. In contrast, a more adaptive set of strategies may be activated, such as cognitive restructuring, behavioral activation, acceptance, mindfulness, and other strategies. Examples of problematic and helpful strategies are shown in Figure 17.4.

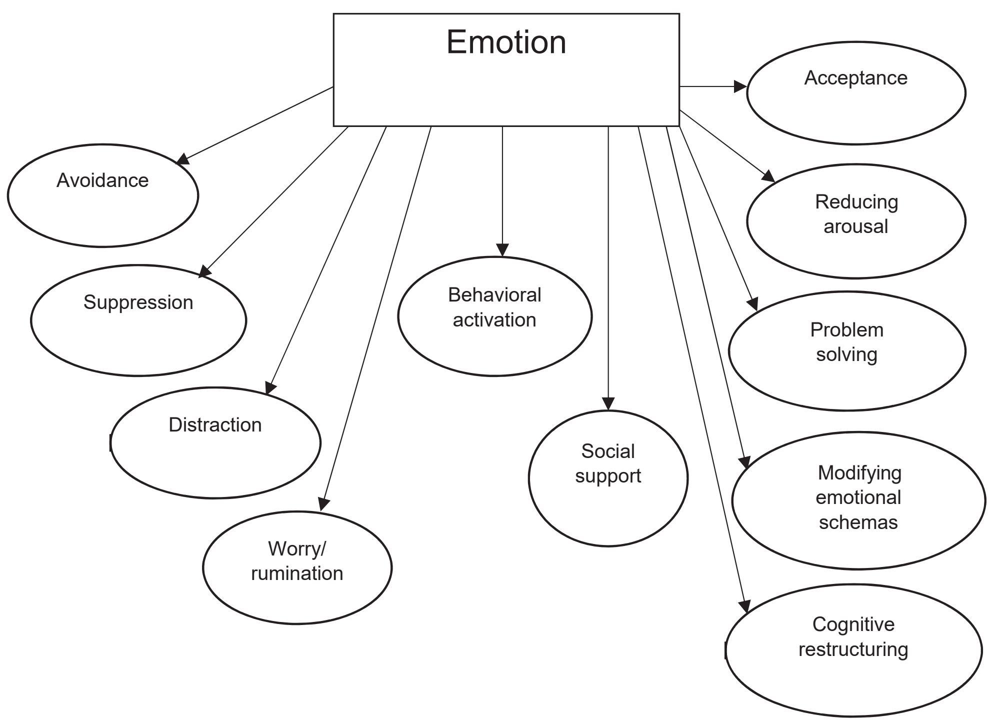


FIGURE 17.4. Strategies of emotion regulation.

The task or challenge that is defined as the "source" (or stress) is only one element in the total stress response. We have identified some of the cognitive distortions and biases that underpin stress, the maladaptive assumptions that are activated that exacerbate the stress response, and problematic beliefs about time urgency, fixed traits, and emotional perfectionism. In this section, we review a number of unhelpful strategies that are often recruited to cope with stress.

Avoidance

The perceived aversive nature of the stressful challenge often results in a person's avoiding confronting or coping with the challenge. The operative automatic negative thought is often, "I can't handle this right now," resulting in the unwillingness to confront the task. Each time the individual thinks of approaching the task, anxiety or stress escalates, which then leads to immediate avoidance and a decrease in anxiety or stress. Following Mowrer's classic two-factor theory, the task is associated with anxiety, and avoidance leads to the decrease in anxiety—combining both acquisition of anxiety through classical conditioning and the maintenance or conservation of anxiety avoidance through operant conditioning (Mowrer, 1956). This reinforcement of the decrease of stress through avoidance leads to an implicit rule: "The way to handle my stress is to avoid." Similar to avoidance learning is the reliance on escape from a stressful situation by quitting or distracting. For example, the individual confronted with the stressor may distract him- or herself with other innocuous activities and note that his or her stress is immediately reduced. The problem that arises is that the source of stress is never resolved.

Procrastination

Related to the two-process learning that underlies avoidance and escape is procrastination. In this scenario, the individual claims that he or she intends to get the work done but continues to put it off until another time. Each time the task is delayed, stress decreases, but as the task begins to loom in the background over a long period of time, the burden of entry to the task becomes greater. Procrastination often involves a range of problematic thoughts, such as "I am too far behind," "It's too late, anyway," "I have to get a lot done if I do it since a small amount won't matter," and "I have other things to do right now." The planning fallacy involves the tendency to predict that plans will be carried out without interference and that everything will be completed on time (Kahneman & Tversky, 1979). The planning fallacy may be part of the procrastination style, such that most of us believe that our plans will eventually be fulfilled, not recognizing how intervening distractions will sidetrack us.

Readiness Demands

The readiness demand reflects the belief that "I cannot start something until am fully motivated and ready to take it on." This is a version of the belief that motivation must precede behavior and that "I have to wait for the motivation to push me forward." The clinician can challenge this readiness demand with the following questions: (1) "What is the consequence for you of believing that you need to be ready?" (2) "What is the worst thing, best thing, and most likely thing that could happen if you started the task without 'feeling ready'?" (3) "What if you saw me walking up and down the sidewalk in front of my office and you asked me, 'What are you looking for?' and I responded, 'I am waiting for my motivation to show up and then I can go to work'?" (4) "Is it possible that starting a task might lead you to develop the motivation and to feel more ready to follow through once you are warmed up and doing something?"

Worry

A common unhelpful strategy in dealing with stress is to worry about the consequences of coping. For example, the client may worry that "If I start working on this, then it won't be any good, and I will get fired." Or the client may worry about being far behind and never coping effectively. Techniques that may prove helpful for worry include examining the costs and benefits of repetitive thinking and distinguishing between productive and unproductive worry— that is, productive worry leads to a to-do list today that moves toward solving the problem, whereas unproductive worry involves concern about something that is not knowable or controllable at the present time. Additional techniques include setting aside worry time so that worry comes under stimulus control, treating intrusive thoughts like telemarketing calls that one does not answer, focusing mindfully on the present moment, and accepting uncertainty (Dugas, Buhr, & Ladouceur, 2004; Leahy, 2005; Wells, 2009).

Emotional Perfectionism

Some clients hold the belief that they should only experience positive emotions when they want them and that emotions such as frustration, boredom, sadness, and ambivalence are unacceptable (Leahy, 2015, 2017). This *emotional perfectionism* adds to stress, as much of life involves tolerating experiences with unpleasant emotions. For example, work may involve frustration and boredom at times, and intimate relationships often entail ambivalence and annoyance. In contrast to this perfectionistic belief about experience and emotion, the clinician may introduce two helpful concepts to cope with stressful experience—successful imperfection and constructive discomfort (Leahy, 2005, 2015, 2017). *Successful imperfection* proposes that one can make progress by gradually doing things imperfectly as one moves toward valued goals. *Constructive discomfort* proposes that discomfort is a means to an end—and even an "investment" in "purchasing success." For example, an individual can reduce his stress in his intimate relationship by realizing that he can imperfectly make progress with his wife in their mutual communication and that he can tolerate some discomfort as he works at making things better. Overcoming the emotional perfectionism that often accompanies stressful experiences allows the individual to normalize the inevitable unpleasantness of some of life's experiences.

HELPFUL STRATEGIES

As indicated in the foregoing discussion, there is a range of useful techniques for coping with the emotion that arises with stress. This includes acceptance of the emotion, reappraising the emotion (e.g., recognizing that it is temporary, controllable, normal, not dangerous), cognitive restructuring of the situation that gives rise to stress (e.g., using cognitive therapy techniques), behavioral activation (e.g., directly engaging in behavior either focused on the task or on rewarding activities), problem-solving strategies, mindfulness, and reducing arousal through relaxation techniques. The question that arises is how problematic interpretations of stressful emotions may result in problematic strategies for coping with the stress. For example, stress is exacerbated if the individual believes that his or her stressful emotions will last indefinitely, cannot be controlled, and will escalate to intolerable levels. Alternatively, the individual who is able to view unpleasant experience as temporary and tolerable will not have the added negative effects of these metacognitive appraisals. The individual who believes that she or he should not have stressful or unpleasant experiences will have difficulty tolerating and compartmentalizing the experience and will be more likely to be angry at the situation in which she or he feels trapped. The client can consider how modifying beliefs about stress can assist in adapting the helpful strategies that are available as alternatives.

INTEGRATING MINDFULNESS INTO COGNITIVE THERAPY FOR STRESS

Much of cognitive therapy for stress aims to change targeted beliefs, such as beliefs about self, others, emotions, thoughts, and methods of coping. The therapist can also take advantage of techniques designed to adjust how one interacts and relates to experience as a whole. Despite differences in emphasis, these methods can be integrated well into change-oriented processes to improve stress coping. Research has found that participants in mindfulness-based interventions experience reductions in stress (Chiesa & Serretti, 2009) and that changes in mindfulness mediate stress reductions (Nyklíček & Kuijpers, 2008). Mindfulness has exhibited consistent inverse relations with stress.

Definitions of Mindfulness

Mindfulness has become both a prominent concept in psychology research and a buzzword in popular culture. Importantly, the meaning of the word *mindfulness* can differ by speaker and audience. Relations between mindfulness and improved mental health have been shown to differ substantially depending upon the components of mindfulness (Woodruff et al., 2014). Unless otherwise specified, use of the word implies consistency with the definitions presented by Kabat-Zinn (1994)—"paying attention in a particular way: on purpose, in the present moment, and nonjudgmentally" (p. 4)—and/or Cardaciotto and colleagues (Cardaciotto, Herbert, Forman, Moitra, & Farrow, 2008)—"the tendency to be highly aware of one's internal and external experiences in the context of an accepting, nonjudgmental stance toward those experiences" (p. 205). Importantly, consistent with these definitions, the clinician aims to foster both awareness of experience and a nonjudgmental orientation toward that experience.

Mindful Awareness and Cognitive Appraisal

The clinician helps the client to improve present-focused awareness and reduce tendencies toward mindlessness or autopilot reactions (Brown & Ryan, 2003). Without active awareness, on the other hand, the client is more vulnerable to errors. The client will be more likely to rely on reflexive, unhelpful interpretations and more poorly equipped to assess factors of the stressor. The "mindless" client will be less able to gauge importance and congruence of a stressor and more vulnerable to schematic interpretations. He or she will have an increased tendency toward simplistic defensive strategies, such as assuming that all stressors are important in a "better safe than sorry" approach, defaulting to interpretations from a recent stressor, equating urgency with importance, equating proximity with importance, and defaulting to others' judgments or behaviors. The client will also be less likely to notice and disengage from rumination and unproductive worry and will miss important new information as a result. He or she may incorrectly estimate coping abilities or prior examples of coping well with other challenging stressors.

With mindful awareness, the client is better equipped to make active, accurate appraisals of a given stressor or to catch cognitive errors. The client will more likely assess the situation through present-focused information, including nuance and contextual factors. He or she will have increased opportunity to build meta-awareness and recognize outdated interpretations. The client can better link the stressor to personal goals and values. In the absence of rumination and unproductive worry behaviors, he or she will more likely integrate new information.

For example, a client who is a rising employee at her firm has spent three late nights working on a high-profile project. During this time, her focus has necessarily drifted away from a low-level project. She began assisting on the project at the request of a colleague, who had asked for help and who continues to lead the project. Currently focused on executing tasks, the client is predominantly inhabiting the "doing mind" (Williams, Teasdale, Segal, & Kabat-Zinn, 2007) and lacks mindful awareness toward her thoughts, feelings, and behaviors. Suddenly, she receives a panicked email from her colleague stating that the materials they prepared contained errors. Thoughts immediately enter her mind, such as "this is a disaster," "if I don't fix this, my promotion is gone," "I shouldn't be so lazy," and "I'm worthless." She further ruminates on other professional mistakes from prior years. Lacking awareness, the client is less likely to experience the thoughts as thoughts, to correctly assess the incongruence and importance of the situation (relatively modest), and to counter rumination with a more adaptive strategy. She may dedicate the rest of the evening to trying to fix a relatively unimportant problem, at the expense of her big assignment, to prove that she is not worthless.

In contrast, the mindfully aware client would be more apt to observe the negative thoughts driving the panic, perhaps recognizing familiar chatter. She would also be better positioned to identify inaccuracies in these thoughts and to reframe thoughts for greater accuracy or compassion. For example, she might think, "I'm feeling anxious and sad about this mistake, but it probably won't end up mattering much"; "I've still been doing great work on my primary project"; or "When I'm working around the clock, mistakes sometimes happen. Bill had a similar thing happen last week." If her mind moved to rumination, she would be better equipped to catch this process and redirect her mind toward beneficial coping strategies and actions that are likely to help the situation (e.g., taking a minute to breathe or meditate, reframing thoughts, and dedicating 15 minutes to help the project leader problem-solve before returning to her primary work).

Similarly, imagine that the romantic partner of the aforementioned client is also working with a therapist. They have identified an unlovable schema and observed that he is prone to become angry, saddened, and anxious by perceived inattention from his partner. When the woman returns home that evening, he asks about work. She gives a curt response, looks down at her phone again, and walks to another room. Lack of care and connection is incongruent with his desires for the relationship, which he deems very important. Thoughts enter his mind, such as "She doesn't care," "Why doesn't she like me?" and "It's happening again!" With such mindless thoughts, the man is more vulnerable to assuming that the brief exchange is emblematic of the relationship as a whole and more likely to see a schema-consistent interpretation as "the fact of the matter." He will be more poorly equipped to notice specific aspects of the situation (she just returned from yet another long day of work and looks tired from poor sleep). He is also more likely to miss the greater context (new responsibilities have led to increased pressure on her) and to fail to notice schema-inconsistent information (she brought home dinner for two from his favorite restaurant). He may default to a familiar critical response, leading to an argument, and finish the evening ruminating on past and present relationship difficulties. The woman ruminates on further evidence that she "can't do anything right."

Alternatively, with mindful awareness, the man might notice his familiar critical thoughts (e.g., "I'm thinking she doesn't care about me—I remember discussing that exact thought at session Tuesday"), as well as the dinner she brought for him. Recognizing his frustration in the moment, he might think to pause before speaking to avoid inflaming the situation. Externally, he might notice the tension on her face as she reads an email. Instead of criticizing, he asks how she is feeling, listens to her discuss difficulties at work, and offers to clean the dishes so she can rest. The couple gets along well and finishes the evening feeling connected.

Mindful Nonjudgment and Cognitive Appraisal

The therapist helps the client foster nonjudgment and acceptance of his or her experience. Judging is fundamental to the process of identifying incongruence, differences between the way things are and the way the client wishes they were. With an emphasis on this discrepancy, the client's judgmental mind can encourage continued focus on areas of disappointment, regardless of the level of their importance.

With nonjudgment, the client is better able to tolerate stressors. Research has found that long-term meditators show less activity in areas of the brain associated with self-referential processing (Hölzel et al., 2011; Wheeler, Arnkoff, & Glass, 2017). With mindful nonjudgment, the client may experience a negative event or challenge as something separate from an existential threat. Nonjudgment allows the client to place greater emphasis on the stressor as it actually is, rather than on the way it is not and should be. With fewer judgments and less incongruence, the client has fewer potential targets for rumination. When facing high incongruence and matters of genuine importance, nonjudgment provides greater distance from the stressors. From an emotional perspective, nonjudgment promotes alternative beliefs to emotional schemas and reduces attributions that promote secondary emotions. The client faces less urgency in reducing negative emotions and, with fewer secondary emotions, a more manageable load to cope with. More resources are left available for problem solving. Equanimity increases the likelihood of handling a situation with skill and building a library of successful coping experiences.

For example, the judgmental client discussed earlier has a presentation with top management from her company. As the presentation approaches, she notices that her heartbeat is rapid, her chest is tight, and her voice quivers subtly. The thoughts "I look like an amateur" and "they'll think I can't handle it" enter her mind. She begins focusing on her anxious thoughts and feelings and, while starting the introduction to her presentation, becomes preoccupied with managing her anxious sensations. She looks down to reduce distress. When that doesn't eliminate the anxiety, she continues to worry about her appearance and loses track of the content that she prepared the prior day. She apologizes, "I'm sorry, I'm very nervous today," in an attempt to commiserate with the audience and express her own frustration at the situation. The presentation continues similarly for several minutes. She remains focused on her anxiety and negative thoughts about her performance. As a result, she continues to lose focus on the content of her talk and halts several times. After the presentation finishes, she replays the moments that she found the worst in her head. She concludes, "I'm a loser" and "I wouldn't be able to handle a big promotion even if they gave it to me." Throughout the evening, she ruminates on her career limitations.

Similarly, the nonjudgmental client notices sensations of anxiety. She observes the sensations and thinks, "I'm feeling anxious about the presentation." When her mind brings up a thought of looking incompetent, she notes "there's a judgment" and returns her focus to the material that she prepared. At various times during the presentation, she notices an anxious sensation and perceives distance from it. After the presentation finishes, she recalls two moments in which she thought members of her audience may have looked distracted. She concludes, "Next time I'll present my conclusions at the start of the talk." During the evening, she notes that she would have preferred to change some things during the presentation but thinks it went well overall.

Building Mindful Awareness and Nonjudgment

Mindful awareness and nonjudgment can be fostered through formal and informal practice. Formal practice includes structured meditation exercises, such as those described in mindfulness-based stress reduction (Kabat-Zinn, 1990). Informal mindfulness practice includes short exercises that are integrated into the client's day-to-day life (e.g., mindful dishwashing). In a small study of undergraduate and graduate students, students who received formal mindfulness instruction experienced a greater decrease in stress than those who received solely informal instruction (Hindman, Glass, Arnkoff, & Maron, 2015). In practice, the therapist can use a combination of both forms of mindfulness instruction.

The clinician can introduce the client to mindfulness exercises in session by leading an extended mindfulness exercise, such as mindfulness of the breath, body scan, or mindful walking. The clinician explains that the exercise is intended to allow the client to nonjudgmentally observe his or her experience, not to feel a certain way (although clients may remark about feeling increased calm or focus during an exercise). During the practice, the therapist encourages the client to focus on a particular experience, such as the breath, and gently return focus without judgment when the mind wanders. Following the exercise, the therapist leads the client through a series of questions about what was observed during the practice. The therapist takes care to highlight examples of inattention and judgment that the client recounts with an accepting attitude toward the inattention and nonjudgment. For example:

THERAPIST: What did you observe during the exercise?

CLIENT: I had a hard time. It was really difficult to focus on anything.

THERAPIST: You noticed your mind was wandering to different topics?

CLIENT: Yeah. First I kept thinking about a conversation I had with my boss. Then about what I need to do when I get back to the office.

THERAPIST: Did you bring the focus back to the breath?

CLIENT: For a little bit, then another thought would come back about what I should have said to my boss.

THERAPIST: So, it sounds like your mind would wander—as minds do—and after some time you noticed that and brought attention back to your breath. That's mindfulness practice.

CLIENT: I guess. I didn't think of that at the time. Eventually I started thinking about how I couldn't focus.

THERAPIST: It sounds like there might have been some judgment there as well.

CLIENT: Yeah. It's frustrating that I can't seem to do anything right.

THERAPIST: That's a judgment. When the mind moves to a judgment, can you just note that judgment and do your best to gently return your focus back to the breath?

CLIENT: So, whatever comes up, I'm just trying to return my focus on the breath?

THERAPIST: Yes. Just do your best to return your focus to the breath.

The therapist can use problem solving to help the client build a structured practice. Elements such as determining the best time for practice, identifying an appropriate location, and working around interpersonal barriers to practice (e.g., sharing a small space with a partner) can help a novice meditator build a consistent mindfulness practice (Ameli, 2014). If the client fails to practice consistently, the clinician encourages a nonjudgmental attitude for both the clinician and client when identifying factors that hindered independent practice. External resources, such as apps, mindfulness studios, and temples can help the client cement mindfulness practice in between sessions. It is highly recommended that the clinician direct the client to familiar resources or inquire about resources the client is already using to vet them for consistency and appropriateness. Several mindfulness programs, such as those at the University of California San Diego Center for Mindfulness and the University of California Los Angeles Mindful Awareness Research Center, provide research-based, guided meditations online at no charge.

Preexisting Experience and Understanding of Mindfulness

It is common to find that clients arrive to therapy with a background knowledge of mindfulness and/or meditation. However, it is also common that the practice of novice and experienced meditators alike has failed to emphasize some of the most beneficial aspects of mindfulness. Popular apps and nonclinical instructors often emphasize the potential for mindfulness to change one's experience, such as increasing calm, feeling better, and improving focus. This can lead the client to develop beliefs such as the following:

- "I'm not good at meditating": Few clients have an innate ability to maintain focus and nonjudgment. Practice aims to foster nonjudgment of experience, including an accepting attitude toward the exercise itself, not to "win mindfulness."
- "I did it, but nothing happened": It would be unusual for something dramatic to happen. The task is simply to observe the present moment, whatever that entails.
- "I had to stop because I kept getting distracted": All minds get distracted. The aim is to return attention in a nonjudgmental manner when wandering is observed.
- "I must have been doing it wrong, because I felt more anxious": Meditation will often increase awareness of anxiety. Again the task is to apply an accepting lens toward this and all experience.
- "It didn't work. I still felt ______": Mindfulness is not designed to eliminate negative feelings or thoughts. In some cases, this may occur as a by-product of the practice.
- "I just need to stay positive": This is not mindfulness, or cognitive therapy for that matter.

The therapist uses continued psychoeducation to counter these notions and models nonjudgment and awareness during the session. Lazarus and Folkman (1984) developed a cognitive model of stress and coping with two stages of appraisal. Under this model, primary appraisal is the process of estimating the perceived importance of a stressor. Secondary appraisal is the process of estimating one's perceived ability to manage the stressor. For example, toward the end of an interview, a job seeker receives a difficult question that he had failed to anticipate. During primary appraisal, he assesses the importance of the question. Does the interview's outcome ride on his response, or has the deal already been sealed by his earlier responses and background? Is the job one that he is likely to accept, or does he strongly prefer the two positions he interviewed for last week? Does he need to earn money immediately, and is career important to him? During secondary appraisal, he estimates how well he can manage the situation. Is he capable of responding well on his feet, or does he typically falter without time to prepare? Can he gracefully stall while he figures out an answer, or is his repertoire limited to a panicked stare? Does he have the skills to answer tough questions, or does he consider himself to generally lack intelligence? The interaction between these areas—importance of the stressor and his perceived ability to handle it—influences his perception of the situation as threatening or possibly a challenge with potential reward.

The process of coping is similarly broken into two areas: emotion-focused and problem-focused coping. The former includes efforts to regulate internal emotional experience, and the latter includes efforts to manage the stressor. Whereas the original model emphasized the role of negative emotions, subsequent research suggested that positive emotions also hold an important role in the process (Folkman, 2008). Much of CBT is concerned with directly influencing methods of emotion-focused coping, with much of problem-focused coping benefiting indirectly from CBT.

RESEARCH ON CBT AND STRESS

Since the previous edition of this book, numerous clinical studies have continued to document the benefits of CBT on stress in a variety of populations. Several recent studies examined the ability of CBT interventions to improve stress for adults in high-stress occupations or situations across a range of populations. A Swedish trial examined the benefits of an 8-week CBT treatment for parents of children with a chronic health condition (Anclair, Lappalainen, Muotka, & Hiltunen, 2018). Participants exhibited significant improvements in perceived stress and burnout, and effect sizes were large (similar results were found for a mindfulness-based intervention). In a Chinese study, 400 new mothers received five sessions of telephone-based CBT (Ngai, Wong, Chung, & Leung, 2016). Participants reported significantly lower levels of parenting stress at both 6 weeks and 6 months after birth relative to mothers receiving routine postpartum care. A study of approximately 100 elementary school teachers in Hong Kong examined the impact of a 12-session CBT program (Tsang et al., 2015). Participants who received CBT showed a significant reduction in stress and cortisol levels relative to a comparison group. A trial of 100 British government workers examined mechanisms of improvement in a brief CBT intervention modeled on acceptance and commitment therapy (Lloyd, Bond, & Flaxman, 2013). Participants receiving the intervention experienced significant improvements in strain and emotional exhaustion compared with the control group. Improvements were mediated by psychological flexibility, the tendency to prioritize deeply held values over internal discomfort.

Benefits extended to samples chosen on the basis of symptomatology. In a Spanish study, a sample of participants reporting high perceived stress completed 14 sessions of CBT for stress (Santos-Ruiz, Robles-Ortega, Pérez-García, & Peralta-Ramírez, 2017). Participants reported improvements in perceived stress and vulnerability to stress. In a Romanian study, 30 adults with high stress completed a 12-session CBT protocol including elements of mindfulness (Holdevici & Crăavciun, 2015). Both stress and cortisol levels were significantly improved. A sample of 80 Jordanian students with depressive symptomatology were randomly assigned to receive either CBT or no treatment (Hamdan-Mansour, Puskar, & Bandak, 2009). Similarly, during a study of 180 university students with test anxiety, participants were assigned to receive one of two variations of a 5-week group CBT program (CBT combined with relaxation or imagery rescripting) or a self-help alternative (Reiss et al., 2018). Although state anxiety improved during a stressor, physiological stress responses did not exhibit significant changes. Following 10 sessions, the CBT group reported significant improvements in stress relative to controls. In addition, a meta-analysis examined 25 studies of CBT for caregivers of patients with dementia (Hopkinson, Reavell, Lane, & Mallikarjun, 2018). Results indicated that stress improved in participants, with a small to medium effect size.

Multiple studies have also assessed the impact of CBT interventions on patients with medical conditions. An Iranian trial studied the effects of 12 sessions of CBT on pregnancy-related stress in women with preeclampsia (Asghari, Faramarzi, & Mohammadi, 2016). Participants exhibited significant reductions in stress relative to preintervention and no-treatment controls. Another Iranian study assessed the impact of CBT-based stress management intervention on women with infertility (Solati, Ja'Farzadeh, & Hasanpour-Dehkordi, 2016). Following 10 sessions of CBT, participants reported improvements in marital satisfaction and more improvements on measures of stress than a no-treatment control group. A Spanish study examined the contribution of a 10-session CBT intervention for a sample of 70 patients with lupus (Navarrete-Navarrete et al., 2010). Participants receiving the intervention exhibited a significant improvement in levels of perceived stress relative to controls. However, a meta-analysis of CBT for breast cancer survivors was equivocal. Improvements in stress narrowly missed significance, possibly due to the sample of only six studies (Zhang, Huang, Feng, Shao, & Chen, 2017).

There is some evidence that a CBT treatment for stress may also be effective when delivered online. A trial of 100 adults with adjustment disorder and exhaustion disorder examined the impact of an Internet-based CBT program (Lindsäter et al., 2018). Participants who received the CBT program demonstrated significant improvements in perceived stress than did wait-list controls, and improvements in stress were consistent, with a large effect size. In addition, an online CBT program for British university students with debt was found to significantly decrease measures of stress in a small sample (Smail, Elison, Dubrow-Marshall, & Thompson, 2017). Taken together, results indicate that CBT is beneficial in reducing the impact of stress across a wide range of populations, environments, and possibly modalities.

LIMITATIONS AND CONTRAINDICATIONS

The cognitive model—especially as described by Beck and Ellis—can, in the hands of less empathic and sensitive therapists, appear disputatious and invalidating. Indeed, this was a major concern that Leahy (2001) described in *Overcoming Resistance in Cognitive Therapy*. The clinician runs the risk of sounding didactic and dismissive—or even condescending—in "challenging" or "refuting" the beliefs that the stressed client may express. Throughout one's work with clients, it is imperative at most—if not all—times to empathize, be nonjudgmental, and validate the client's thoughts and comments, while recognizing that clients who are stressed about life events do not need to add to their stress by engaging in a debate with a therapist. Indeed, in supervising therapists in the CBT tradition, this is a recurring issue that we address in our own work with trainees. We recognize that the human relationship and the respect for and acceptance of the client's feelings take precedence over the "truth" and "rationality" of cognitive content. This is especially true for clients whose emotion regulation and interpersonal coping is compromised. It is not unusual for some clients to view "rational responses" as "taking sides" against the client. We find that bringing this up in therapy sessions can help "inoculate" the client from this sense of being criticized or marginalized. For example, saying, "There is often a dilemma in this kind of work where I might suggest that there is a different way to view things, but you might interpret this as my being critical of you or in taking sides against you. That is not my intention. I hope that you can share with me any feelings you have that I might be invalidating you or seeming condescending or dismissive of you. Your feelings are of central importance."

Similarly, we recognize that mindfulness and acceptance, even in the hands of experienced and well-meaning clinicians, can appear to be trivializing to some clients. In fact, it is important to realize that difficult challenges are quite real and cannot be minimized simply through acceptance or mindful detachment. Sometimes, the struggle to cope with unfairness and impossible demands is realistically stressful. Sometimes, no matter what one does, no matter what the best advice is, and no matter how much one tries putting things in perspective and accepting that things are difficult—things really *are* difficult. Each of these tools from each of these therapeutic orientations can be helpful in coping with the problem of stress. But, in the last analysis, the problem may persist.

Finally, we recognize that the tools available from a modern integrative CBT approach may go far, but not far enough for many clients. In these cases, medication consultations are indicated. A wide range of medications addressing different symptoms such as insomnia, attention deficits, depression, and anxiety can provide the additional support that some clients may need to cope with their stress. For many, this may be viewed as a temporary support, but for some it may be a longer lasting need. Ongoing evaluations of stress and need for additional tools is part of the clinical picture for some clients.

CONCLUDING COMMENTS

Stress is a general term that can refer to the difficulties, frustration, anger, anxiety, depression, helplessness, and hopelessness that the individual can experience. Cognitive therapy—especially the expanded cognitive-behavioral model described in this chapter—can provide the clinician with a wide range of conceptualizations of stress, descriptions of cognitive biases that contribute to stress, models of emotion regulation that help us understand what can perpetuate even more stress—especially stress over being stressed—and strategies and techniques for reducing stress. No one technique will work for every client. But the research on CBT for stress is quite promising.

The growing body of empirical evidence supports the effectiveness of cognitive and more integrative CBT approaches. Indeed, it is difficult to find a DSM diagnosis for which CBT hasn't been shown to add some advantage. Because stress is often accompanied by anxiety, depression, rumination, worry, anger, substance abuse, relationship conflict, and other significant problems, the techniques and conceptualizations that can be derived from these CBT models are especially relevant to treating stress. Moreover, because the emphasis is on helping clients acquire self-help skills, the use of CBT contributes to the sense of self-efficacy that clients can experience.

The cognitive model—now often referred to as CBT—has advanced significantly since Beck and colleagues' (1979) model in *Cognitive Therapy of Depression*. This modern CBT model includes the foundational model that Beck and colleagues proposed. We can see the importance of automatic thoughts, maladaptive assumptions, and personal and interpersonal schemas. However, a cognitive model is not limited to any particular taxonomy of cognitive content. Here, we have suggested including the roles of overgeneralized thinking, looming vulnerability, maximizing versus satisfying beliefs, fixed versus growth beliefs about skill, attribution style, beliefs about persistence, metacognitive beliefs, emotional schemas, emotional perfectionism, affective forecasting, emotion regulation strategies, and mindfulness. Each of these content areas has strong empirical support as contributing to the experience of stress and significantly expands the relevant content of a modern CBT model. We can think of this as an integrative approach to the various processes underlying stress.

An advantage of such an expanded model, as illustrated in this chapter, is that the clinician can match the interventions to the needs of a specific client. Our approach is not limited to a specific model or content, but rather views stress as a multifaceted experience affected by a wide range of processes. Indeed, the processes underlying stress can also be viewed in a time sequence such that the experience of stress may be initially affected by the content of standards and expectations, automatic thoughts, and schemas, later by negative appraisals of ability to cope (e.g., fixed beliefs), and still later by emotional schemas in which the experience of stress is viewed as long-lasting rather than temporary and as incapacitating rather than inconvenient. By providing this multifaceted CBT model, the clinician and the client are empowered in coping with stress.

REFERENCES

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87,* 49–74.

Alloy, L. B. (Ed.). (1988). *Cognitive processes in depression*. New York: Guilford Press.

Ameli, R. (2014). *25 lessons in mindfulness: Now time for healthy living*. Washington, DC: American Psychological Association.

Anclair, M., Lappalainen, R., Muotka, J., & Hiltunen, A. J. (2018). Cognitive-behavioural therapy and mindfulness for stress and burnout: A waiting-list controlled pilot study comparing treatments for parents of children with chronic conditions. *Scandinavian Journal of Caring Sciences, 32*(1), 389–396.

Asghari, E., Faramarzi, M., & Mohammadi, A. (2016). The effect of cognitive-behavioural therapy on anxiety, depression and stress in women with preeclampsia. *Journal of Clinical and Diagnostic Research, 10,* QC04–QC07.

Bandura, A. (1982). Self-efficacy mechanism in human agency. *American Psychologist, 37*(2), 122.

Beck, A. T., Freeman, A., & Davis, D. D. (2004). *Cognitive therapy of personality disorders* (2nd ed.). New York: Guilford Press.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness and its role in psychological well-being. *Journal of Personality and Social Psychology, 84*(4), 822.

Cardaciotto, L., Herbert, J. D., Forman, E. M., Moitra, E., & Farrow, V. (2008). The assessment of present moment awareness and acceptance: The Philadelphia Mindfulness Scale. *Assessment, 15*(2), 204–223.

Chiesa, A., & Serretti, A. (2009). Mindfulness-based stress reduction for stress management in healthy people: A review and meta-analysis.

Journal of Alternative and Complementary Medicine, 15(5), 593–600.

Dugas, M. J., Buhr, K., & Ladouceur, R. (2004). The role of intolerance of uncertainty in the etiology and maintenance of generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 143–163). New York: Guilford Press.

Dweck, C. S. (1975). The role of expectations and attributions in the alleviation of learned helplessness. *Journal of Personality and Social Psychology, 31,* 674–685.

Dweck, C. S. (2006). *Mindset: The new psychology of success*. New York: Random House.

Folkman, S. (2008). The case for positive emotions in the stress process. *Anxiety, Stress, and Coping, 21*(1), 3–14.

Frederick, S., Loewenstein, G., & O'Donoghue, T. (2002). Time discounting and time preference: A critical review. *Journal of Economic Literature, 40,* 351–401.

Gilbert, P. (1998). The evolved basis and adaptive functions of cognitive distortions. *British Journal of Medical Psychology, 71,* 447–463.

Hamdan-Mansour, A., Puskar, K., & Bandak, A. (2009). Effectiveness of cognitive-behavioral therapy on depressive symptomatology, stress and coping strategies among Jordanian university students. *Issues in Mental Health Nursing, 30,* 188–196.

Hindman, R. K., Glass, C. R., Arnkoff, D. B., & Maron, D. D. (2015). A comparison of formal and informal mindfulness programs for stress reduction in university students. *Mindfulness, 6*(4), 873–884.

Holdevici, I., & Crăciun, B. (2015). Cognitive-behavioral therapy interventions and mindfulness in diminishing the stress level and cortisol blood level. *Procedia—Social and Behavioral Sciences, 187,* 379–383.

Hölzel, B. K., Carmody, J., Vangel, M., Congleton, C., Yerramsetti, S. M., Gard, T., et al. (2011). Mindfulness practice leads to increases in regional brain gray matter density. *Psychiatry Research: Neuroimaging, 191*(1), 36–43.

Hopkinson, M., Reavell, J., Lane, D., & Mallikarjun, P. (2018). Cognitive-behavioral therapy for depression, anxiety, and stress in caregivers of dementia patients: A systematic review and meta-analysis. *Gerontologist, 59*(4), e343–e362.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain and illness*. New York: Delacorte.

Kabat-Zinn, J. (1994). *Wherever you go, there you are: Mindfulness meditation in everyday life*. New York: Hyperion.

Kahneman, D. (2011). *Thinking, fast and slow.* New York: Farrar, Straus & Giroux.

Kahneman, D., & Tversky, A. (1979). Intuitive prediction: Biases and corrective procedures. *Management Science, 12,* 313–327.

Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping.* New York: Springer.

Leahy, R. L. (1999). Decision-making and mania. *Journal of Cognitive Psychotherapy, 13,* 1–23.

Leahy, R. L. (2001). *Overcoming resistance in cognitive therapy.* New York: Guilford Press.

Leahy, R. L. (2002). A model of emotional schemas. *Cognitive and Behavioral Practice, 9*(3), 177–190.

Leahy, R. L. (2005). *The worry cure: Seven steps to stop worry from stopping you*. New York: Harmony Books.

Leahy, R. L. (2009). *Anxiety free: Unravel your fears before they unravel you.* Carlsbad, CA: Hay House.

Leahy, R. L. (2011). Emotional schema therapy: A bridge over troubled waters. In J. Herbert & E. Forman (Eds.), *Acceptance and mindfulness in cognitive behavior therapy: Understanding and applying the new therapies* (pp. 109–131). New York: Wiley.

Leahy, R. L. (2015). *Emotional schema therapy.* New York: Guilford Press.

Leahy, R. L. (2017). *Cognitive therapy techniques: A practitioner's guide.* New York: Guilford Press.

Leahy, R. L., & Beck, A. T. (1988). Cognitive therapy of depression and mania. In R. Cancro & A. Georgotas (Eds.), *Depression and mania* (pp. 517–537). New York: Elsevier.

Leahy, R. L., Beck, A. T., & Beck, J. S. (2005). Cognitive therapy of personality disorders. In S. Strack (Ed.), *Handbook of personology and psychopathology: Essays in honor of Theodore Millon* (pp. 442–461). New York: Wiley.

Lindsäter, E., Axelsson, E., Salomonsson, S., Santoft, F., Ejeby, K., Ljótsson, B., et al. (2018). Internet-based cognitive behavioral therapy for chronic stress: A randomized controlled trial. *Psychotherapy and Psychosomatics, 87*(5), 1–10.

Lloyd, J., Bond, F., & Flaxman, P. (2013). The value of psychological flexibility: Examining psychological mechanisms underpinning a cognitive behavioural therapy intervention for burnout. *Work and Stress, 27,* 1–19.

Mowrer, O. H. (1956). Two-factor learning theory reconsidered, with special reference to secondary reinforcement and the concept of habit. *Psychological Review, 63*(2), 114–128.

Navarrete-Navarrete, N., Peralta-Ramírez, M. I., Sabio-Sánchez, J. M., Coín, M. A., Robles Ortega, H., Hidalgo-Tenorio, C., et al. (2010). Efficacy of cognitive behavioural therapy for the treatment of chronic stress in patients with lupus erythematosus: A randomized controlled trial. *Psychotherapy and Psychosomatics, 79*(2), 107–115.

Newman, M. G., Zuellig, A. R., Kachin, K. E., Constantino, M. J., Przeworski, A., Erickson, T., et al. (2002). Preliminary reliability and validity of the Generalized Anxiety Disorder Questionnaire–IV: A revised self-report diagnostic measure of generalized anxiety disorder. *Behavior Therapy, 33*(2), 215–233.

Ngai, F. W., Wong, P. W. C., Chung, K. F., & Leung, K. Y. (2016). The effect of telephone-based cognitive-behavioural therapy on parenting stress: A randomised controlled trial. *Journal of Psychosomatic Research, 86,* 34–38.

Nyklíček, I., & Kuijpers, K. F. (2008). Effects of mindfulness-based stress reduction intervention on psychological well-being and quality of life: Is increased mindfulness indeed the mechanism? *Annals of Behavioral Medicine, 35*(3), 331–340.

Read, D., & Read, N. L. (2004). Time discounting over the lifespan. *Organizational Behavior and Human Decision Processes, 94*(1), 22–32.

Reiss, N., Warnecke, I., Tibubos, A., Tolgou, T., Luka-Krausgrill, U., & Rohrmann, S. (2018). Effects of cognitive-behavioral therapy with relaxation vs. imagery rescripting on psychophysiological stress responses of students with test anxiety in a randomized controlled trial. *Psychotherapy Research: Journal of the Society for Psychotherapy Research, 29*(8), 974–985.

Riskind, J. H. (1997). Looming vulnerability to threat: A cognitive paradigm for anxiety. *Behaviour Research and Therapy, 35*(8), 685–702.

Riskind, J. H., Black, D., & Shahar, G. (2010). Cognitive vulnerability to anxiety in the stress generation process: Interaction between the looming cognitive style and anxiety sensitivity. *Journal of Anxiety Disorders, 24*(1), 124–128.

Riskind, J. H., Williams, N. L., Gessner, T. L., Chrosniak, L. D., & Cortina, J. M. (2000). The looming maladaptive style: Anxiety, danger, and schematic processing. *Journal of Personality and Social Psychology, 79*(5), 837.

Santos-Ruiz, A. I., Robles-Ortega, H., Pérez-García, M., & Peralta-Ramírez, M. (2017). Effects of the cognitive-behavioral therapy for stress management on executive function components. *Spanish Journal of Psychology, 20,* 1–9.

Schwartz, B., Ward, A., Monterosso, J., Lyubomirsky, S., White, K., & Lehman, D. R. (2002). Maximizing versus satisficing: Happiness is a matter of choice. *Journal of Personality and Social Psychology, 83*(5), 1178–1197.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse.* New York: Guilford Press.

Smail, D., Elison, S., Dubrow-Marshall, L., & Thompson, C. (2017). A mixed-methods study using a nonclinical sample to measure feasibility of ostrich community: A web-based cognitive behavioral therapy program for individuals with debt and associated stress. *JMIR Mental Health, 4,* E12.

Solati, K., Ja'Farzadeh, L., & Hasanpour-Dehkordi, A. (2016). The effect of stress management based on group cognitive-behavioural therapy on marital satisfaction in infertile women. *Journal of Clinical and Diagnostic Research, 10,* VC01–VC03.

Tsang, H., Cheung, W., Chan, A., Fung, K., Leung, A., & Au, D. (2015). A pilot evaluation on a stress management programme using a combined approach of cognitive behavioural therapy (CBT) and complementary and alternative medicine (CAM) for elementary school teachers. *Stress and Health: Journal of the International Society for the Investigation of Stress, 31,* 35–43.

Weiner, B. (1985). An attributional theory of achievement motivation and emotion. *Psychological Review, 92*(4), 548.

Wells, A. (2009). *Metacognitive therapy for anxiety and depression.* New York: Guilford Press.

Wheeler, M. S., Arnkoff, D. B., & Glass, C. R. (2017). The neuroscience of mindfulness: How mindfulness alters the brain and facilitates emotion regulation. *Mindfulness, 8*(6), 1471–1487.

Williams, J. G., Teasdale, J. D., Segal, Z. V., & Kabat-Zinn, J. (2007). *The mindful way through depression: Freeing yourself from chronic unhappiness.* New York: Guilford Press.

Williams, J. G., Teasdale, J. D., Segal, Z. V., & Soulsby, J. (2000). Mindfulness-based cognitive therapy reduces overgeneral autobiographical memory in formerly depressed patients. *Journal of Abnormal Psychology, 109*(1), 150–155.

Wilson, T. D., & Gilbert, D. T. (2003). Affective forecasting. *Advances in Experimental Social Psychology, 35,* 345–411.

Wilson, T. D., Gilbert, D. T., & Centerbar, D. B. (2003). Making sense: The causes of emotional evanescence. In I. Brocas & J. D. Carrillo (Eds.), *The psychology of economic decisions: Vol. 1. Rationality and well being* (pp. 209–233). New York: Oxford University Press.

Wilson, T. D., Wheatley, T., Meyers, J. M., Gilbert, D. T., & Axsom, D. (2000). Focalism: A source of durability bias in affective forecasting. *Journal of Personality and Social Psychology, 78*(5), 821.

Woodruff, S. C., Glass, C. R., Arnkoff, D. B., Crowley, K. J., Hindman, R. K., & Hirschhorn, E. W. (2014). Comparing self-compassion, mindfulness, and psychological inflexibility as predictors of psychological health. *Mindfulness, 5*(4), 410–421.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit formation. *Journal of Comparative Neurology and Psychology, 18*(5), 459–482.

Zhang, M., Huang, L., Feng, Z., Shao, L., & Chen, L. (2017). Effects of cognitive behavioral therapy on quality of life and stress for breast cancer survivors: A meta-analysis. *Minerva Medica, 108,* 84.

SECTION D

HYPNOTIC METHODS

CHAPTER 18

The Autogenic Training Method of J. H. Schultz

Wolfgang Linden

HISTORY OF THE METHOD

In the practice of stress management, autogenic training (AT) is and remains a "classic." It is one of the oldest biobehavioral techniques known and used. Although widely practiced all over Europe, in Russia, and in Japan, AT is less popular in North America, and may—undeservedly—have lost some of its luster given current advances in and enthusiasm about mindfulness therapies (Davidson et al., 2003), which in many ways have overlapping rationales with AT. The objective of this chapter is to describe AT, its rationale, practice, and outcomes, and also to place AT within the current trends of self-regulation therapies.

The German neurologist Johannes Heinrich Schultz (1884–1970) is credited with the development and promulgation of AT, which is considered a self-hypnotic procedure. During his medical training in dermatology and neurology, Schultz became fascinated with heterohypnosis, which, however, had a dubious image among many of his medical supervisors and peers at that time. Initially, Schultz worked with hypnosis only on his own time, outside of his regular clinic duties. The dominant therapeutic approach then for mental and psychosomatic problems was psychoanalysis, but Schultz rejected analysis as a promising treatment for psychosomatic disturbances. In a brief biography, Schaefgen (1984) cites Schultz as having said that "it is complete nonsense to shoot with psychoanalytic guns after symptom-sparrows."

The breakthrough of AT came after Schultz opened his own medical practice in neurology and psychiatry in Berlin in 1924, where he promulgated AT without the constraints of medical superiors who did not share his vision. His first formal presentation of his experiences with AT was in 1926, in front of his colleagues in the medical society; his first book followed 6 years later (Schultz, 1932). In all, he is accredited with more than 400 publications, numerous books, and translations of these into six languages. His groundbreaking book on AT had seen 18 editions by 1984.

The development of AT has two sources: Schultz's own experiences with clinical hypnosis and Oskar Vogt's observations in brain research. Schultz himself noted that his hypnotized patients regularly reported two distinct sensations—a strange heaviness, especially in the limbs, and a similarly unfamiliar sensation of warmth. He was convinced that hypnosis was not something that the hypnotist actively did to the learner but that individuals did to themselves. For the patient to enter this state, there had to be a "switch," a point of change. Activating this switch—placing the control in the hands of the patient—was what Schultz wanted to achieve. Oskar Vogt's experiences further strengthened Schultz's belief that it was possible to reliably trigger an autogenic state, because Vogt, a brain researcher, had observed that his patients could volitionally produce the sensations of heaviness and warmth and could switch into self-hypnotic trance. Herein lay the seed for autogenic formulas, which represent a set of mental self-instructions to seek out particular physical sensations. Over several years, Schultz further developed the idea of formulas to reliably achieve deep relaxation and its accompanying sensations in various parts of the body. The publication of his 1932 book on AT was the culmination of his efforts to standardize the procedure.

AT remained unknown on the other side of the Atlantic Ocean until one of Schultz's followers, Wolfgang Luthe, a physician, emigrated to Canada and began clinical work, teaching, and research about AT in English. A benchmark paper appeared in the *American Journal of Psychotherapy* (Luthe, 1963), and this was later followed by a hefty six-volume book series that Luthe coauthored with Schultz (Luthe, 1970a, 1970b, 1970c; Luthe & Schultz, 1969a, 1969b; Schultz & Luthe, 1969). These volumes provide extensive descriptions of supporting experimental research, case studies, and clinical success reports of AT for a wide range of clinical problems. For the reader with a strong empiricist bent, however, reading the original works will likely be a frustrating task, because, in the ultimate evaluation of AT's effectiveness, no distinction is made by Schultz and Luthe among opinions, single-case reports, and controlled studies (of which there were precious few at the time). For a more detailed description of the background research and applications, I refer the reader to my book *Autogenic Training: A Clinical Guide* (Linden, 1990), and for diligent reports on outcome the reader may want to peruse Stetter and Kupper's (2002) work, Grossman and colleagues' (Grossmann, Niemann, Schmidt, & Walach, 2004) excellent meta-analysis, or a detailed review paper that combines a narrative with a meta-analytic review approach (Linden, 1994).

THEORETICAL UNDERPINNINGS

Given the apparent similarities among meditation, hypnosis, biofeedback, muscular relaxation training, and AT (Benson, 1975), it requires a fine-grained analysis to reveal differences in underlying rationale, technique, and—possibly—outcome. Among the many descriptors used for AT is "a psychophysiological self-control therapy" (Pikoff, 1984, p. 620). The emphasis is on "self-control," "which the patient carries out." This also explains why AT manuals do not come with a CD that the patient can (or should) take home. In contrast, the popular progressive muscular relaxation (PMR) approach, as described by Bernstein and Borkovec (1973), combines the written manual with a record to facilitate relaxation practice, although this method also cannot be effectively taught by audio recordings alone (Lehrer, 1982).

The term *autogenic* is derived from the Greek words *autos* and *genos* and can aptly be translated as "self-exercise" or "self-induction therapy." It is, furthermore, important to present in detail how, in AT, a conceptually sensible, physiological rationale and self-hypnotic suggestions are woven into a type of intervention linking "mind" and "body."

Schultz was a firm believer in the self-regulatory capacities, if only the body was left alone to do its work. Homeostatic models (Cannon, 1933) and more recent formulations of biological self-regulation theory (Linden, 1988) were foreshadowed by Schultz when he conceptualized AT (Schultz, 1932). Although the most typical application of AT is to reduce excessive autonomic arousal (i.e., it serves as a relaxation technique), the AT rationale embraces a bidirectional homeostatic model, suggesting that AT should be equally useful in also raising dysfunctionally low levels of an autonomic function (e.g., low heart rate variability).

The objective of AT is to permit self-regulation in either direction (i.e., deep relaxation or augmentation of a physiological activity) through "passive concentration," also described as "self-hypnosis." The trainee concentrates on his or her body sensations in a passive manner, without trying to directly or volitionally bring about change. "Passive concentration" may sound paradoxical, in that "concentration" usually suggests effort. What it means in AT is that the trainee is instructed to concentrate on particular inner sensations, rather than environmental stimuli, and just to observe them, not necessarily try to produce them. This is indeed somewhat effortful, especially for the novice. When this concentration does not come easily, the trainee is told to let thoughts wander for a while, or to rearrange the body position for more comfort, rather than to force inner concentration. Not forcing, allowing sensations to happen, and being an observer rather than a manipulator are what "passive" refers to. The AT trainee is warned that trying too hard is counterproductive: It may lead to negative reactions such as muscle spasms, and it stands in the way of acquiring the necessary self-regulatory attitude.

The principle of passive concentration clearly differentiates AT from Jacobson's (1938) progressive muscle relaxation (see McGuigan & Lehrer, Chapter 7, this volume) and biofeedback (Schwartz & Andrasik, 2003; see also Gevirtz, Chapter 6, Lehrer, Chapter 10, and Meuret & Ritz, Chapter 11, this volume), in which patients actively attempt to acquire control over physiological functions. A feature that AT shares with biofeedback, however, is the assumption that bidirectional change (increase or decrease of a physiological activity) is possible and, in some instances, desirable as well. Although AT is considered self-hypnotic, the differences between self-hypnosis and heterohypnosis need to be stressed. In heterohypnosis, the hypnotic trance is induced by another individual (i.e., the hypnotist), who will typically make relaxation and trance suggestions, followed by suggestions for behavioral changes such as stopping smoking or feeling release from pain (see Karlin, Chapter 19, this volume). The key differences are self- versus other-control and dependence versus independence from a therapist. AT is designed to strengthen independence and to give control back to the learner, thus eliminating the need for either physiological feedback devices (as in biofeedback) or a hypnotherapist.

The claimed uniqueness of AT is supported by (1) experimental studies showing that biobehavioral methods have differential effects on a variety of clinical problems (a summary is provided later in this chapter) and (2) basic experimental findings that relaxation and hypnosis can be psychophysiologically distinguished from autogenic states. Diehl, Meyer, Ulrich, and Meinig (1989) investigated regional cerebral blood flow in 12 healthy male volunteers during autogenic training and during hypnosis. Hypnotic states were verified via successfully performed arm levitation and persistent catalepsy of the right arm. These researchers observed that global hemispheric blood perfusion increased significantly, relative to the participants' own baseline resting values. Perfusion during AT was significantly less than during hypnosis.

Shapiro and Lehrer (1980) contrasted psychophysiological effects in participants who had learned either PMR or AT in a 5-week training program. All active training reduced anxiety, depression, and reports of physical symptomatology, but only AT triggered self-perceived heaviness and warmth, as well as changes in depth of breathing. Similarly, in a contrast of effects for AT versus PMR for anxiety reduction, subjective reports revealed reduced anxiety for both active treatments, but AT showed some additional advantages via heart rate reductions not seen in progressive relaxation (Lehrer, Atthowe, & Weber, 1980). Unfortunately, published studies that suggest effects specific to AT, but not to other methods, are rare. The three studies described here support the potential of distinct physiological and subjective effects for various self-regulation methods, without, however, offering conclusive evidence.

The core ingredients of AT that make it distinct as a method are six standard formulas referring to specific body sensations. These formulas are subvocally repeated by the patient; in addition, the patient is encouraged to develop vivid, personally meaningful images to accompany and enhance these formulas. An important feature that also distinguishes AT from PMR (Jacobson, 1938; McGuigan & Lehrer, Chapter 7, this volume) and meditation (Wallace, 1970; see also Carrington & Lehrer, Chapter 13, Kristeller, Chapter 14, Chen, Chapter 15, and Telles, Kala, Gupta, & Balkrishna, Chapter 16, this volume) is the inherent claim of specific effects for each formula. Each formula targets a specific bodily function, and the sensations and images suggested by the formulas are derived from patient reports of deep relaxation and trance states rather than being theoretically derived. The formulas suggest sensations that a relaxed trainee is likely to experience anyway and create positive expectations of distinct somatic experiences; their occurrence then reinforces the effort and lends further credibility to the formulas. The "magic" of hypnosis (whether induced by a hypnotherapist or self-induced, as in AT) thereby derives from a focus on and increasing awareness of real somatic sensations.

There is growing evidence that AT may not only affect sympathetic tone but may also achieve some of its benefits through its impact on parasympathetic activation. To examine the hypothesis that the AT response is associated with an increase in cardiac parasympathetic tone, the frequency components of heart rate variability (HRV) during relaxation training were investigated in 16 college students (Sakakibara, Takeuchi, & Hayano, 1994). Electrocardiograms and pneumograms were recorded during a 5-minute baseline period, followed by three successive 5-minute sessions of the AT (relaxation) or by the same periods of quiet rest (control), while participants breathed synchronously with a visual pacemaker at an average adult baseline respiration rate (0.25 Hz). Although neither the magnitude nor the frequency of respiration showed a significant difference between relaxation and control, the amplitude of the high-frequency (HF) component of HRV increased only during relaxation ($p = .008$). There was no significant difference in the ratio of the low-frequency (LF; 0.04–0.15 Hz) to the HF amplitudes. The increased HF amplitude without changes in the respiratory parameters indicates enhanced cardiac parasympathetic tone. Thus our results support the initial hypothesis of this study. Enhanced cardiac parasympathetic tone may explain an important mechanism underlying the beneficial effect of the relaxation response.

Similarly, physically healthy individuals with high trait anxiety (TA) were studied for changes in HRV during two opposite psychophysiological conditions of mental stress and relaxation induced by autogenic training. The main finding was that high anxiety was associated with reduced R–R spike intervals and HF power across conditions. In comparison with mental stress, autogenic training increased HRV and facilitated the vagal control of the heart. There were no significant effects of TA or the psychophysiological conditions on LF power, or LF:HF ratio (Miu, Heilman, & Miclea, 2009).

The assumption of AT that somatic imagery can trigger underlying physiological activity is also consistent with Lang's (1979) theory of emotional imagery coding and experience-based somatic–visceral responding. In a series of studies (Lang, Kozak, Miller, Levin, & McLean, 1980; Lang, Levin, Miller, & Kozak, 1983), Lang and his collaborators showed experimentally that focusing imagination on a distinct physiological response (e.g., sweating or heart rate) did indeed provoke the imagined visceral response with reasonable specificity.

The heaviness formula in AT is directed at muscular relaxation and has been found to be associated with reduction in muscle tone, reductions in blood pressure, and increases in skin resistance (Fischel & Mueller, 1962; Ohno, 1965; Schultz, 1973; von Siebenthal, 1952; Wallnoefer, 1972). The warmth formula is directed at vascular dilation, and researchers have observed peripheral vasodilation in hands and face with an accompanying increase in skin temperature and reduced alpha sympathetic activity, as well as occasional light sweating (Dobeta, Sugana, & Ohno, 1966; Pelliccioni & Liebner, 1980; Polzien, 1953; Schwarz & Langen, 1966). Practice of the heart regulation formula has been associated with reduction in heart rate, reduced cardiac output with simultaneously improved CO_2 utilization, and stabilization of labile electrocardiogram signals (Luthe, 1970a; Polzien, 1953). Participants practicing the breathing regulation formula displayed reduced breathing rates and volume and showed shifts from predominantly thoracic to more abdominal breathing patterns (Ikemi et al., 1965; Luthe, 1970a; Polzien, 1953). Practice of the "sun rays" formula is supposed to regulate visceral organ activity, and researchers have indeed reported normalization of dysfunctional stomach and intestinal function. Increased blood flow to the gastric mucous and vasodilation of peripheral blood vessels have also been noted (Ikemi et al., 1965; Sapir & Reverchon, 1965; Lantzsch & Drunkenmoelle, 1975). Finally, the "cool forehead" formula, which is meant to regulate brain activity and forehead blood flow, has been associated with reduced frequencies of beta waves and increased frequencies of alpha and theta waves in the electroencephalogram (Israel & Rohmer, 1958; Jus & Jus, 1968; Katzenstein, Kriegel, & Gaefke, 1974). Dierks, Maurer, and Zacher (1989) also reported increased theta-wave and reduced beta-wave activity; alpha-wave activity, however, increased slightly with AT practice. Furthermore, Dierks et al. (1989) noted that the reduction in beta-wave activity was specific to the right hemisphere, which is commonly presumed to be the site of emotional function.

A phenomenon not described in the literature for other self-regulation techniques is that of "autogenic discharges," which are seen as a sudden and unpredictable form of "unloading" of pent-up thoughts, sensory processes, and muscular activity (Luthe, 1970b). Although AT is presumed to have an overall gentle, slow effect on autonomic self-regulation, the concept of autogenic discharges incorporates the idea that some of the self-regulation may occur through short bursts of central nervous system activity. Luthe (1970b) differentiates (1) reactive discharges (i.e., responses to acute provocation); (2) normally occurring spontaneous discharges (e.g., motor discharges during presleep stages); (3) discharges that originate from the brain and characterize forms of pathology (e.g., epilepsy); and (4) discharges that may occur during sensory deprivation and during the practice of AT.

Luthe (1970b) also reported that some autogenic discharges are experienced as pain memories from previous injuries, illnesses, or operations. Similarly, there have been reports that the quality of the autogenic discharge may be related to the particular formula being practiced at the time. This can take on the form of a discharge sensation experienced in the body part that is currently being concentrated on and may be functionally related (although typically in the opposite direction) to the target sensation (e.g., heart palpitations during the heart regulation formula).

Unfortunately, the discharge phenomenon is experienced with considerable variation in intensity and can take on many different forms. In consequence, one can debate whether trainees who do not report sudden discharges have them nevertheless but at a subliminal level (Luthe's position; 1970b), or whether they do not occur at all. Also, given that the discharges may take on different forms, it cannot be ruled out that the label "autogenic discharge" may simply cover a variety of phenomena with heterogeneous underlying neuro- or psychophysiological origins. One thing, however, is clear: Autogenic discharges, when noticed by trainees, are usually interpreted as bothersome and unwanted side effects of the procedure. The traditional view in the AT literature, however, is that autogenic discharges are necessary in a "hydraulic" sense and are considered signs of progress, because they suggest a reduction in physiological and psychological inhibition and provide an opportunity for release of excessive pressure in the system. It is important for the AT instructor to interpret discharge experiences for confused trainees and to provide sensible, comforting explanations. Such practicalities aside, a group of French and Italian psychoanalysts have discussed how autogenic discharge can be used systematically to foster free association (Gonzalez de Rivera, 1977; Ranty, 2007).

Data collected by Luthe (1970b) on two experimental groups may further serve to explain the phenomenon and illustrate the variety of possible autogenic discharge experiences. The two groups of participants were all AT trainees classified either as openly sexually active or as sexually deprived because of their particular life situations (i.e., they were members of the clergy or were otherwise prohibited by their religion from being sexually active). The two groups were similar in male–female proportion, age, clinical condition, and level of professional achievement. The experimental prediction was that the sexually deprived individuals would display more sexuality-related autogenic discharges. Luthe's (1970b) observations suggested that the sexually deprived group indeed had more sexuality-related and general discharge symptoms than did the controls. The sexually deprived group reported more itching, tingling, pain, and muscular twitches; they also reported more erections and vaginal spasms, as well as more sexual fantasies. The perceived sites of the most frequent sensory and motor discharges were the thighs, lower abdomen, and genital regions.

Autogenic discharges are similar in some ways to phenomena described in connection with other techniques (e.g., "relaxation-induced anxiety," as described in the PMR literature; see McGuigan & Lehrer, Chapter 7, this volume; the "side effects of tension release" described by Carrington & Lehrer, Chapter 13, this volume). The AT literature, however, presents a more detailed picture of these phenomena than literature on other techniques and gives more specific suggestions for how to manage them when they do occur.

ASSESSMENT

Studies of therapy effectiveness typically provide statistical demonstrations of between-group means (based on comparisons of treated patients with themselves before training or with wait-list or other treatment controls) as "proof of a positive outcome" (Linden & Wen, 1990). Hidden in such mean change comparisons, however, is considerable variability in treatment response: Some patients benefit, whereas others do not change or get even worse (Jacobson, Follette, & Revenstorf, 1984). A particularly striking demonstration of treatment effect variability is provided by Aivazyan, Zaitsev, and Yurenev (1988), who randomly assigned patients with hypertension to either AT or a no-treatment control condition. When mean changes were broken down into "percentage improved" ratings, the following figures emerged: In the AT-treated group, 32% improved, 59% remained unchanged, and 9% deteriorated; in the control group, 59% also remained unchanged, 11% improved, and 30% deteriorated. Clearly, therapy did little for the majority of patients, whereas the between-group difference is effectively attributable to treatment effects consisting of both direct improvement and the prevention of worsening. Thus valuable health care funds may be better invested if patients who are not going to benefit from treatment can be identified a priori and left out of the treatment comparison.

Especially in light of these observations on treatment outcome variability, a clinician teaching individual clients cannot be satisfied with knowing that a statistically significant mean change of a treated group occurred; instead, the practitioner needs to attend to each individual's progress. Therefore, it is of great importance for practitioners using AT to be aware of what kind of client can learn and benefit from AT and to know in advance whether AT is indeed the best method of treatment for a given client. The question of AT's suitability, given certain individual characteristics, is addressed in this section.

The literature and my own experience indicate clearly that the mechanics of AT can be taught to a wide variety of individuals; nonetheless, caveats are in order. Adults of all ages and many children have learned AT, but children below school age lack the discipline to master AT. Depending on a child's maturity, intelligence, and imaginative abilities, the youngest age at which AT can be taught effectively is between 6 and 10 years. Individuals with developmental disabilities, those with acute central nervous system disorders, and those with uncontrolled psychoses are also likely to be unable to process and follow the instructions. Thus, with these relatively few exceptions, AT can be taught effectively to a wide range of populations, not only as a treatment but also as a preventive skill.

Although few individuals are unable to learn the mechanical aspects of autogenics, this does not mean that every learner will necessarily show clinical benefit, and the practitioner has to consider the possibility that AT is not the treatment of choice for a given person. Three lines of research contribute valuable information in this respect. The first is research on relaxation-induced anxiety (Heide & Borkovec, 1984). A second pertinent area of research has attempted to predict relaxation training success by considering differences in initial resting levels (Jacob, Chesney, Williams, Ding, & Shapiro, 1991) and interindividual differences in response to the first training sessions (Vinck, Arickx, & Hongenaert, 1987). Finally, personality factors as predictors of success have been specifically targeted (Badura, 1977).

A number of potential explanations exist for the paradoxical effect of anxiety increase during relaxation (Heide & Borkovec, 1983). The first explanation is that during relaxation a shift toward greater parasympathetic dominance occurs, which results in peripheral vasodilation and feelings of warmth and heaviness (the first and second formulas in AT; Budzynski, Stoyva, & Pfeffer, 1980). Unfamiliarity with parasympathetic activity sensations may make these feelings particularly disturbing to individuals with chronic tension or anxiety. Also, relaxation frequently brings about unfamiliar spontaneous muscular-skeletal events such as myoclonic jerks, spasms, twitches, or restlessness ("autogenic discharges" in AT). Another explanation centers around the notion of fear of loss of control. Individuals with chronic anxiety may have learned to control their anxieties in the past by never letting go; they typically work in a compulsive, rigid manner and cannot permit themselves to relax (Martin, 1951). Finally, Ley (1985), on the basis of his work with panic disorder, has proposed that relaxation-induced anxiety may be linked to "relative hyperventilation" ("relative" in this context means that the perceived pace of one's own breathing is above what would be metabolically needed in a given situation). The discrepancy between perceived need and actual respiration pace serves as an alarm cue triggering additional anxiety cognitions, which may then create an upward spiral toward even more arousal.

These findings suggest that patient characteristics such as an anxiety experience can predict differential relaxation treatment outcome, and they deserve consideration in individual treatment plans involving relaxation therapy. Unfortunately, the replicated findings in this research domain involve only meditation, exercise, and PMR, and it is not clear how AT outcome may be affected by individual predispositions such as pretreatment anxiety levels.

The second and third lines of research deal with pretreatment or early-treatment differences between individuals. Vinck et al. (1987) attempted to predict blood pressure treatment responses in normotensives who learned either PMR (Jacobson, 1938) or AT. Training was provided weekly for 6 weeks. Relaxation effects were measured as within-session changes during the first treatment session, overall changes in resting values from the first to the last treatment session, and within-session changes during the last treatment session. Although no differential effects for PMR relative to AT were reported, Vinck et al. (1987) did replicate Jacob, Kraemer, and Agras's (1977) findings that higher initial blood pressure levels also predicted the greatest reduction after relaxation training. A recent review and a controlled trial of therapy outcome for hypertension treated via relaxation strategies (Jacob et al., 1991; Linden, Lenz, & Con, 2001) clearly confirm an earlier contention that patients with initially high blood pressure show greater reductions. Vinck et al. (1987) also found that trainees with the smallest changes within the first training session of either AT or PMR were the ones who showed the greatest reductions during the last training sessions. Attempts to predict blood pressure treatment response via personality indices was unsuccessful. Vinck el al. (1987) may have failed to identify personality factors as predictors of AT success because their participants were healthy individuals who probably reflected a relatively narrow range of associated personality features. No such range restriction was apparent in the work of Badura (1977), who related Minnesota Multiphasic Personality Inventory (MMPI) profiles to AT outcome in 200 patients who displayed neurotic, functional, and/or psychosomatic symptomatologies. Badura's patients were subdivided into "successes" and "failures" on the basis of their reported ability to achieve formula-specific autogenic sensations. Patients in the "failure" group were characterized by relative elevations on the Hypochondriasis, Depression, Hysteria, and Social Introversion subscales. Discriminant function analysis indicated that, with these distinct MMPI profiles, 80% of the success–failure incidences in AT could be correctly classified.

A number of conclusions and suggestions appear justified. Patients with elevated baselines on an autonomic index (e.g., blood pressure) profit more from AT (or other relaxation therapies) than those with lower baselines. Similarly, those patients showing the least initial response to treatment improve relatively more over time. Also, clinical elevations on the MMPI scales noted above predict lack of success with AT. Such individuals may be better served with another form of psychotherapy.

METHOD

The Training Format

AT can be taught individually or in groups. The advantages for each mode of training are fairly obvious and are the same as for other forms of psychological therapy. Individual training is much more expensive, but training can also be easily adjusted to likely differences in the pace of learning and other individual needs (this is especially true when AT is taught as part of a complex intervention package). The existence of a personal therapeutic relationship may also serve to enhance compliance and credibility. Group training is more cost-effective but permits less individualized attention and may thus reduce compliance. On the other hand, groups also have the potential to develop cohesion and serve as mutual support systems, which in turn will have a positive impact on compliance. My personal preference is to teach AT in groups of 8–12 participants, as long as the group can be expected to have more or less homogeneous needs and learning paces.

Another important point that cuts across the learning process is that of realistic expectations. At the outset of AT, trainees should be alerted to the probability that learning will be slow. The great majority of practitioners feel little, if anything, during their first practices, and it is perfectly normal for the desired sensations to remain weak for the first weeks of practice. This is true even for the avid practitioner who is fully compliant with the instruction to practice twice per day.

The Physical Setting

The ideal physical setting is one of comfort, with minimal likelihood of disruption, a room temperature of 20–24°C (68–75°F), a couch or exercise mattress (plus pillows) to stretch out on, and adjustable lighting conditions (a slightly darkened room is best). Training success is facilitated by an environment that permits trainees to concentrate on their inner sensations. Accordingly, any speech while training impedes with the basic principle of autogenics. If the trainer talks during the exercise or plays a recording, the trainee cannot really learn to exercise autogenically (i.e., independently); instead, he or she will go through a light heterohypnosis. Therefore, autogenic training necessitates tranquility. In a tranquil setting, AT, with its focus on six functional systems (muscles, blood vessels, heart, breathing, inner organs, and the head), can be learned best.

In order to go through the training procedure, a very comfortable sitting—or, even better, lying—position is necessary. The entire body position must be comfortable, because body position itself may lead to muscle tension, which will interfere with progress in the exercises. It is most advantageous to exercise in a supine position, so that the neck especially is well supported. The arms should be placed flat beside the body with slightly bent elbows, and the interior of the hands should be placed on the surface of the couch or mattress. The tips of the feet should fall slightly to the outside.

If lying down is not possible (e.g., if a trainee wants to practice in the clinician's office), a chair with a high back and armrests is best, so that the head and arms are supported. The elbows should be bent at nearly a right angle, because this ensures that the stretching and bending muscles in each arm are in a balanced state. The entire back and the back of the head should be fully supported. Small pillows may facilitate this support. The feet should rest flat on the ground and close to each other, and the knees should fall slightly to the outside, which will help to prevent mechanical tension in the thigh musculature. Most people will tend to close their knees even while sitting, although this position is often associated with unconscious muscular tension.

When it is not possible either to sit comfortably or to lie down, a third position may be used for the exercises: A trainee can sit on a bench or a chair without back support. In this position, the head should be allowed to sink forward to the chest, so that the arms will hang at the sides and the head will be in a perfectly vertical position over the spine. It is important for the trainee not to bend forward; instead, the torso must be in a vertical position, although somewhat reduced in height. In this position, no muscular activity is necessary and no muscular tension is created, because the skeleton is held by the spine and its tendons. Now the arms can be moved loosely and can be supported on the widely spread thighs, so that the underarms (close to the elbow) will be supported by the thighs. The arms are again bent in the above-described manner. The body now hangs without any muscular work in its own bone structure.

These positions need to be assumed carefully before the exercises begin. In one of these positions (preferably lying down), the trainee can now begin with the first exercise. The eyes should be closed to facilitate passive concentration, and the trainee should now try to imagine the sensation in the formula as well as possible, without making any movement or trying to speak or do anything else. The ideas, images, and memories that will necessarily develop in each individual should not be fought off, because this attempt in itself would lead to tension. Ideas and images other than the formula-based sensations should be ignored.

Content and Sequence of Exercises

First Exercise: The Heaviness Experience (Muscular Relaxation)

The first AT exercise involves the musculature, because muscle activity is familiar to people and is most easily influenced by conscious efforts; in addition, experience with hypnosis and relaxation suggestions has shown that notable muscular relaxation can be achieved rapidly. Muscular relaxation is experienced as a heaviness of the extremities. Intentional concentration on outside stimulation is associated with muscular tension (e.g., looking, speaking, and reaching out are based on muscular movement). Attentional anticipation can also justifiably be called "tension," because muscles are already tensed in anticipation of movement. Even profound thinking may be associated with muscular activity, as many individuals crease the forehead while thinking. Each intention, or even vivid imagination, of a motion will result in increased tone of the musculature in the extremities.

It is not advisable to use the entire body as an object of training at once, because in this case the necessary focus would be difficult to achieve. The training should begin with the dominant arm. If this arm has been trained for a reasonable period of time, the experience of heaviness during muscle relaxation will generalize to the other arm, the legs, and other body systems, because all extremities and organs are controlled by the same nervous system. The exercise is executed on the arm until it has generalized to the other three extremities. It is important to achieve a maximal concentration in the one arm first and to permit a generalized overflow of relaxation into the other extremities before good results can be expected.

The steps in the heaviness formula are as follows: (1) "The right (left) arm is very heavy" (this is repeated six times); (2) "I am very quiet" (this is said only once, and then alternates with the first step until six cycles have been completed). In typical individuals, a noticeable experience of heaviness will develop soon, particularly in the area of the elbow and lower arm. After the heaviness formula is practiced, the instructions are "taken back." *Taking back* refers to a systematic set of activities designed to bring the trainee gradually from a state of relaxed, low muscle tone back to an alert state. This needs to be performed in a consistent manner to facilitate the reflex nature of the process. It is executed in the following steps: (1) The arm is bent and stretched a few times with an energetic pull; (2) the individual breathes profoundly in and out briefly; (3) the eyes are opened. As brief versions, one can use the following: (1) "Bend arm," (2) "Breathe deeply," (3) "Open eyes."

It is important that the trainee pay attention to the timing of the exercise. Training should be repeated in two or three practice sessions per day. In each training session, one can practice the heaviness formula twice for about 1 minute each. Because many trainees want to do the exercise particularly well, the individual steps may be extended in the beginning, and semiconscious tensions may arise. Trainees will realize that the experience of heaviness, instead of increasing, decreases more and more with excessively long practices.

Within the first week of training, the feeling of heaviness in the trained arm will be more pronounced and will occur more rapidly; also, the same feeling will be experienced in the other extremities, usually at the same time as in the other arm. When the experience of heaviness in both arms is quite pronounced, the formula can now be changed into "Arms are heavy." The taking-back procedure for both arms involves a count from 1 to 4, in which each number is associated with a specific instruction: (1) "Make a couple of fists," (2) "Bend the arms a few times," (3) "Breathe in deeply," and (4) "Open the eyes and sit up." Heaviness experienced in the legs does not necessitate a particular taking-back procedure, as legs function more autonomically. Normally, within a week, the exercise has proceeded so far that with only a brief moment of inner concentration arms and legs can be perceived as quite heavy. It is then time to approach the second exercise.

Second Exercise: Experience of Warmth (Vascular Dilation)

Muscular exercises are something that the naive individual finds natural, as muscular activity is typically considered to be a voluntary act. It is a more novel idea that blood vessels may constrict or dilate through intentional effort. However, it should be noted that all emotional activity tends to be associated with a change in blood flow (flushing or paleness). Furthermore, there are systematic types of activities (e.g., the sauna) in which individuals systematically train blood vessels; these activities are reasonably familiar to many individuals. The second AT exercise, which aims at the warmth experience, affects the entire peripheral cardiovascular system: It affects blood flow through arteries, capillaries, and veins in the skin, organs, and musculature. The distribution of blood in the vessels is regulated through constriction and dilation, which take place as a response to nervous system innervation; their magnitude and direction are determined by physical activity, general state of arousal, and inhibition.

Once the first exercise with the heaviness experience has been well trained and can be induced rapidly and reliably, training sessions can then be extended by inclusion of the second formula, as follows:

1. "Arms (legs) are very heavy" (this is repeated for a total of six times).
2. "I am very quiet" (this is said once).
3. "The right (left) arm is very warm" (this is repeated six times; the word *quiet* is then repeated once).

A healthy individual will notice an inner, streaming, flowing sensation of warmth very rapidly, typically in the area of the elbow and the lower arm. Quite frequently, trainees who master the heaviness sensation will also spontaneously report warmth sensations before they are instructed to imagine them. Specific instructions for taking back the experience of warmth are not necessary, as the blood vessels are elastic and governed by a compensatory self-regulation, which will trigger a return to their usual position in an autonomous manner.

The first and second training exercises are executed in the same manner for a period of at least 1 week, until warmth is experienced easily and rapidly in the trained arm first and then in all four extremities. The experience of heaviness and warmth will then also generalize to the entire body. The blood vessel dilation and associated relaxation have a particularly tranquilizing and sleep-inducing effect. Training exercises directed at blood vessel dilation are not necessarily innocuous, because the changed distribution of blood influences the entire organism. The exercise should be instituted only in healthy individuals for whom no vascular risks are known to exist.

When a new exercise step is added in AT (e.g., when the experience of warmth is added to the feeling of heaviness, as above), the individual should always concentrate initially on the already learned exercises and should add a new exercise only for brief periods (typically 1 minute). New exercises are added only for brief periods in order to keep the overall exercise length brief and to prevent trainees from attempting to achieve "perfect success" (i.e., taking it too seriously). The choice of 1-minute segments is somewhat arbitrary; it is suggested because 1 minute is an even unit of time, and because when all training steps are added together they amount to a reasonable practice length of 10–15 minutes. Once heaviness and warmth are achieved rapidly and reliably, the third exercise can be added.

Third Exercise: Regulation of the Heart

The awareness of heart activity varies considerably among people. How does one feel heart activity? Many individuals are aware of it in times of strain, excitement, and fever, but many others do not feel heart activity without prior training. These trainees need to be sensitized to their own heart activity.

Trainees who do not perceive their heart activity at any particular point in their bodies can use their pulses for orientation. With further training, they will also experience the activity of the heart itself. If this help is not sufficient, a trainee may try to become aware of heart activity by other means. This can be done by lying flat on the back so that the right elbow is fully supported and lies at the same height as the chest. Now the right hand is placed in the heart area; the left arm's position remains unchanged. Now the trainee can go into the usual state of heaviness, warmth, and quietness and can concentrate on the sensations in the chest area just where the hand touches the skin. The pressure of the hand functions as a directional indicator. After a few exercises, the trainee is now likely to recognize heart activity, and with continuing repetition of the entire exercise the experience will become more obvious. The heart formula is "The heart is beating quietly and strongly" (or, in the case of easily arousable individuals, "quietly and regularly"); this formula is repeated six times, and the word "quiet" is added once.

When the heart sensation has been learned (and, in a sense, been "discovered"), the hand does not need to be placed any longer in the area of the heart, but the exercise can be continued in the usual position. It should be strongly emphasized that the intent of the exercise is not actively to slow down the heartbeat, as this would prevent self-regulation. The emphasis of this exercise is on regular and strong beats, but not on a reduction of the heartbeat frequency.

Fourth Exercise: Regulation of Breathing

Breathing is partially intentional and partially an autonomous activity. In AT, the muscular, vascular, and heart relaxation become immediately integrated with the rhythm of breathing, much as heaviness and warmth automatically generalize from the trained arm to all the other extremities. In the AT procedure, however, any intentional influence on or modification of breathing is undesired, as an intentional change would be associated, through a reflex-type mechanism, with tension and voluntary activity. Again, the trainee is to enter all the other exercise levels before the new, fourth formula is added: "It breathes me" is repeated six times, and then the word "quiet" is added.

For many individuals, it is very seductive to attempt voluntary changes of breathing, as in a systematic breathing exercise (e.g., in yoga). This intentional modification needs to be prevented in AT, because breathing is supposed to function autonomously and in a self-regulatory system without any active adjustment. In order to prevent intentional change, the passive wording "It breathes me" has been chosen. This statement is intended to make it clear to the trainee that relaxation and the regulation of breathing will come by themselves—that the trainee will be carried by and is to give in to his or her natural breathing rhythm. It typically takes another week to make good progress with this exercise.

Fifth Exercise: Regulation of Visceral Organs ("Sun Rays")

For self-regulation of visceral organs, the trainee focuses on the area of the solar plexus, which is the most important nerve center for the inner organs. The image associated with this nerve center is that of a sun from which warm rays extend into other body areas. The solar plexus is found halfway between the navel and the lower end of the sternum in the upper half of the body. The trainee now concentrates on the solar plexus area: The formula "Sun rays are streaming quiet and warm" is repeated six times, and "quiet" is repeated once. This exercise also takes approximately 1 week for normal individuals to learn. The image of the breath streaming out of the body when the person breathes out can also help with this particular exercise.

Sixth Exercise: Regulation of the Head

The well-known relaxing effect of a cool cloth on the forehead forms the basis for the sixth exercise. In order to learn the sixth exercise, the individual will engage in the first five exercises in the same careful and progressive manner as described above and will then (initially only for a few seconds) proceed with the following formula: "The forehead is cool" (repeated six times). Just as warmth is associated with vasodilation, the experience of freshness on the forehead leads to a localized vasoconstriction and thereby to a reduced supply of blood, which in turn accounts for the cooling effect. Because all blood vessels of the entire organism are interconnected, a localized vasoconstriction may generalize to other blood vessels. This can be demonstrated by placing a finger in a basin filled with cold water; the entire hand (and at times even the opposite hand as well) is likely to feel cool and look pale. During AT, the concentrative relaxation will originate from the cortex as a central organ, which also possesses the capability of changing the distribution of blood within the body. The "cool forehead" exercise can be learned in about the same time as the other exercises, although up to a third of trainees never acquire a strong response to this formula (Mensen, 1975).

Because most walls and windows are not entirely airtight, there will likely be a slight movement of air in any room. Therefore, the cool forehead may be sensed and described as a cool breeze.

Summary of Exercises

With these six formula-specific exercises, AT has been described in its basic but complete form. The entire exercise sequence can now be summarized as follows:

- "Arms and legs are heavy" six times; "quiet" once.
- "Arms and legs are very warm" six times; "quiet" once.
- "The heart is beating quietly and strongly" six times; "quiet" once.
- "It breathes me" six times; "quiet" once.
- "Sun rays are streaming quiet and warm" six times; "quiet" once.
- "The forehead is cool" six times; "quiet" once.
- Now "taking back": "Make fists," "bend arms," "breathe deeply," "open eyes."

After about 8 weeks of training, most individuals have acquired the complete set of sensations, and the emphasis can be placed on ease in achieving the described sensations reliably and rapidly. Daily training for another 4–6 months will lead to more profound and stronger sensations, and generalization of training to different environments can be targeted. It is important to go through the taking-back procedure after each session (except when the trainee has fallen asleep during AT). Thus the trainee will acquire a readily available mechanism for switching from active tension to deep relaxation, and vice versa.

Monitoring Progress and Maximizing Compliance

Compliance and the monitoring of progress are intricately linked and are therefore discussed jointly in this section. Clearly, a trainee who does not see any progress despite twice-daily practice and weeks of training will quickly lose the motivation to continue. In some ways, this section could also be entitled "Maximizing Motivation," because this is the cornerstone of progress and compliance. Because progress is not immediately obvious, a trainee with high initial motivation is more likely to succeed; the therapist needs to radiate confidence in the effectiveness of AT from the very beginning of training. It is recommended that the therapist give an optimistic but reasonable picture of the success to be expected:

> "I have trained *X* number of people or groups, and there is hardly anybody who has not benefited considerably. Even after *X* number of years, I still practice it myself. Within the first 2 weeks you can expect the first training effects, which will only become stronger and easier to bring about as you keep on practicing."

It is important to reinforce compliance with daily practice, especially until the training effects themselves become apparent and take over as motivation enhancers. Even motivated learners, however, do not perfectly adhere to relaxation homework assignments (Taylor, Agras, Schneider, & Allen, 1983). Taylor and colleagues tested compliance with relaxation practice using a special tape recorder that displayed instructions but also monitored unobtrusively the number of times it was actually used; 71% of clients adhered to the instructions. Hoelscher, Lichstein, and Rosenthal (1986) similarly tested compliance with home practice instructions; they found that self-reported compliance exceeded monitored compliance by 91% and that only 32% of trainees averaged one practice a day. These results leave no doubt that poor compliance is a major problem and needs to be taken seriously. The implication for clinical researchers is that compliance needs to be monitored carefully and that only those participants who comply should be included in statistical analyses of outcome.

On the basis of empirical findings on compliance and my own past experience with AT, I can recommend a number of concrete steps for monitoring progress and enhancing compliance.

Have Trainees Keep a Diary

Trainees should keep a diary in which they record their daily practices and particular success or failure experiences. Of course, trainees may cheat and record a practice that they actually skipped, but this does not happen often in my experience, and in fact the diary serves as a potent reminder to trainees. It is recommended that trainees rate the intensity of their perceived sensations in order to maximize the principle of the self-fulfilling prophecy. When trainees rate each practice after being told that the sensation will get stronger and stronger, they are likely to expect steady improvement, which will become even more obvious when they see the progressive ratings they have made. The diary is, of course, very useful for the review of the previous week's training experiences, which should be undertaken at the beginning of a given therapy session. For maximum convenience and compliance, as well as to facilitate standardization, I actually supply all trainees with a preprinted diary that has a page for every training week. This prevents uneven record keeping and eliminates the excuse of "I could not find an appropriate booklet for a diary."

Emphasize Regular Timing of the Home Practice

Lack of compliance is a profound problem plaguing all behavioral prescriptions and treatments that require specific daily routines. Research on medication use (Haynes, Taylor, & Sackett, 1979) has revealed that having patients take medications at predetermined times of day, coupled with other already existing routines, is an important vehicle for enhancing compliance. In the same vein, I ask my trainees to think about and commit themselves to such practice times in the first training sessions. I would rather deal with their scheduling difficulties before they start practicing than find out a week later that they did not practice at all because they could not find the time. When I say "predetermined" times, I do not mean "6:47 P.M. every day" but "every time after I finish watching the evening news" or "when I am in bed before falling asleep." AT practice must become a routine that requires no thinking or planning; otherwise, it is much too vulnerable to daily mood fluctuations or outside disturbances.

Emphasize the Need for Frequent Practice

AT trainees may find the rule of twice-daily practice for 2 months (or more) overly compulsive; when it is combined with other competition for their time, they may be tempted to cut down on practicing. My recommendation is to be understanding if one or two practices a week are skipped; however, trainees should be urged to stick to the rule. Frequent practicing is more likely to occur if trainees clearly understand the reason for this rule. In the first session, it should be emphasized that relaxation is a skill that requires practice, just as learning to talk or walk is for a small child, or reacquiring good balance is for somebody with a complicated leg fracture and a cast. One can also compare AT practice with throwing a baseball or playing the backhand in tennis; any and all of these are skills that require practice, practice, practice.

Examine Reasons for Dropout

Although AT is popular, patients drop out for a variety of reasons: They move away; there is too much competition for their time; the training effects are too slow in coming; or a variety of other reasons. Even the most experienced therapist will have to face dropout and noncompliance rates of 20–25% in AT. If the dropout rates are noticeably higher than this, the therapist should question his or her own ability to motivate patients. Lack of trainer enthusiasm, poor communication skills, or poor session planning is sometimes the culprit. I have also seen—although rarely—that some groups never develop cohesion, for no apparent reason, or that one or more members are considered so obnoxious that other members stay away.

Highlight Success

Nothing succeeds like success, as the old saying goes. The therapist can use this principle by regularly asking the trainees whether they have tried AT in acute stress situations (e.g., anticipating an exam or facing a confrontation with a superior) and highlighting their success stories. Trainees can be asked regularly whether they have noticed any generalizations of training effects, such as improvement in their ability to fall asleep or to relax after a hard day of work, or reduction in occasional tension headaches. Even if they have not personally experienced such benefits, hearing that somebody else has benefited from AT can serve as an extra motivator.

Also, the trainer should frequently praise the learners not only for apparent positive outcome but also for coming regularly to the training sessions and keeping up with the home practice.

Know Possible Problems and Potential Solutions

Anybody attempting to apply a standardized treatment such as AT will soon find out that clinical reality and full standardization are often incompatible. Trainees lose motivation, have unpredictable and confusing experiences, have medical or psychological problems that may interfere with learning and/or practicing AT, or have obligations that may prevent regular practice. Good general clinical skills are required to complement the training manual and still bring training to a fruitful end. Nevertheless, some problems are well known to experienced teachers of AT and are endemic either to specific exercises of AT or to the practice of relaxation at large. A full discussion is beyond the scope of this chapter, but typical problems that can be anticipated and some suggested solutions are presented in the manual (Linden, 1990).

CLINICAL APPLICATIONS AND CASE EXAMPLE

This section serves as a bridge between the prescriptive, standardized procedure described above and the recurrent need of practitioners to apply, modify, and adjust this procedure to the realities of the clinical situation. The practical approach taken in this section is of greatest value for the clinician who needs to make therapy plans on a case-by-case basis and who may have to make modifications to "classic" AT or create a multicomponent therapy package. Modifications of the AT formulas to suit specific case needs, a case example, and a possible integration of an AT component into a stress management package are described below to illustrate the clinical applications of AT.

Modifications of Formulas to Suit Specific Clinical Needs

Modifications of the standard formulas are typically of three types: (1) Only a few of the formulas are taught (often the heaviness and warmth formulas only); (2) the standard set is taught, but one specific formula is left out or modified; or (3) the standard formulas are taught and an additional, problem-specific formula is created and appended.

Teaching abbreviated AT would be cost-efficient if comparative effectiveness with the long version had been demonstrated empirically. Unfortunately, no such direct comparisons are available, although some abbreviated applications of AT have been found to produce therapeutic benefit (see Linden, 1990). Given the absence of clear comparative evaluations, I argue that teaching abbreviated AT methods (e.g., the heaviness and warmth formulas only) may be inadvisable if full therapeutic benefit is expected. The need for elimination or modification of a certain formula from the standard set often results from an unanticipated difficulty. One possibility is that certain formulas trigger negative associations, images, and memories for a particular trainee. Another possibility is a rationale–application mismatch: For example, a cardiac patient may (at least initially) be hypersensitive to all cardiac sensations, and elimination of the heart regulation formula may be advisable.

Many other formula-specific patient problems are possible. I noted in one case that a trainee experienced searing heat sensations at the words "very warm" in the warmth formula, and a toning down to "pleasantly warm" was judged more appropriate. The "sun rays" formula may be contraindicated for ulcer patients; a non-heat-related image may be preferable in order to set the desired sensation apart from the burning sensation of ulcer pain. Or the formula may be left out altogether in order not to direct even more attention to a potential pain site. Such decisions require clinical, on-the-spot judgment, and excessive standardization and prescription via a manual may be unnecessary. Such a modification of the standard protocol is supported by evidence that symptoms of palpitations were indeed more frequently seen after the heart formula but that the overall effect by the end of training was a greater decrease in HR with AT than with PMR (Lehrer et al., 1980).

A particularly appealing modification for many therapists and their patients is use of a person- or disorder-specific additional formula. Lindemann (1974) has provided a useful catalogue of formulas for specific applications, from which I have selected a subset for demonstration here. There really are no limits for adapting such formulas (also called "intentional formulas") to idiosyncratic preferences in imagery and word choices or descriptions of desirable target behaviors. Characteristics of effective intentional formulas are brevity, a pleasant rhyme or rhythm, a positive choice of words, high relevance to the trainee, and good match to his or her personality. Guidance for creating formulas with these characteristics can be drawn from Erickson and Rossi (1979).

Some of Lindemann's intentional formulas are as follows:

- "First work, then pleasure" to help against procrastination.
- "I am happy, relaxed, and free of hunger" to accompany a weight-reduction program.
- "I sleep deeply, relaxed, and restful" against insomnia.
- "I am calm and relaxed; my cheeks stay cool" against blushing.
- "I am completely relaxed and free; my stomach and bowels are working steadily and smoothly" against gastrointestinal complaints.
- "I am totally quiet and in peace; my joints are moving freely and without discomfort; they feel warm" against arthritis pain.

Case Example

Jane M was referred by her family physician because of elevated blood pressure. This 25-year-old woman had a 10-week-old baby at home and had developed high blood pressure during the pregnancy. Pregnancy-induced hypertension tends to disappear quickly after birth, but this had not happened in her case.

The assessment consisted of a 1-hour interview in which Jane and I attempted to identify major sources of distress in her life. Throughout the interview, the patient's blood pressure was sampled at 2-minute intervals, using a fully automated blood pressure monitor with digital displays (Dinamap Model 850, Critikon Corp., Tampa, FL). I routinely use this procedure with all referrals for stress-related problems, because it may help identify emotion triggers that patients themselves may not be aware of (Linden, 2006). The diagnosis of elevated blood pressure was confirmed, in that the 1-hour average reading was 138/95 mm Hg; these readings also supported her family physician's recommendation that drug treatment was not indicated for blood pressure at this level.

Jane remembered that at the age of 18 she had become aware of the family's positive history for high blood pressure, and she had been preoccupied with her own blood pressure ever since. Although I explained that this was probably not accurate, she claimed an awareness of sudden blood pressure changes and attributed subjective feelings of stress to excessive demands in her job as an administrative assistant. When she became pregnant and developed blood pressure problems, she had quit her job and did not plan to return to work in the near future.

Neither Jane's verbal reports nor my attempts to link these reports with accompanying changes in her blood pressure identified specific stress triggers that could have become the targets for a stress management approach. Instead, I chose to teach her AT, which appeared to hold credibility as an intervention for her.

Over an 8-week period with a total of seven 1-hour sessions, Jane learned the full AT package with six formulas. Using a daily diary system, she charted her practice times and successes, thus also documenting her compliance with the twice-daily home practice requirement. At the end of the seventh session, she was clearly comfortable with the full six-formula AT procedure. She continued to be puzzled that her subjective evaluations of when her blood pressure was high or low were as inaccurate at the end of training as they had been before. Her average blood pressure during the last session was 128/78 mm Hg, indicating a 10-point drop in systolic blood pressure and a 17-point drop in diastolic blood pressure from the readings in the first session. Although this averaging procedure is inferior to 24-hour ambulatory blood pressure monitoring, it is nevertheless better than determinations that are based on two or three readings only (Selenta, Hogan, & Linden, 2000). The 1-hour averaging procedure at least captures the adaptation processes typical for repeated measurement (Selenta et al., 2000). At a 3-month follow-up, Jane reported that her blood pressure was still in the normal range (this was verified by her family physician). She continued to practice AT, although less often than during the acute training phase.

AT in a Multicomponent Treatment Package

In the clinic, patients often present with multiple complaints, and/or the therapist discovers during an individual assessment that a given problem is probably caused or exacerbated by a multiplicity of factors. This, in turn, calls for a program of therapy with multiple components. Although multicomponent therapy is the norm in everyday clinical work and is associated with better clinical outcome than single-component therapies (Shapiro & Shapiro, 1982), infinite numbers of such treatment combinations are possible; this makes extensive comparative outcome testing for each combination extraordinarily difficult. Clinical judgment, good training, experience, and an awareness of research findings are needed to judge the appropriateness of a treatment package for a given patient. The best packages tend to be those with strong individually tailored rationales and with components that have been shown to be efficacious when tested alone. Because there are so many possible combinations of treatment techniques, only a stress management combination including AT is described here.

A multicomponent package including AT has become the standard stress management approach in my own clinical work. First, the client is provided with a rationale that describes stress as a three-step process, involving (1) environmental stress triggers, (2) behavioral and cognitive responses to the challenge, and (3) the ultimately ensuing physiological stress response. For each of the three elements of the stress process, different intervention techniques are taught: (1) situational analysis for identification of stress triggers and use of stimulus control procedures to prevent these from triggering stress, (2) modification of the acute response to challenge via cognitive restructuring and assertiveness skill training, and (3) acquisition of a behavioral coping skill for reducing the physiological and subjective arousal via AT. Learning to relax via AT not only has desirable acute effects but also tends to generalize insofar as patients typically learn to perceive themselves as being in control of their stress responses; this, in turn, has a positive impact on the way they perceive potential stress triggers and how they respond to them.

REFLECTIONS ON THE CLINICAL OUTCOME OF AT

Early clinical reports of AT are heavily dominated by case studies and uncontrolled research (Luthe, 1970a; Pikoff, 1984). If taken at face value, these clinical findings suggest that AT possesses treatment potential for almost every psychological and psychosomatic problem ever listed in a medical catalogue. Pikoff (1984) reviewed the available clinical studies published in English and found that the quality of published research was uneven, with few properly controlled trials. Also, because most researchers used time-limited training programs and rarely trained participants in more than the heaviness and warmth formulas, he concluded that AT had never really been tested in this body of literature. Nevertheless, the overall evaluation of AT was quite promising; positive outcomes were reported for AT and insomnia, test anxiety, and migraine.

There have been two comprehensive reviews on AT outcomes, one a combination of narrative and meta-analytic review of outcomes (Linden, 1994) and the other a meta-analysis (Stetter & Kupper, 2002). No similarly comprehensive reviews have appeared since then. Both reviews used similar search and inclusion criteria. Results are reported here as effect sizes (*d*), which can be defined two ways. They can refer to raw means at posttest and pretest, respectively, when change within a group is determined. They can also reflect change scores or means obtained at posttest (but adjusted for pretest differences) for comparisons between treatment groups (e.g., AT vs. attention control groups). Quantitative, meta-analytic findings indicated that AT was associated with medium-sized pre- to posttreatment effects ranging from $d = -0.43$ for biological indices of change to $d = -0.58$ for psychological indices in the Linden (1994) review, and $d = -0.68$ (biological indices) and $d = -0.75$ (psychological outcomes) in the Stetter and Kupper review (2002). The pooled effect size estimates hide considerable variability in behavioral/psychological effects for individual target problems; moderately sized improvements were reported for tension headache and migraine, hypertension, coronary heart disease rehabilitation, asthma, somatoform pain disorder, Raynaud's disease, and anxiety and sleep disorders.

To place these observed outcomes of AT in context, one can compare the effect of AT with those reported for other psychophysiological arousal reduction strategies and other psychotherapies. Data from six meta-analyses permit aggregation and comparison of results (Godfrey, Bonds, Kraus, Wiener, & Toth, 1990; Grossman, Niemann, Schmidt, & Walach, 2004; Hyman, Feldman, Harris, Levin, & Malloy, 1989; Linden, 1994; Luebbert, Dahme, Hasenbring, 2001; Stetter & Kupper, 2002). Effect sizes, when averaged for all types of arousal reduction strategies and classes of endpoints, were:

- $d = -0.56$ for pre–post comparisons
- $d = -0.58$ for arousal reduction versus no treatment
- $d = -0.52$ for arousal reduction versus attention placebo
- $d = -0.15$ for arousal reduction versus other active psychotherapies

Interestingly, although effect sizes for self-reported distress weakened with increasing levels of nonspecific effects inherent in different controls, the effect sizes of AT for biological indices of stress remained at the same level. Overall, the effect sizes for AT fall clearly in the same range as those reported for other arousal reduction methods, and all of these are slightly less effective than psychotherapy at large. In sum, a consistent picture of comparable, moderate effect sizes emerges; subgrouping for techniques produced no meaningful differences, and effect sizes for pre–post changes are essentially the same as for active treatment versus no-treatment controls. Biological indices were more robust than self-report indices to varying types of control comparison.

Given that the aggregation principle of meta-analyses means, at times, indiscriminate "lumping," there still remains the question of which technique is the best match for which problem. Note, however, that this literature is much broader than is needed for a discussion of AT effects. Lehrer and his collaborators (Lehrer, Carr, Sargunaraj,

& Woolfolk, 1994) have presented a detailed review of effective technique to area-of-application matches for arousal reduction strategies. These researchers classify techniques on the basis of their cognitive versus behavioral/autonomic emphasis, with meditation and mindfulness forming the more cognitive end of the spectrum, autogenic training possessing both a cognitive and autonomic rationale, and muscular relaxation and biofeedback being the most physiological, autonomically based techniques. Stress, anxiety, and phobias were considered most responsive to interventions with both strong cognitive and behavioral elements.

Meta-analytic reviews may not reveal much about design differences, which could, however, influence treatment outcomes. In this light, it is important to stress that the majority of AT studies have used less than ideal training programs (because of taped instructions, very brief treatments, and/or only a subset of the six-formula set). I suspect that comprehensive training and personal delivery are bound to make AT more effective. The effect sizes reported here may, therefore, underestimate the maximal effects possible with more appropriate training procedures.

Furthermore, the results of AT and similar self-regulation approaches can be evaluated with a variety of different endpoints. One such example is the use of AT to facilitate the reduction of prescription drug use, as was done with headache patients (Zsombok, Juhasz, Budavari, Vitrai, & Bagdy, 2003). Within 3 months, patients showed significant reductions in use of analgesics and anxiolytics, a welcome finding given the cost of drug treatment and the lower risk of side effects with reduced medication intake.

As mentioned above, there are no recent, large-scale meta-analyses of AT's effectiveness. Nevertheless, AT remains a topic of interest, and small reviews and individual trials continue to be conducted, often for innovative applications. A sampling of such wide-ranging work is offered in Table 18.1.

TABLE 18.1. A Sampling of Recent Reviews and Individual Clinical Outcome Studies with Innovative Treatment Targets

Reviews

- Relaxation training for anxiety: Manzoni, Pagnini, Castelnuovo, and Molinari (2008)
- AT for headache: Seoa et al. (2018)

Individual studies

- Performance enhancement in female collegiate swimmers: Thiese and Huddleston (1999)
- Cardiac autonomic nervous activity in fire service workers at high risk for PTSD: Mitani, Fujita, Sakamoto, and Shirakawa (2006)
- AT for psychosomatic patients with depression: Ota, Majima, Shimura, and Ishikawa (2007)
- Mind–body skills for PTSD and depression in Palestinian children and adolescents: Staples, Abdel, Jamil, and Gordon (2011)
- Stress response and heart rate variability in nursing students: Lim and Kim (2014)
- Effects of audiovisual and autogenic relaxation on amplitude of alpha EEG band and mental work performance in athletes: Mikicin and Kowalczyk (2015)
- Effects of AT on lung capacity, competitive anxiety, and subjective vitality: Ortigosa-Márquez, Carranque-Cháves, and Hernández Mendo (2015)
- Progressive relaxation versus AT in addition to psychotherapy for depression: Krampen (2015)
- AT and cognitive function in women: Singh, Singh, and Singh (2018)

CONCLUSIONS

AT continues to deserve a place in the practice of clinicians working with psychophysiological disorders and in every stress management book, given its long history, enthusiastic endorsement around the world (although somewhat less prominent in English-speaking countries), and the extensive database available for critical evaluations of outcome. Furthermore, this database continues to grow, with demonstrations of numerous applications for target areas previously untested (see Table 18.1). However, it is also clear that there are still remaining questions. There is tentative evidence that AT may be more useful than other self-regulation methods for certain disorders, whereas for others a different method (e.g., thermal biofeedback) may be better. Detecting specific effects will remain difficult, however, because the average effect associated with AT is medium when AT's effects are tested in a pre–post manner or when they are compared against those of no-treatment controls. When different target problems are lumped together, the comparison with other active biobehavioral treatments generally reveals that these interventions produce similar medium-sized effects. It is safe to presume that shared nonspecific treatment elements account for a portion of this effect. A more promising approach is to permit clients to select their own treatment after a range of treatments and their rationales have been presented; clients who then choose AT may respond more strongly because of the a priori credibility that self-chosen methods embody.

At this time, there is no evidence that full-length training (i.e., suggested to last at least 8 weeks) is superior to brief training with a selected subset of formulas. However, this question has never been subjected to a direct test in a single study that has targeted one clearly delineated clinical problem and in which trainees have been randomly assigned to either short or full-length training. The conclusion of no evidence for a difference between short and long training is based on comparisons of effect sizes from short versus long treatments across different studies. This also implies likely confounding of effect size with problem severity, in that less severe problems may have received shorter treatment, and quick recovery may have been attributable to lesser problem severity rather than shorter treatment length.

In summary, there is a strong research base supporting AT's rationale and clinical outcome, but it also seems true that AT is overall comparable in rationale and effect to other self-regulation interventions (such as meditation, muscular relaxation, yoga, or mindfulness; Grossman et al., 2004). Notwithstanding this observation of overall comparable outcomes, further research is welcomed to test possible specificity effects (e.g., some clients may benefit more from AT than others, and we need to know who they are). Also, as Lehrer and Woolfolk (Chapter 22, this volume) suggest, some applications may favor AT for stronger outcomes than others. Resolving issues of ideal client-to-problem-to-technique matches is a laborious task because of the large number of possible comparisons that have to be tested.

A POSTSCRIPT

In over four decades of publishing, the author of this chapter never saw a need to wrap up a chapter or manuscript with a postscript of the following nature. The body of this chapter is devoted purely to a psychophysiological self-regulation technique that was, and is, free of ideology, political aspiration, or demagoguery. AT was developed and propagated solely to help people. As such, AT's developer and propagator, Johannes Heinrich

Schultz, deserves much credit. I also wish I could say "unconditional" credit, but unfortunately I cannot because as an individual and a practicing physician Schultz had vigorously embraced Nazi philosophy (Brunner, Schrempf, & Steger, 2008). He advocated for the literal elimination of people who were feeble-minded individuals whom he perceived as of low value. He also conducted inhumane experiments on homosexual prisoners in concentration camps. Despite my lifelong desire to be tolerant of different beliefs, Schultz's worldviews and actions were not merely different but hateful and inhumane. This then leaves the reader with a very personal decision whether a useful technique can be "surgically detached" from its developer.

REFERENCES

Aivazyan, T. A., Zaitsev, V. P., & Yurenev, A. P. (1988). Autogenic training in the treatment and secondary prevention of essential hypertension: Five-year follow-up. *Health Psychology, 7*(Suppl.), 201–208.

Badura, H. O. (1977). Beitrag zur differentialdiagnostischen Validitaet des MMPI zur Prognose der Efflzienz des Autogenen Trainings [Contribution to the differential diagnostic validity of the MMPI for predicting the outcome of autogenic training]. *Archiv fuer Psychiatrie und Nervenkrankheiten, 224,* 389–394.

Benson, H. (1975). *The relaxation response.* New York: Morrow.

Bernstein, D. A., & Borkovec, T. D. (1973). *Progressive relaxation training: A manual for the helping professions.* Champaign, IL: Research Press.

Brunner, J., Schrempf, M., & Steger, F. (2008). Johannes Heinrich Schultz and national socialism. *Israeli Journal of Psychiatry and Relational Science, 45,* 257–262.

Budzynski, T. H., Stoyva, J. M., & Pfeffer, K. E. (1980). Biofeedback techniques in psychosomatic disorders. In A. Goldstein & E. B. Foa (Eds.), *Handbook of behavioral interventions.* New York: Wiley.

Cannon, W. B. (1933). *The wisdom of the body.* New York: Norton.

Davidson, R. J., Kabat-Zinn, J., Schumacher, J., Rosenkranz, M., Muller, D., Santorelli, S. F., et al. (2003). Alterations in brain and immune function produced by mindfulness meditation. *Psychosomatic Medicine, 65,* 564–570.

Diehl, B. J. M., Meyer, H. K., Ulrich, P., & Meinig, G. (1989). Mean hemispheric blood perfusion during autogenic training and hypnosis. *Psychiatry Research, 29,* 317–381.

Dierks, T., Maurer, K., & Zacher, A. (1989). Brain mapping of EEG in autogenic training (AT). *Psychiatry Research, 29,* 433–434.

Dobeta, H., Sugano, H., & Ohno, Y. (1966). Circulatory changes during autogenic training. In J. J. Lopez Ibor (Ed.), *Fourth World Congress of Psychiatry, Madrid* (International Congress Series No. 117.48). Amsterdam: Excerpta Medica.

Erickson, M. H., & Rossi, E. L. (1979). *Hypnotherapy: An exploratory casebook.* New York: Irvington.

Fischel, W., & Mueller, V. P. (1962). Psychogalvanische Hautreaktionen im Autogenen Training und waehrend der Hypnotherapie. *Zeitschrift fur Psychologie, 167,* 80–106.

Godfrey, K. J., Bonds, A. S., Kraus, M. E., Wiener, M. R., & Toth, C. S. (1990). Freedom from stress: A meta-analytic view of treatment and intervention programs. *Applied HRM Research, 1,* 67–80.

Gonzalez de Rivera, J. L. (1977). Autogenic abreaction. In W. Luthe (Ed.), *Autogenic methods: Application and perspectives.* Rome: Pozzi.

Grossmann, P., Niemann, L., Schmidt, S., & Walach, H. (2004). Mindfulness-based stress reduction and health benefits: A meta-analysis. *Journal of Psychosomatic Research, 57*(1), 35–43.

Haynes, R. B., Taylor, D. W., & Sackett, D. L. (1979). *Compliance in health care.* Baltimore: Johns Hopkins University Press.

Heide, F. J., & Borkovec, T. D. (1983). Relaxation-induced anxiety: Paradoxical anxiety enhancement due to relaxation training. *Journal of Consulting and Clinical Psychology, 51,* 171–182.

Heide, F. J., & Borkovec, T. D. (1984). Relaxation-induced anxiety: Mechanisms and their theoretical implications. *Behaviour Research and Therapy, 22,* 1–12.

Hoelscher, T. J., Lichstein, K. L., & Rosenthal, T. L. (1986). Home relaxation practice in hypertension treatment: Objective assessment and compliance induction. *Journal of Consulting and Clinical Psychology, 54,* 217–221.

Hyman, R. B., Feldman, H. R., Harris, R. B., Levin, R. F., & Malloy, G. B. (1989). The effects of relaxation training on clinical symptoms: A meta-analysis. *Nursing Research, 38,* 216–220.

Ikemi, Y., Nakagawa, S., Kimura, M., Dobeta, H., Ono, Y., & Sugita, M. (1965). Blood flow change by autogenic training—including observations in a case of gastric fistula. In W. Luthe (Ed.), *Autogenes training: Correlations psychosomaticae* (pp. 64–68). Stuttgart, Germany: Thieme.

Israel, L., & Rohmer, F. (1958). Variations electroencephalographiques au cours de la relaxation autogene et hypnotique. In P. Aboulker, L. Chertok, & M. Sapir (Eds.), *La relaxation: Aspects theoriques et pratiques*. Paris: Expansion Scientifique Franchise.

Jacob, R. G., Chesney, M. A., Williams, D. M., Ding, Y., & Shapiro, A. S. (1991). Relaxation therapy for hypertension: Design effects and treatment effects. *Annals of Behavioral Medicine, 13,* 5–17.

Jacob, R. G., Kraemer, H. C., & Agras, W. S. (1977). Relaxation therapy in the treatment of hypertension: A review. *Archives of General Psychiatry, 34,* 1417–1427.

Jacobson, E. (1938). *Progressive relaxation* (2nd ed.). Chicago: University of Chicago Press.

Jacobson, N. S., Follette, W. L., & Revenstorf, D. (1984). Psychotherapy outcome research: Methods of reporting variability and evaluating clinical significance. *Behavior Therapy, 75,* 336–352.

Jus, A., & Jus, K. (1968). Das Verhalten des Elektroencephalogramms waehrend des Autogenen Trainings. In D. Langen (Ed.), *Der Weg des Autogenen Trainings* (pp. 359–375). Darmstadt, Germany: Wissenschaftliche Buchgesellschaft.

Katzenstein, A., Kriegel, E., & Gaefke, I. (1974). Erfolgsuntersuchung bei einer komplexen Psythotherapie essentieller Hypertoniker. *Psychiatrie, Neurologie, Medizinische Psychologie, 26,* 732–737.

Krampen, G. (2015) Progressive relaxation vs. autogenic training in addition to psychotherapy of depression. *Verhaltenstherapie und Verhaltensmedizin, 36,* 7–24.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16,* 495–512.

Lang, P. J., Kozak, M. J., Miller, G. A., Levin, D. N., & McLean, A., Jr. (1980). Emotional imagery: Conceptual structure and pattern of somato-visceral response. *Psychophysiology, 17,* 179–192.

Lang, P. J., Levin, D. N., Miller, G. A., & Kozak, M. J. (1983). Fear behavior, fear imagery, and the psychophysiology of emotion: The problem of affective response integration. *Journal of Abnormal Psychology, 92,* 276–300.

Lantzsch, W., & Drunkenmoelle, C. (1975). Studien der Durchblutung in Patienten mil Essentieller Hypertonie [Studies of the circulation in patients with essential hypertension]. *Psychiatrica Clinica, 8,* 223–228.

Lehrer, P. M. (1982). How to relax and how not to relax: A reevaluation of the work of Edmund Jacobson. *Behaviour Research and Therapy, 20,* 417–428.

Lehrer, P. M., Atthowe, J. M., & Weber, B. S. P. (1980). Effects of progressive relaxation and autogenic training on anxiety and physiological measures, with some data on hypnotizability. In F. J. McGuigan, W. E. Sime, & J. McDonald Wallace (Eds.), *Stress and tension control* (pp. 171–184). New York: Plenum Press.

Lehrer, P. M., Carr, R., Sargunaraj, D., & Woolfolk, R. L. (1994). Stress management techniques: Are they all equivalent, or do they have specific effects? *Biofeedback and Self-Regulation, 19*(4), 353–401.

Ley, R. (1985). Blood, breath and fears: A hyperventilation theory of panic attacks and agoraphobia. *Clinical Psychology Review, 5,* 271–285.

Lim, S. J., & Kim, C. (2014). Effects of autogenic training on stress response and heart rate variability in nursing students. *Asian Nursing Research, 8,* 286–292.

Lindemann, H. (1974). *Ueberleben im Stress: Autogenes Training.* Muenchen, Germany: Bertelsmann.

Linden, W. (Ed.). (1988). *Biological barriers in behavioral medicine*. New York: Plenum Press.

Linden, W. (1990). *Autogenic training: A clinical guide*. New York: Guilford Press.

Linden, W. (1994). Autogenic training: A narrative and a meta-analytic review of outcome. *Biofeedback and Self-Regulation, 19,* 227–264.

Linden, W. (2006). Treating hypertension. In A. Kuczmierczyk & A. Nicevic (Eds.), *A case formulation approach to behavioral medicine.* London: Routledge & Brunner.

Linden, W., Lenz, J. W., & Con, A. (2001). Individualized stress management for essential hypertension: A randomized trial. *Archives of Internal Medicine, 161,* 1072–1080.

Linden, W., & Wen, F. (1990). Therapy outcome research, health care policy, and the continuing lack of accumulating knowledge. *Professional Psychology: Research and Practice, 21,* 482–488.

Luebbert, K., Dahme, B., & Hasenbring, M. (2001). The effectiveness of relaxation training in reducing treatment-related symptoms and improving emotional adjustment in acute non-surgical cancer treatment: A meta-analytical review. *Psycho-Oncology, 10,* 490–502.

Luthe, W. (1963). Autogenic training: Method, research and application in medicine. *American Journal of Psychotherapy, 17,* 174–195.

Luthe, W. (1970a). *Autogenic therapy: Vol. 4. Research and theory.* New York: Grune & Stratton.

Luthe, W. (1970b). *Autogenic therapy: Vol. 5. Dynamics of autogenic neutralization.* New York: Grune & Stratton.

Luthe, W. (1970c). *Autogenic therapy: Vol. 6. Treatment with autogenic neutralization.* New York: Grune & Stratton.

Luthe, W., & Schultz, J. H. (1969a). *Autogenic therapy: Vol. 2. Medical applications.* New York: Grune & Stratton.

Luthe, W., & Schultz, J. H. (1969b). *Autogenic therapy: Vol. 3. Applications in psychotherapy.* New York: Grune & Stratton.

Manzoni, G. M., Pagnini, F., Castelnuovo, G., & Molinari, E.(2008). Relaxation training for anxiety: A ten-years systematic review with meta-analysis. *BMC Psychiatry, 8,* 41.

Martin, A. R. (1951). The fear of relaxation and leisure. *American Journal of Psychoanalysis, 11,* 42–50.

Mensen, H. (1975). *ABC des Autogenen Trainings.* Muenchen, Germany: Goldmann.

Mikicin, M., & Kowalczyk, M. (2015). Audiovisual and autogenic relaxation alter amplitude of alpha EEG band, causing improvements in mental work performance in athletes. *Applied Psychophysiology and Biofeedback, 40,* 219–227.

Mitani, S., Fujita, M., Sakamoto, S., & Shirakawa, T. (2006). Effect of autogenic training on cardiac autonomic nervous activity in high-risk fire service workers for posttraumatic stress disorder. *Journal of Psychosomatic Research, 60,* 439–444.

Miu, A. C., Heilman, R. M., & Miclea, M. (2009). Reduced heart rate variability and vagal tone in anxiety: Trait versus state, and the effects of autogenic training. *Autonomic Neuroscience, 145,* 99–103.

Ohno, Y. (1965). Studies on physiological effects of autosuggestion centered around autogenic training. *Fukuoka Acta Medico, 56,* 1102–1119.

Ortigosa-Márquez, J. M., Carranque-Cháves, G. A., & Hernández Mendo, A. (2015). Effects of autogenic training on lung capacity, competitive anxiety and subjective vitality. *Biomedical Research, 26,* 71–76.

Ota, Y., Majima, F., Shimura, M., & Ishikawa, T. (2007). Applicability of autogenic training for psychosomatic patients with depression. *Japanese Journal of Autogenic Therapy, 27,* 45–52.

Pelliccioni, R., & Liebner, K. H. (1980). Ultraschall-Doppler sonographische Messungen von Blut-stroemungsaenderungen waehrend der Grunduebungen im Rahmen des Autogenen Trainings. *Psychiatrie, Neurologie, und Medizinische Psychologie, 32,* 290–297.

Pikoff, H. (1984). A critical review of autogenic training in America. *Clinical Psychology Review, 4,* 619–639.

Polzien, P. (1953). Versuche zur Normalisierung der S-T strecke und T-zacke im EKG von der Psyche her. *Zeitschrift fur Kreislauf-Forschung, 42,* 9–10.

Ranty, Y. (2007). The evolution of Schultz's autogenic training in France. *European Journal of Autogenic and Bionomic Studies, 1*(2), 55–69.

Sakakibara, M., Takeuchi, S., & Hayano, J. (1994). Effect of relaxation training on cardiac parasympathetic tone. *Psychophysiology, 31,* 223–228.

Sapir, M., & Reverchon, F. (1965). Modifications objectives—circulatoires et digestives—au cours du Training Autogene. In W. Luthe (Ed.), *Autogenes training: Correlations psychosomaticae* (pp. 59–63). Stuttgart, Germany: Thieme.

Schaefgen, E. (1984). Lebensweg von J. H. Schultz nach seinem "Lebensbilderbuch eines Nervenarztes." In G. Iversen (Ed.), *Dem Wegbereiter Johann Heinrich Schultz.* Koln, Germany: Deutscher Aerzte Verlag.

Schultz, J. H. (1932). *Das Autogene Training-Konzentrative Selbstentspannung.* Leipzig, Germany: Thieme.

Schultz, J. H. (1973). *Das Autogene Training-Konzentrative Selbstentspannung. Versuch einer Klinisch-praktischen Darstellung.* Stuttgart, Germany: Thieme.

Schultz, J. H., & Luthe, W. (1969). *Autogenic therapy: Vol. 1. Autogenic methods.* New York: Grune & Stratton.

Schwartz, M. S., & Andrasik, F. (2003). *Biofeedback: A practitioner's guide.* New York: Guilford Press.

Schwarz, G., & Langen, D. (1966). Gefaessreaktionen bei Autogenem Training und niedrigen Raumtemperaturen. In J. J. Lopez Ibor (Ed.), *Fourth World Congress of Psychiatry, Madrid* (International Congress Series No. 117.48). Amsterdam: Excerpta Medica.

Selenta, C., Hogan, B., & Linden, W. (2000). How often do office blood pressures fail to detect true hypertension? *Archives of Family Medicine, 9,* 533–540.

Seoa, E., Hong, E., Choi, J., Kim, Y, Brandt, C. L., & Soo, K. (2018). Effectiveness of autogenic training on headache: A systematic review. *Complementary Therapies in Medicine, 39,* 62–67.

Shapiro, D. A., & Shapiro, D. (1982). Meta-analysis of comparative therapy outcome studies: A replication and refinement. *Psychological Bulletin, 92,* 581–604.

Shapiro, S., & Lehrer, P. M. (1980). Psychophysiological effects of autogenic training and progressive relaxation. *Biofeedback and Self-Regulation, 5,* 249–255.

Singh, A., Singh, T., & Singh, H. (2018) Autogenic training and progressive muscular relaxation interventions: Effects on mental skills of females. *European Journal of Physical Education and Sport Science, 5,* 134–141.

Staples, J. K., Abdel, A., Jamil, A., & Gordon, J. S. (2011). Mind–body skills groups for posttraumatic stress disorder and depression symptoms in Palestinian children and adolescents in Gaza. *International Journal of Stress Management, 18,* 246–262.

Stetter, F., & Kupper, S. (2002). Autogenic training: A meta-analysis of clinical outcome studies. *Applied Psychophysiology and Biofeedback, 27,* 45–98.

Taylor, C. B., Agras, W. S., Schneider, J. A., & Allen, R. A. (1983). Adherence to instructions to practice relaxation exercises. *Journal of Consulting and Clinical Psychology, 57,* 952–953.

Thiese, K. E., & Huddleston, S. (1999). The use of psychological skills by female collegiate swimmers. *Journal of Sport Behavior, 22,* 602–610.

Vinck, J., Arickx, M., & Hongenaert, M. (1987). Predicting interindividual differences in blood pressure response to relaxation training in normotensives. *Journal of Behavioral Medicine, 10,* 395–410.

von Siebenthal, W. (1952). Eine vereinfachte Schwereuebung des Schultz'schen Autogenen Trainings. *Zeitschrift fuer Psychotherapie und Medizinische Psychologie, 2,* 135–143.

Wallace, R. K. (1970). Physiological effects of transcendental meditation. *Science, 167,* 751–754.

Wallnoefer, H. (1972). *Seele ohne Angst-Hypnose, Autogenes Training, Entspannung.* Hamburg, Germany: Hoffmann & Campe.

Zsombok, T., Juhasz, G., Budavari, A., Vitrai, J., & Bagdy, G. (2003). Effect of autogenic training on drug consumption in patients with primary headache: An 8-month follow-up study. *Headache, 43,* 251–257.

CHAPTER 19

The Treatment of Pain and Stress with Hypnosis

A Series of Emerging Literatures

Robert A. Karlin

It has been 14 years since the last version of this chapter. During that time, there have been relatively few studies directly focusing on control of stress with hypnosis. However, there has been considerable work on hypnosis for disorders causing chronic pain and procedural pain. Furthermore, it seems possible, at least to some degree, to specify the mechanisms underlying success with chronic and procedural pain.

HYPNOSIS: A (VERY) BRIEF HISTORY

Hypnosis may be seen as the oldest psychotherapeutic procedure still used in mainstream psychotherapy. Although claims for the use of hypnotic phenomena in more remote periods are common, we can certainly trace the roots of modern hypnotic procedures to the medical practice in Vienna and Paris of Franz Anton Mesmer (1734–1815) during the 1770s and early 1780s. Mesmer, a qualified physician, thought that there was an invisible force, the animal magnetic fluid, which he could accumulate in his own body, then transmit to ill people and thereby restore them to health.

Mesmer's methods were a combination of laying-on-of-hands and the application of magnets to the patient's body. In fact, they bore a close relationship to exorcism, a procedure that he, as a scientist-practitioner, derided. Mesmer's treatment often resulted in epileptiform seizures, followed by a sleep from which the patient awakened cured of symptoms. Today, Mesmer's theory of an animal magnetic fluid is absurd in light of the science of our time, and it was falsified by the scientists of the 1780s (Franklin et al., 1784/1970). However, it was also the case that he and his disciples cured illnesses and ameliorated symptoms that more orthodox practitioners found untreatable (Ellenberger, 1970).

Mesmer moved to Paris in 1777 and soon acquired proponents for his views, eventually including the Marquis de Puységur, the head of one of France's oldest and wealthiest families. Mesmer retreated to Switzerland after a scientific commission, chaired by Benjamin Franklin (1784/1970), showed Mesmer's theory about the fluid to be incorrect. However, Puységur and other members of the mesmeric Societies of Harmony carried on Mesmer's work. Within the next 30 years, Puységur and his colleagues had demonstrated all the major hypnotic phenomena, including amnesia, analgesia, motor catalepsies and automatisms, positive and negative hallucinations, and posthypnotic effects of suggestion (Ellenberger, 1970). Additionally, they demonstrated enduring individual differences in responsiveness to hypnotic suggestion (Shor, 1979).

Hypnosis went through a number of ups and downs during the first three-quarters of the 19th century (including having animal magnetism and mesmerism renamed *neurohypnosis* by Braid in 1843). The modern study of hypnosis began with the work of two French neurology professors, Jean Charcot in Paris (Charcot & Richer, 1882/1978) and Hippolyte Bernheim in Nancy (Bernheim, 1884). Presaging the state–no-state controversy of the last half of the 20th century, Bernheim emphasized the lack of difference between waking suggestion and response to hypnosis, whereas Charcot (erroneously) delineated specific stages of hypnosis and their unique signs. Freud studied with both Charcot and Bernheim. He then returned to Vienna, used hypnosis in his neurology practice, and then abandoned it. In fact, hypnosis was the first victim of Freud's problematic "symptom substitution" hypothesis (Ellenberger, 1970).

Interest in hypnosis has continued throughout the 20th century. In addition to Freud, Charcot, and Bernheim, a highly regarded group of psychologists and psychiatrists spent some part of their careers working on hypnosis during the years from 1878 through 1945. Among others, these included Vladimir Bechterev, Henri Bergson, Alfred Binet, Eugen Bleuler, Sandor Ferenczi, Clark Hull, William James, Pierre Janet, William McDougall, Henry Murray, Ivan Pavlov, Morton Prince, Charles Richet, and Wilhelm Wundt (Laurence & Perry, 1988). The second half of the 20th century was dominated by another group of superb researchers: Theodore Barber, Milton Erickson, Erika Fromm, Ernest and Josephine Hilgard, Martin and Emily Orne, and Phillip Sutcliffe. Their students (e.g., Kenneth and Patricia Bowers, Gail Gardner, Campbell Perry, Ronald Shor, and Nicolas Spanos) and their student's students (e.g., Jean-Roch Laurence and Kevin McConkey), as well as other talented and careful young scholars (e.g., Irving Kirsch, Steven Lynn, and Judith Rhue) have spent a good deal of their careers studying hypnosis. As well as elucidating the domain of hypnosis, their careful investigations have increased understanding of the potential pitfalls that appear in many forms of experimentation with human research participants (cf. Orne, 1959, 1970).

Modern experimental studies of hypnotic phenomena have involved such issues as the existence of a "trance" state (cf. Barber, 1969; Kirsch & Lynn, 1995), the modifiability of hypnotizability (cf. Gfeller, Lynn, & Pribble, 1987; Gorassini, Sowerby, Creighton, & Fry, 1991; Perry, 1977), the role of social and cognitive factors in hypnotic phenomena (cf. Lynn & Rhue, 1991; Spanos, 1996) and neural correlates of hypnotic phenomena (cf. Horton & Crawford, 2004; Karlin & Orne, 2001; Raz, 2004; Vaitl et al., 2005). Modern clinical work on hypnosis has involved an integration of science, art, and clinical lore (cf. Lynn, Kirsch, & Rhue, 1996; Jensen & Patterson, 2014) and to some degree has attempted to deal with the differentiation of specific versus common effects of clinical hypnosis (Rhue, Lynn, & Kirsch, 1993; Wampold & Imel, 2015).

THEORETICAL FOUNDATIONS

Typical Response to Hypnotic Suggestions

A contemporary observer, watching a demonstration of hypnosis with highly hypnotizable research participants (highs), sees a series of simple verbal suggestions that result in relatively spectacular alterations of behavior, thought, emotion, and perception. For example, the hypnotized burn patient lies quietly and reports little or no pain, seemingly relaxing while dressings are changed (a highly painful procedure). If laboratory research participants who are asked to reexperience the distant past respond well to hypnosis, they seem to become childlike, entirely captured by the delusion that it is many years earlier. Asked to hallucinate the absence of an obstacle between where the participants sit and some other place, such participants claim to see nothing in the intervening space. But when asked to walk to that place (and thus through the obstacle whose absence is being hallucinated), they may walk around the obstacle without seeming to notice doing so. Asked to look back and see that there is nothing there, they agree that there is nothing there and show no seeming sense that walking around the obstacle has just contradicted this statement. Asked to forget all that has happened until a pencil is tapped twice, they will later "awaken" with reversible amnesia.

Thus, in response to brief verbal suggestions, highly hypnotizable individuals seem to see, hear, feel, smell, and taste in apparent contradiction to the stimuli actually present. Their thinking seems to tolerate logical incongruities more easily than usual. Memory, the sense of volition, mood, and even awareness of self may be altered. With appropriate suggestions, such effects may be extended for some time into the posthypnotic period.

Hypnotic phenomena are easy to elicit and are robust. A graduate student, given 2–3 hours of training and a standardized script (such as the one for the Stanford Hypnotic Susceptibility Scale: Form C; Weitzenhoffer & Hilgard, 1962), can elicit all the major hypnotic phenomena in a psychology laboratory. There, the hypnotized research participant can be studied under controlled conditions. Note that, for all practical purposes, hypnotic suggestions are likely to be carried out only in settings seen as culturally appropriate. These include therapeutic, classroom, entertainment, research, and forensic settings.

Hypnotizability as a Stable Individual Difference

People manifest remarkably stable differences in response to hypnotic instructions. For example, Piccione, Hilgard, and Zimbardo (1989) examined responses to the standardized inductions and suggestions of the Stanford Scale of Hypnotic Susceptibility, Form A (Weitzenhoffer & Hilgard, 1959), starting when participants were Stanford undergraduates and then at 10, 15, and 25 years later. The correlation coefficient over 25 years was .70. That test–retest reliability correlation is of the same order as that found for multimeasure tests of IQ.

Some people are highly hypnotizable (and are called "highs"); some are moderately hypnotizable (called "moderates"); and a few are not hypnotizable at all ("lows").[1] Measurement of hypnotizability is relatively simple: One takes a work sample. Hypnotizability is defined by participants' responses to standardized scripts. In such scripts, the induction of hypnosis is followed by a standardized series of suggestions. There have been a number of attempts to create such scales, but most recent research has used the Stanford Scales (SSHS; Weitzenhoffer & Hilgard, 1959, 1962, 1967) or one of its derivatives. Scales directly derived from or strongly correlated with the Stanford Scales have been used in a variety of situations and with a variety of different populations. For example, there are scales for children, self-scored group scales, scales to be administered without formal induction, and scales for clinical rather than experimental settings. In general, the scales have contained 5–12 suggestions that sample the realm of hypnotic phenomena. These suggestions range from easy suggestions (to which most willing participants respond with the suggested behavior and experience) to difficult suggestions that are "passed" by only a small minority. Easy suggestions include requests for motor movement without seeming volition (e.g., "As you feel the [hallucinated] force pulling your hands together, your hands will begin to actually move together"). Slightly harder items involve challenges. These challenge suggestions are usually related to some form of motor catalepsy (e.g., "Your arm is so heavy, you cannot lift it. Try to lift it. Just try"). Suggestions passed by the smallest percentage of the normal population require a direct and major change in cognition, perception, affect, or memory. Suggestions for positive and negative hallucinations, amnesia, and the like fall into this category. For example, one might present a display board showing three large colored squares and suggest that participants open their eyes and see the two colored squares on the board. A highly hypnotizable participant and some moderates will be aware of only two squares, not three, in response to that suggestion. In general, no more than 30% of participants successfully respond to these more difficult suggestions.[2]

Responsiveness to hypnosis is usually measured by observing the presence or absence of a key behavioral response to each suggestion (although experiential scoring can also be used). For example, the "hands together" suggestion starts with the hands about a foot apart. The suggestion takes less than a minute to make. If at the end of that period the hands are 6 or fewer inches apart, the item is scored as a pass. Similarly, if someone is asked to hallucinate a voice asking a question over a loudspeaker, passing or failing is determined by whether or not the participant verbally responds to the imaginary question. Score on the total scale is simply the number of items passed. Point–biserial correlations between passing or failing a single item and the number of other items passed on a scale range from about .4 to .7, and average about .6 (Hilgard, 1965). So there is good evidence for an underlying hypnotizability factor similar to "G."

Hypnotizability is not easily modifiable and governs the large majority of the variance in most experimental studies of hypnotic phenomena (cf. Perry, 1977).[3] It is important to note that what has come to be called "hypnotizability" is observable in response to suggested alterations in perception, memory, and/or cognition with or without formal hypnosis. That is, motivational instructions and/or creation of appropriate set and setting can abrogate the need for the formal induction of hypnosis when making suggestions usually seen as "hypnotic" (cf. Barber, 1969).

Are there truly differences between highs and their less hypnotizable counterparts other than self-reported hallucinations? Although peripheral physiological measures show no differences unique to hypnosis or specific hypnotic suggestions, studies indicate that hypnotic suggestion to hallucinate alters central nervous system (CNS) activity among highs but not among moderates or lows. Using a number of measures of brain function, one finds that highs differ from their moderately hypnotizable counterparts in ways consistent with their reported hallucinatory experiences. In an early study, Karlin, Morgan, and Goldstein (1980) compared the quantitated EEG responses of high and moderately hypnotizable individuals to unilateral cold-pressor pain after hypnotic analgesia instructions. Moderates responded to hypnotic analgesia instructions with lower levels of self-reported pain, but their EEG responses were what one would expect with unilateral stimulation: activation of the contralateral hemisphere. The highs showed even lower levels of reported pain and no change in hemispheric activation when right versus left hands were immersed in 0–2°C (approximately 32–35°F) water and ice. These findings—that highs show hallucination-consistent brain activation during reported hallucinations—have now been replicated by other laboratories across a number of tasks and a variety of measures of brain function. For example, in evoked-response studies involving both visual and auditory stimuli and across experiments, highs showed evoked responses whose components were consistent with suggested hallucinations. When positive hallucinations were suggested, highs showed augmented or faster positive components of evoked potentials and/or decreased or delayed negative components of the evoked potentials. Conversely, when negative hallucinations were suggested,[4] when compared to their less hypnotizable counterparts, highs show decreased or delayed positive components of evoked potentials and/or augmented or faster negative evoked potentials (cf. De Pascalis, Cacace, & Massicolle, 2008; Karlin & Orne, 2001; Rainville, Carrier, Hofbauer, Bushnell, & Duncan, 1999; Spiegel, 1989) Thus we are fairly sure that reported positive and negative hallucinations are at least accompanied by theoretically relevant changes in function of the brains of highly hypnotizable research participants, but not by their less hypnotizable peers.

Trance versus No Trance: A Definition of the Situation

As noted above, the vast majority of the variance in response to experimental hypnosis centers on hypnotizability and questions of the reality of hallucinations in response to verbal suggestion. But what about "trance"? Researchers have tried to determine whether or not there is something really special going on after a hypnotic induction. Much of the academic debate in the second half of the 20th century was spent on the question of *trance* (that hypnosis involves the induction of a special state of consciousness) versus *no trance* (that we can explain hypnotic phenomena with concepts drawn from social psychology, such as set and setting). Although that question was more or less settled with the conclusion that trance was a purely subjective experience, irrelevant to most experimental findings, in the clinical situation the experience of trance seems quite important. I will discuss this important difference between clinical and experimental settings later in the chapter.

People commonly speak of being "hypnotized" or in a hypnotic "trance." The underlying assumption is that hypnosis is a distinct, altered state of consciousness. Most early theorizing (and much current clinical thought) about the hypnotic state employed circular reasoning that goes something like this: *Q:* Why do you say that person is in a hypnotic trance? *A:* Because he is responding to hypnotic suggestions. *Q:* Why does he respond to hypnotic suggestions? *A:* Because he is very suggestible. *Q:* Why is he so suggestible? *A:* Because he is in a hypnotic trance.

In fact, the hypnotized participant acts as shared cultural expectations suggest (Orne, 1959, 1970). The deeply relaxed hypnotic participant, sitting quietly while listening attentively to the hypnotist's voice, is demonstrating only one of many forms hypnosis has assumed. In the 1770s, Mesmer's Parisian patients often had epileptiform seizures during hypnosis. A century later, Charcot's Parisian patients demonstrated first catalepsy, then lethargy, and finally somnambulism (but generally did not have seizures) when deeply hypnotized. At the same time, in the south of France, Bernheim's hypnotized patients simply reclined restfully (Ellenberger, 1970). Most modern research participants and patients usually sit quietly and look very relaxed during hypnosis, although hypnosis can be induced while the participant engages in aerobic exercise (Banyai & Hilgard, 1976).

Peripheral neural activity during these varying hypnotic activities differs widely but generally reflects the activity, not hypnosis. There has been little, if any, evidence of differential brain activity that accompanies either induction or the entire hypnotic experience.[5] After 70 years of modern research, the lack of evidence for any unique psychophysiological marker of a hypnotic trance state is one of the factors that have led researchers to largely abandon the concept of a hypnotic trance as a causal explanation for response to hypnotic suggestions (cf. Kirsch & Lynn, 1995).

As noted above, the ability to respond to hallucinatory suggestions lies largely in the individual, not in the skill or will of the hypnotist, nor in the particular wording of an induction, nor in the evocation of a deep trance. Reports of being hypnotized or in a trance and reports about the depth of trance experienced are seen as reflecting a subjective experience that is one of the many effects of cognitive and emotional set, social and historical setting, and suggestion (Lynn & Rhue, 1991). However, in the clinical situation, these subjective reports about the experience of trance are important. As a clinician, one usually wants patients to attribute their responses to hypnotic induction to a special state of mind. Viewing themselves as hypnotized raises the patients' expectancies and, as will be discussed later, improves their outcomes in major ways (cf. Kihlstrom, 2013).

Hypnotizability in the Clinical Context

Stable individual differences in hypnotizability allow interesting interpretations of clinical efficacy studies. Lows, who show a clear lack of responsiveness to hypnosis, are relatively rare in clinical practice. Moderates, who constitute the large majority of patients seen in clinical practice, are motivated to experience hypnosis and garner its benefits but are unable to experience major hallucinations in response to verbal suggestions. With all the goodwill in the world, they do not see a suggested elephant in one's hand that is plainly visible to the highs. However, moderates do respond to easier suggestions with both objective movement and subjective experience. For example, it is the relatively rare patient who will not report a force pulling their hands together when one is suggested or one arm getting lighter and the other heavier when that is suggested. In this way, moderates can experience themselves as hypnotized and expect themselves to improve accordingly.[6]

Thus, the large majority of patients in clinical practice are willing to enact the role of hypnotized participants. In fact, as healing may be involved, most actively want to experience themselves as hypnotized. Hypnotic effects involve beliefs, role enactment, outcome expectancies, and similar factors. The majority of the effects of hypnosis in the clinic and hospital may be seen as contextual or placebo effects, although some may result from high hypnotizability. Fortunately, we can look at the literature and generally distinguish which mechanisms are being accessed in specific instances.

ASSESSMENT OF THE EFFICACY OF HYPNOSIS

Hypnosis is used to ameliorate a very wide range of problems in behavioral medicine and psychotherapy. The authors in Elkins's (2017) recent compendium discussed 34 medical conditions and situations and 22 psychological applications. The medical conditions ranged from asthma to prostate cancer, from irritable bowel syndrome (IBS) to warts. The psychological conditions ranged from generalized anxiety disorder (GAD) to improving sleep, from addictions to obsessive–compulsive disorder (OCD). Hypnosis is used for the hyperemesis of pregnancy and the anticipatory and consequent side effects of chemotherapy. It is used pre-, intra-, and postoperatively and before and after other painful medical procedures, such as bone marrow aspiration and changing the dressings of severely burned patients. Hypnosis is used in psychotherapy to aid psychoanalysis and cognitive-behavioral therapy (CBT). It may be used alone or as an adjunct to other approaches.[7]

There has been a virtual explosion of work with hypnosis in medical contexts in the last 30 years. Unfortunately, there has not been a corresponding explosion in work on psychological problems. In the last edition of this book, I was able to focus on both pain and stress. This time the emphasis must be on pain research far more than on hypnosis in the treatment of stress.[8] However, with my potential audience in mind, I have left the script for treatment and the case description below focused on psychological, not physical problems.

Hints from Hypnotic Analgesia in the Laboratory

Given that hypnotic analgesia is of interest both to experimental hypnosis researchers, who view it as a negative hallucination, and by clinicians interested in ameliorating pain and distress, it is unsurprising that the largest single body of research on the clinical efficacy of hypnosis has been in this area. As discussed earlier in regard to physiological correlates of specific hypnotic suggestions, researchers have given us an interesting view of some of the possible brain and attentional mechanisms that underlie the hallucinatory aspect of hypnotic analgesia among highly hypnotizable patients. But the ability to hallucinate and thereby banish or ameliorate painful sensation is far from the whole story in regard to clinical phenomena.

Highly hypnotizable individuals constitute 0.3–30% of the population, depending on how strictly one sets the criteria for being a "high." Lows, about 15% of the population in experimental settings, are quite rare in clinical settings, making up no more than 2–5% of patients hypnotized in the course of treatment.[9] The large majority of patients will be moderately hypnotizable; they will be cooperative and doing their best but unable to engage in hallucinatory control of moderate or severe pain. For this large majority, other mechanisms than hallucination, including beliefs, outcome expectancies, role enactment, and distraction, will have to ameliorate the pain.

Some initial understanding of the interaction among these factors can be gained through thoughtful, careful reviews, such as the work of Montgomery, DuHamel, and Redd (2000). These authors did a meta-analysis of the effect of hypnotic analgesia instructions on reported pain using 27 effect sizes from 18 studies. After weighting effect sizes and variability for sample size, they found that hypnosis had a moderate effect on reported pain ($d = 0.67$, $\mathrm{Var}(d) = 0.26$, $p < .01$) when hypnotic and nonhypnotic conditions were compared. Ten of the 27 effect sizes came from patient samples, and 17 effect sizes came from studies with student volunteers. There was no significant difference between the two (clinical, $d = 0.74$; experimental, $d = 0.64$).[10] Additionally, meta-analytic examination allowed comparison among high ($d = 1.16$), moderate ($d = 0.64$), and low ($d = -0.01$) hypnotizability groups.

Notice that the overall effect size of 0.67 is almost identical to that seen among the moderates ($d = 0.64$).[11] As noted, moderates make up the large majority of patients. Generally, they cannot produce major hallucinations involving the absence of painful stimulation in controlled (laboratory) settings. Thus this effect must be based on the kind of contextual factors (e.g., beliefs, outcome expectancies, role enactment, and distraction) inherent in hypnotic analgesia instructions.[12] Among the highs (*d* = 1.16), we get another half a standard deviation of effect size in the laboratory. Allowing for sampling fluctuation, this suggests two additive effects in the range of 0.5–0.7, one related to defining the situation as hypnosis and the other related to the hallucinatory abilities of the highs when being treated for a condition to which hallucinatory ability is relevant.

Hypnosis provides a setting for and a social role in which suggestions to control pain and experience other benefits are seen as reasonable and appropriate. Moreover, hypnosis provides a method for managing and coping with a variety of problems. It changes expectancies about controlling pain and provides a kind of relaxation and cognitive distraction that help patients do so. Finally, for those who can actually alter perception in response to verbal suggestion, an additional, hallucinatory effect may emerge if the painful stimulus is not overwhelming. This view is in line with the results of the most careful investigations of experimental pain (e.g., McGlashan, Evans, & Orne, 1969).

Hypnosis as an Adjunct to CBT for the Treatment of Stress

There is an enormous volume of research on clinical outcomes involving CBT. In a highly influential meta-analysis, Kirsch, Montgomery, and Sapirstein (1995) found 18 studies in which similar CBT treatments were administered in a social context that was either hypnotic or nonhypnotic.

The 18 studies that were analyzed in the Kirsch et al. (1995) report comprised 20 comparisons of hypnotic with nonhypnotic CBT groups, with a total of 90 effects and 577 participants. Larger positive effects tended to occur in larger samples. The average weighted effect was 0.66, so the average person receiving CBT in a hypnotic context did as well as the person at the 75th percentile or so of those receiving CBT without hypnosis.

Of these studies, Kirsch et al. (1995) identified 14 in which the only difference between hypnotic and nonhypnotic conditions was the use of the word *hypnosis* during relaxation instructions and training. The average weighted effect size for interventions labeled *hypnotic,* compared with the same intervention without the label *hypnosis,* for these 14 studies was *d* = 0.63. Thus labeling an intervention as *hypnotic* increases its efficacy by more than half a standard deviation.

Seven studies comprising 157 patients compared the effectiveness of hypnotic versus nonhypnotic studies in the treatment of anxiety or anxiety-related disorders. There were two studies each of anxiety, insomnia, and hypertension, and one of snake phobia. Examining these seven studies, one can compute an average weighted effect size for these seven studies as *d* = 0.54. So internal analyses provide a similar picture to the one provided by the analysis of all 18 studies: Defining a situation as hypnotic results in an increase in efficacy of about half a standard deviation (*d* ~ 0.5), what Cohen (1998) would identify as a moderate-sized effect.[13]

Since the Kirsch et al. (1995) review, the small literature on hypnosis and CBT supported Kirsch and colleagues' conclusions (e.g., Bryant, Moulds, Guthrie, Dang, & Nixon, 2003; Schoenberger, 2000). However, in the Bryant et al. (2003) study, positive results were unrelated to hypnotizability as measured by the Stanford Hypnotic Clinical Scale (SHCS; Morgan & Hilgard, 1978a).

There have also been a number of studies on the effects of pain and hypnosis on brain activation and immune system function. The two major studies on hypnosis and immune system dysregulation in response to stress had somewhat different findings. Kiecolt-Glaser, Marucha, Atkinson, and Glaser (2001) found that hypnosis reduced the immune system problems occasioned by acute stress. The strongest effects were seen in those who practiced hypnotic relaxation more frequently. Whitehouse and colleagues (1996) examined medical students at various points in the school year. Hypnosis improved self-report of stress but did not produce differences in immune function when compared with the control group. However, in the hypnosis group, those who reported more relaxation during self-hypnosis practice did show some evidence of better immune system function. The parallel in the two studies is intriguing. Finally, there have been studies on CNS response to clinical pain and hypnosis. Although generally positive, a detailed review of these studies lies beyond the scope of this chapter.

Overall Snapshot of the Effects of Hypnosis: Methodology Matters

In the preceding discussion, the results seemingly supported the notion that hypnosis has two specific effects, one related to hypnotizability and the other related to the particular history, myths, and role-related expectancies associated with hypnosis.

Flammer and Bongartz (2003) provided the most comprehensive overall review of the literature on the effects of hypnosis in clinical situations. Their review, still the most comprehensive, also looks more briefly at the relationship between research design and outcome. From a database of 444 reports, the authors found 57 randomized clinical trials involving between-groups comparisons. These studies yielded an overall weighted effect size of $d = 0.56$.[14] They found 18 studies with randomized designs in which effect sizes were computed from pre–post difference scores. These 18 studies had a mean weighted effect size of $d = 0.93$. Nonrandomized trials with between-groups comparisons (22 studies) had an average effect size of $d = 0.98$. The remaining 36 studies were nonrandomized trials using pre–post difference scores and had a much larger effect size ($d = 2.29$).[15]

Experimenters who also have clinical practices often complain that the large effects they see in the clinic are seldom reflected in their own randomized controlled trials (RCTs). In this light, it is intriguing to realize that clinical practice involves interaction similar to that found in trials with nonrandomized, pre–post designs, designs that have an average effect size in the vicinity of 2.00.

The Effects of Hypnotic Analgesia Instructions Outside the Laboratory

As noted earlier, the literature on the use of hypnosis for pain control in the clinic has grown enormously over the last 20 years or so. In the recent past, there were some controlled trials and a high number of anecdotal reports. Now, we have a literature ranging from the neurobiology of pain (e.g., Apkarian, 2015) to the effects of hypnosis on pain from chronic disease (cf. Elkins, Jensen, & Patterson, 2007; Jensen & Patterson, 2014) to hypnosis for the amelioration of acute procedural pain (Kendrick et al., 2016). Additionally, there are illness-specific studies that range from syndromes accompanied by a lifetime of gradually increasing pain, as in sickle cell disease (Dinges et al., 1997), to moderate, chronic episodic pain such as in IBS (Palsson, Turner, Johnson, Burnett, & Whitehead, 2002; Whorwell, Prior, & Faragher, 1984) and most chronic back pain (e.g., Tan et al., 2015). Finally, there has been a good deal of work on hypnosis for both children and adults who need to undergo painful medical procedures, such as bone marrow aspiration (e.g., Liossi & Hatira, 2003; Liossi, White, & Hatira, 2006; Montgomery, David, Winkel, Silverstein, & Bovbjerg, 2002).

Hypnosis, Sickle Cell Disease, and Severe Pain

The most careful work on hypnosis as a treatment for unpredictable, severe episodic pain among adults was done by Orne, Dinges, and their colleagues in the late 1980s and early 1990s (Dinges et al., 1997). This group, directed by David Dinges since Martin Orne's death, is justly famous for the care and quality of its research, as well as its clinical expertise. In research headed by Dinges, they treated 37 patients with sickle cell disease—11 children, 17 adolescents, and 9 adults. The patients were referred by local hospital sickle cell clinics and were on the more severe part of the sickle cell spectrum, with 24 of the 37 participants having the full homozygous syndrome. This, in fact, was a pilot study. Only the rare quality of care and of the research, combined with the severity of the disorder, renders it important.

Sickle cell disease is a quite terrible genetically based disorder resulting from misshaped oxygen-carrying red blood cells. It is found in about 0.3% of infants with an African heritage and is the most serious genetic disorder facing African Americans. The sickle-shaped cells occlude normal blood flow in the capillaries, resulting in ischemia with consequent moderate to severe pain. If the cells block blood flow to a major organ, it is a medical emergency. Anti-inflammatories and opioids are used as palliative treatment. Pain, starting in early childhood, tends to worsen as patients age, and a lifespan of 40–60 years is expected.

The Dinges et al. (1997) study began with a 4-month treatment-as-usual (TAU) baseline. Then patients received 18 months of cognitive-behavioral and self-hypnosis treatment. Treatment was by two senior psychiatrists, experts in pain control, in small, age-appropriate groups. Patients were seen in groups once each week for the first 6 months of treatment, once every 2 weeks during months 7–12, and once every 3 weeks for months 13–18. Patients completed daily records of pain and medication. Parents aided their children, supportive phone contacts were made, and reports for about 75% of days, a very high level of compliance, were collected. Overall, 89% of pain days were treated at home, while 11% of pain days required emergency room treatment.

The treatment resulted in fewer reported days with pain. However, this finding reflected a decrease in days of relatively mild pain. Self-hypnosis failed to affect the number or disabling nature of the more severe pain days. Hypnotizability was measured with the Harvard Group Scale of Hypnotizability (Shor & Orne, 1962) for about half the patients, but it had no discernible relationship with any of the study's outcome measures. The authors concluded that self-hypnosis was some help, specifically with response to mild pain days and emphasized the role of nonspecific effects.

I have dwelt at some length on this study because of the quality of the work and its orientation. Dinges, Orne, and their colleagues could not influence the experience of unpredictable, severe, episodic pain, although patients had 18 months of expert treatment and self-hypnosis practice. The Dinges et al. (1997) study strongly suggests that there are chronic disorders and pains beyond sufficient amelioration with any psychosocial treatment (cf. Heutink et al., 2012.) More specifically, these results suggest that success in similar situations will be limited to what the context of treatment with hypnosis can provide.

Clinical Findings on the Use of Hypnosis for IBS

Let's turn to the treatment of a less severe episodic pain disorder, IBS, with hypnotherapy and other nonpharmacological methods. IBS is a diagnosis reached only after the exclusion of a number of gastrointestinal problems, such as colorectal cancer, inflammatory bowel disease, and celiac disease. Onset is usually early in life, and about two-thirds of patients are women. Usual medical management involving diet, medications, and minor lifestyle changes is helpful in about half the cases. Despite the ongoing problem of failure to find any pathophysiology concurrent with or worsening with IBS, the symptomology is far from trivial.

In IBS, abdominal pain is usually accompanied by diarrhea, constipation, or both. Often, there is distension and/or flatulence. Pain often decreases with defecation, although, especially with diarrhea, several trips to the toilet may be necessary after a meal. IBS symptoms generally decrease quality of life, sometimes prevent attending school or work, and can make voluntary socializing difficult. It also occasions decreased physical activity and decreased sex drive. IBS is often accompanied by comorbidities such as depression, anxiety, and chronic fatigue syndrome.

A variety of psychological interventions have been tried, including stress management training, psychodynamic approaches, CBT, and hypnosis. Hypnosis has produced quite positive results with patients and has aroused a good deal of interest. Generally, these patients have been unsuccessfully treated with diet and medication and have fairly severe remaining symptoms. About half of these refractory patients show a reduction in symptom severity of at least 50% with hypnotherapy. Following induction, such hypnotherapy usually involves images of a healthy flow of material through the digestive system.

Systematic research on the utility of hypnotherapy for IBS was first reported by Whorwell and his colleagues in the 1980s (Whorwell et al., 1984). In the 1984 paper, 30 patients were randomly assigned to either a hypnotherapy or placebo–psychotherapy condition. In the hypnosis condition, patients were asked to put their hand on the abdomen, feel a sense of warmth, and then a sense of asserting control over gut function. All patients in both conditions were treated by Whorwell. He administered seven individual sessions, each 30 minutes long. Assessment was conducted by an independent rater (the second author, Prior). The hypnotherapy patients lost almost all pain and distension, whereas the control patients showed little change.[16] Follow-up research by Whorwell and his colleagues continued to show strong positive hypnotherapy effects on refractory IBS patients (e.g., Whorwell, Prior, & Colgan, 1987). Moreover, as Whorwell reported and later research showed, these positive effects lasted over periods as long as studies continued, up to 5 or 6 years (cf. Gonsalkorale, Miller, Afzal, & Whorwell, 2003). Gonsalkorale and colleagues (2003) reported that 71% of referred patients showed an initial positive response to hypnotic therapy. Of these responders, 81% maintained their improvement over time, and most of the other 19% reported only slight deterioration. Again, hypnotizability did not seem to play a part in these results, and we are left with contextual elements rendered more powerful by the ritual of hypnosis.

Of course, as positive findings continued to appear, research designs generally improved. Multiple clinicians were used with some attempts at blinding. Treatment manuals evolved, and other research groups used differing control conditions (e.g., Dobbin, Dobbin, Ross, Graham, & Ford, 2013; Gonsalkorale, 2006; Palsson et al., 2002). Almost all published trials showed continued positive effects of hypnotherapy on IBS.

It must be noted that the control conditions created by the authors of these studies usually fell, at best, into the pseudo-placebo control category discussed by Wampold and Imel (2015). Control conditions had less structure, allegiance, and credibility, and some staff were less well trained, than in the hypnotic condition(s). Sometimes, control conditions lacked even the label "therapy." So, although hypnosis may have emerged triumphant in a number of IBS randomized studies, we don't know whether the differences are related to hypnosis per se or to experimental designs that advantaged hypnosis. Toward the end of this chapter, we discuss the importance of this from the points of view of researcher and clinician. It should come as little surprise that those points of view may be quite different from each other and that each may be legitimate in their spheres (cf. Hilgard & Hilgard, 1975).

However, this seems the place to briefly illustrate this problem. In a very recent registered trial called the IMAGINE study, Flik and colleagues (2019) studied 354 IBS patients referred to 11 hospitals in the Netherlands.[17] Patients were randomly assigned to six sessions of individual hypnotherapy (n = 150), group hypnotherapy (n = 150), or a "group educational supportive therapy" (n = 54). Individual and group therapy patients were treated by the same clinicians: all qualified psychologists previously trained as hypnotherapists.

The "group educational supportive therapy" control group provided participants with information about IBS, including the need for exercise and some of the dietary aspects of IBS. Treatment of the control group was provided by somewhat lower level professionals, nurse practitioners, or psychological assistants. From the description of the control condition, one is left with the impression that it is a less credible and less structured condition than either hypnosis condition. Purely on that basis, it could be expected to engender less, if any, response (cf. Frank, 1961; Wampold & Imel, 2015). Unsurprisingly, both individual and group hypnotherapy resulted in better outcomes than the control condition. Individual and group hypnotic treatments did not significantly differ from each other.

With very occasional exceptions (e.g., Dobbins et al., 2013), other recent RCTs studying IBS have looked similar to the IMAGINE study (Flik et al., 2019). So the most intensely researched area in the last 35 years leaves us where most "empirically supported therapies" (Chambless & Hollon, 1998) wind up. The procedure works fairly well for a reasonable number of people but has not been shown to outperform other active, brand-name, credible control conditions administered by their advocates. As for mechanisms, hypnotizability was rarely examined, and attempts to define other specific mechanisms have failed (cf. Palsson et al., 2002). It seems likely that contextual effects account for the majority of the variance. However, the contribution of hallucinatory ability by some highs cannot be definitively dismissed.

It Ain't Hypnotizability: Patterson and Jenkins

Patterson and Jensen have produced the most comprehensive line of recent research on the efficacy of hypnosis for the control and amelioration of chronic pain among adults. They have studied chronic pain syndromes from backache (Tan et al., 2015) to multiple sclerosis (e.g., Jensen et al., 2016), from amputation (cf. Jensen, Smith, Ehde, & Robinson, 2001) to chronic neuropathic pain (Oneal, Patterson, Soltani, Teeley, & Jensen, 2008). They have used control groups ranging from attention and relaxation to cognitive restructuring (e.g., Jensen et al., 2011) to immersive virtual reality (e.g., Patterson, Jensen, Wiechman, & Sharar, 2010; Soltani et al., 2018). Their reviews of hypnosis and chronic pain (e.g., Jensen & Patterson, 2006; Patterson & Jensen, 2003) have been widely influential, as has their advice to clinicians (Jensen, 2011a, 2011b; Jensen & Patterson, 2014; Patterson, 2010). They have explored beneficial side effects of hypnotic pain control instructions that are not explained by reported pain relief (e.g., Jensen et al., 2006).

In fact, they provide excellent support for less hypnotizable participants exhibiting very positive changes in response to hypnosis. Finally, they have worked with patients irrespective of measured hypnotizability (which they call "general hypnotizability"; Jensen & Patterson, 2014). In fact, none of their work suggests a strong relationship between successful treatment and hypnotizability as measured by the Stanford Scales or their derivatives (Hilgard & Hilgard, 1975; Morgan & Hilgard, 1978a, 1978b).

Let's first look at their early review of the literature. Patterson and Jensen (2003) provided a comprehensive and thoughtful "box score" review of the literature on controlled trials of hypnosis and pain in clinical settings, ignoring the experimental studies with student volunteers that were included in the Montgomery et al. (2000) report. They began the review with studies of acute pain, pain in which there is clear tissue damage or a painful medical procedure. (Acute pain is expected to be largely eliminated when the tissue damage is resolved or the procedure ends.) There were 17 studies identified, comprising 12 comparisons of hypnosis and a control condition and 8 comparisons of hypnosis to an alternative psychological treatment. Most of the 17 acute clinical pain studies were based on painful medical interventions (e.g., bone marrow aspirations, burn wound care, or surgery) or on childbirth. In the 12 comparisons in which a hypnotic intervention was compared with a control condition (wait list, standard care, or attention placebo), hypnosis was more effective in 8 comparisons, equivalent in three studies, and one study had mixed results. In the 8 comparisons between hypnosis and alternative psychological treatments (CBT, relaxation training, distraction, and emotional support), hypnosis was superior in 4 and did not differ significantly in the other 4. There was no case in which hypnosis was inferior in regard to pain severity as assessed by patient report. In light of these findings, it seemed appropriate to classify hypnotic analgesia instructions for acute pain as "efficacious and specific."

Then Patterson and Jensen (2003) looked at chronic pain studies, studies without specific tissue damage causally related to the pain. Most of these studies focused on headache pain. In this area, hypnosis was equivalent, but not superior, to other treatments, such as relaxation or autogenic training. The authors identified four RCTs involving nonheadache chronic pain (breast cancer, mixed etiologies, and refractory fibromyalgia). Two of these studies showed hypnosis to be superior to an alternative treatment (group supportive treatment for breast cancer pain and physical therapy for refractory fibromyalgia). Alternatively, two studies involving patients with mixed etiologies (most usually back pain) provided equivocal results.

Several explanations for the difference between the clear effectiveness of hypnosis for acute pain and its less clear effects on chronic pain might be suggested. Distraction will have some effects on chronic pain, but not enough to free one of the effects of pain. Moreover, most patients with chronic pain will already have done what they can to distract themselves. The reduction of anxiety with hypnosis, also central to its success with moderates in the face of acute pain, is also less useful with chronic pain. In acute pain, anything that relieves anxiety helps with pain. In chronic pain, depression and inactivity rather than anxiety are central. One often strives to substitute achievement and consequent sense of mastery and self-efficacy for being pain-free as the goal of treatment. Finally, both the ability to hallucinate the absence or amelioration of pain and positive expectancies may well fail over the long term when confronted with chronic pain.

Since then, Patterson, Jensen and their colleagues have continued to do research on hypnosis and pain. Like Dinges, Orne, and their colleagues, these are careful, sophisticated researchers. They have compared hypnosis in the treatment of chronic pain resulting from a number of disorders, including multiple sclerosis (Jensen et al., 2011), physical disability accompanied by chronic pain (Jensen et al., 2008), ongoing background burn pain (Patterson, Everett, Burns, & Marvin, 1992) and pain from spinal cord injury (Jensen & Barber, 2000). They have often focused on methodological issues related to control groups (Jensen & Patterson, 2005) and have used a variety of control groups ranging from cognitive restructuring to Jacobsen relaxation to immersive virtual reality (e.g., Patterson, Wiechman, Jensen, & Sharar, 2006).

In almost all of their studies, the hypnosis group has done better than the comparison group. However, hypnotizability, as measured by response to standard hypnosis scales, has played no real role in their results. Rather, the overall benefits of the hypnotic context overwhelm any other effects. Though they occasionally acknowledge the need for the measurement of hypnotizability in future research (e.g., Patterson et al., 2010), their essential point is that the benefits of hypnosis are found even among patients whose pain is not reduced much or at all by hypnosis.[18]

An underlying theme of this research (and a good deal of other research on coping with ongoing pain) is that patients need to actively manage their pain and disability. This fact is basic to the cognitive-behavioral treatment of pain (cf. Loeser, Butler, Chapman, & Turk, 2001). If one must cope with injury or illness, actively struggling to create a satisfying life is critically important. The symptoms of pain and illness make such a struggle difficult, especially if one is alone. Thus, for example, few patients with ongoing pain will wake up with a strong desire to do some form of exercise. Further, many might wish to accept some isolation rather than force themselves to undertake social obligations. Remember, with chronic pain it is depression, not anxiety, that is your patient's principle enemy. Managing one's disability by engaging in structured activity, physical and social, produces better results for patients, hurting or not (cf. Henchoz & Kai-Lik So, 2008).

In this vein, Jensen and colleagues (2006) looked at patients who routinely reported satisfaction with treatment, whether their pain was relieved or basically unchanged. For example, increased overall well-being and improved sleep were reported in a population in which exhaustion is widespread and makes everything worse (Jensen & Patterson, 2014). This led Jensen and Patterson to return to those patients and have a research assistant call them and ask a standardized series of questions about the effects of hypnosis (Jensen et al., 2006). Of the original 33 patients, 30 completed the interview. Ten of the 30 patients had experienced substantial pain relief from hypnosis; 20 had not. Nevertheless, most of those who had *not* experienced pain relief continued to use self-hypnosis and reported a variety of positive side effects. For some, pain relief could be found for a limited time during or after self-hypnosis. Additionally, despite ongoing pain, these patients reported increased positive feelings, such as increased relaxation, general well-being, heightened acceptance, and a better attitude.

Given the volume of their work, it is unsurprising that there are places where Patterson and Jenson make invaluable suggestions to the practitioner. For example, at an American Psychological Association (APA) meeting, Patterson (2014) suggested a series of questions that could be asked in most cases.[19] Additionally, their recent books with their many scripts are required reading for anyone interested in the nonpharmacological treatment of chronic pain (Jensen, 2011a, 2011b; Patterson, 2010).

In sum, Patterson and Jensen have taught us that hypnosis is a simple, useful intervention for patients afflicted with chronic pain. That these effects are not limited by hypnotizability and are associated with positive side effects emphasizes the utility of hypnosis.

Hypnosis and Procedural Pain

The use of hypnosis to treat pain caused by medical procedures emerged early in the history of hypnosis. For example, Esdaile (1846/1957) reported 345 major surgeries without pain with hypnosis. Much of the modern work on hypnotic analgesia has focused on such procedural pain. A great deal of that work has been done with both pediatric and adult patients undergoing cancer treatment (e.g., Liossi, White, & Hatira, 2006, 2009; Smith, Barabasz, & Barabasz, 1996; Wall & Womack, 1989; Zeltzer & Lebaron, 1982). Additionally, Montgomery and others have thoughtfully studied hypnotic analgesia for breast surgery procedures (e.g., Montgomery et al., 2007; Montgomery et al., 2010). Further, a number of investigators, along with Patterson and Jensen, have studied hypnosis for pain relief during the treatment of burns (cf. Ewin, 1986).

I have suggested that the utility of hypnosis with clinical pain is dependent in part on pain severity and chronicity. If that is correct, response to acute procedural pain should be closer to pain studied in the laboratory. Specifically, the hallucinatory abilities of highs and the context of hypnosis might both play a part here, at least with less than excruciating pain. In procedural pain, there is time for preparation, and the pain is expected to lessen and end as the procedure ends and any resulting tissue damage heals. When that does not occur, we are almost back in the chronic pain case that Jensen and Patterson (2014) examined. Does hallucinatory ability matter in this set of studies, as it does in the laboratory?

To focus our view, we can look at a recent review by Gary Elkins and his group at Baylor University (e.g., Kendrick et al., 2016). Kendrick and colleagues (2016) critically examined 29 available RCTs on the effectiveness of hypnosis for acute pain caused by a medical procedure (procedural pain). The reviewers noted procedure type, sample size, pain and other measures employed, results on each measure, hypnotizability (when measured), and hypnotic and control condition details. They also rated methodological adequacy indicators using a modified version of the Jadad score (Jadad et al., 1996).[20]

Unsurprisingly, Jadad scores, and the methodology they indicated, generally improved with date of publication.[21] However, Jadad scores do not include an assessment of the equivalence of experimental or control conditions. Unfortunately, and almost necessarily, control conditions routinely differed from experimental conditions in terms of believability, structure, allegiance of study staff, provision of meaningful patient behavior during the procedure, and so on. As we have seen, these and other such contextual factors are known to contribute to the success of interventions (cf. Wampold & Imel, 2015). So hypnotic procedures were simply favored by the experimental designs. In a purely scientific sense, the evidence is tainted. However, in terms of the clinical effects, the advantage of the hypnosis condition in these studies is very clear. Hypnosis works for many patients. It lessens procedural pain and anxiety among both children and adults.

For example, Liossi and her colleagues studied children with cancer whose treatment involved regular lumbar punctures, venipunctures, and bone marrow aspirations. The pain from ordinary lumbar punctures and bone marrow aspirations is severe enough to create sensitization to the procedure over time. That is, children's responses routinely get worse over time, not better, despite use of local anesthetics. This was reversed with hypnosis. Similar findings emerge in burn debridement, breast surgery, and other cancer treatments among adults.

For the first time in the clinical literature, when measured, hypnotic ability sometimes seems to correlate with outcome. Specifically, in the Liossi and Hatira (2003) study of children enduring lumbar puncture, 80 children were randomized to one of four groups, all of whom received local anesthesia prior to the procedure. There were two hypnosis groups, each with 20 children. One of the hypnotic conditions used standard direct hypnosis, the other employed Ericksonian-type indirect suggestion similar to that found in Hammond (1990). Hypnotizability was measured by the Stanford Hypnotic Clinical Scale for Children, a good measurement tool (SHCS:C; Morgan & Hilgard, 1978b). There were strong correlations in the standard hypnosis condition between hypnotizability and decreases in self-reported pain ($r = -.81$, $p < .01$), anxiety ($r = -.81$, $p < .01$), and observed behavioral distress ($r = -.67$, $p < .01$). Similar correlations were found in the indirect condition between hypnotizability and decreases in self-reported pain ($r = -.82$, $p < .01$), anxiety ($r = -.85$, $p < .01$), and observed behavioral distress ($r = -.80$, $p < .01$). The problem is that these are very strong correlations and are methodologically problematic. There is no statement about blinding in the study. It seems likely to me that Liossi did all the hypnosis. So some of the strongest evidence we have for the role of hypnotizability may well reflect problems in the independence of outcome measurement and SHCS:C scores.

There were similar findings in the Liossi and Hatira (1999) study of the treatment of children receiving bone marrow aspirations. Here the conditions were response to hypnosis plus TAU (lidocaine injection) versus a cognitive-behavioral intervention plus lidocaine versus lidocaine alone.[22] Hypnotized participants did better than TAU across all measures, with the cognitive-behavioral intervention scoring midway between the hypnosis and lidocaine alone. Again, a nonblind hypnotist seems to have done the hypnosis in both studies, making artifact impossible to dismiss.

In sum, for procedural pain, the hypnotic context is primarily responsible for its positive effects. If we leave out the Liossi studies, the positive effects we have seen are unrelated to standard measures of hypnotizability. In a meta-analysis, Montgomery, Schnur, and David (2011) noted that when hypnotizability is measured in the clinic, relatively weak effects are seen and are not worth the cost and effort. Despite arguments for hypnotizability assessment in the clinic (e.g., Lynn, Boycheva, & Barnes, 2008), a consensus for avoiding such measurement, outside purely research settings, seems to be emerging.

What Is Missing in RCTs That Is Very Important to Us

RCTs are our gold standard in academia. However, in attempts to measure the effectiveness of a technique, they simplify the clinical situation. In doing so, some factors are controlled or ignored. One of these factors is the clinician and the kind of patients with whom he or she works well. Another factor is differences among patients that are relevant to our treatments. Let me make some minor suggestions about each.

We all know that therapists differ in effectiveness. However, the vast majority of people who enter our field are basically good with people. We are good listeners and/or convincing talkers. Hypnosis is like any other aspect of what we are doing. As I noted earlier, it is possible to train graduate students to induce hypnosis in a couple of hours and then let them induce all the basic hypnotic phenomena (e.g., temporary amnesia, hallucinations, pain control). However, being able to use your knife at dinner is very different from being a surgeon. Using hypnosis clinically requires listening to and watching those recognized as good clinicians at workshops. For example, I learned a great deal about hypnosis from the late Martin Orne and Dabney Ewin at conferences and from their publications (Ewin, 2009; Ewin & Eimer, 2006; Orne, 1959, 1970). In his 2009 book, Ewin reflects on over 50 years of practice. Ewin is now over 90 and retired, but there will be others in his place. There is no substitute for some time spent as an apprentice. Go to the workshops of the Society for Clinical and Experimental Hypnosis and those of the American Society of Clinical Hypnosis. The theoretical rationales of presenters may have more holes than iconic Swiss cheese, but watch and listen to what they do! Copy it where it fits and make it your own.

Second, there are many differences among patients. For example, some people have a high tolerance for pain. Others do not. Which is which can be determined with a simple work sample. For example, one can use hand immersion in 0–2° C (approximately 32–35°F) water and ask for responses about pain severity.[23] That can tell you a good deal about whether some form of central sensitization has occurred (cf. Woolf & Chong, 1993). The results of such a work sample may modify one's approach to some degree.[24]

HYPNOSIS: A MINI-MANUAL

Many standardized hypnotic inductions and suggestions are available (cf. Hammond, 1990; Lynn et al., 1996). In the following version, I have included an induction mostly based on the SHCS (Hilgard & Hilgard, 1975) and then followed it with some specific suggestions.[25]

Discussing and Modeling Hypnosis as Anticipatory Socialization for Hypnotic Treatment

Clinicians often tend to be quite concerned about inducing hypnosis; experimentalists rarely have such concerns. In the experimental context, if research participants fail to respond to hypnotic suggestions and do not experience anything they would define as "being hypnotized" or being in a "trance," nothing is lost. Each participant has contributed useful data. On the other hand, in a clinical setting, there is a good deal at stake when inducing hypnosis for the first time. Failure wastes two precious commodities—time and hope. One invests time in explaining how hypnosis works and how it may help the patient in a variety of ways. If nothing happens, one must backtrack and convince the patient that other techniques will work. Second, patients often come to treatment because internal forces are getting them to react in embarrassing, uncomfortable, and/or ineffective ways in important situations and with important others in their lives. If, in the presence of the therapist, they are unable to respond to hypnosis, the credibility of treatment is threatened, and patients again find themselves blocked by inimical internal forces with which the therapist cannot help. It is preferable for each patient to experience something that both the patient and the therapist can define as hypnosis.

To avoid "failure" at hypnosis, the clinician should inquire about previous hypnotic experiences and discuss any myths about hypnosis that may interfere with the patient's response or expectancies (e.g., that during hypnosis, people entirely lose touch with their surroundings and/or control of their behavior; that deep trance is required for a positive effect). In my own practice, if patients seem skeptical or I simply feel it will be helpful, I will ask patients whether they have seen someone in a hypnotic state from close up. Answered in the negative, I give myself audible suggestions and put myself into a light self-hypnotic state, closing my eyes, then counting and having my hands move together in ways similar to those I will teach during the patients' hypnosis. This models hypnotic behavior for the patient and lowers patient embarrassment and other interfering thoughts and emotions. I then introduce and induce hypnosis using a relatively standard hypnotic induction, a modified version of that created by Hilgard and Hilgard (1975). A model of this follows.

Introductory Remarks

"In just a moment, we will begin hypnosis. Remember that, while you will be hypnotized, you will at all times be able to hear me. You may be less or more aware of your surroundings than you are now. It doesn't matter. What does matter is that you listen to my voice, pay attention to my voice. Then, just let happen whatever you find is happening, even if it isn't what you expect. Remember that at all times, you will stay in complete control, no matter how deeply hypnotized you become. You will always be able to talk aloud, if quietly, without it disturbing your concentration. If at any time you wish to ignore a suggestion, you can say "no" aloud and the suggestion will have no effect. Similarly, if, for whatever reason is sufficient to you, you wish to come out of trance, you simply say "three, two, one, out," and you come out relaxed and alert, feeling fine. If you wish to come out, give yourself these instructions *aloud,* so that you can't get confused by merely thinking about coming out of trance and wondering what effect that will have. In this state, we aren't much concerned with what you are thinking, but we are very concerned about what instructions you are getting. Giving instructions aloud allows you to clearly tell the difference between thoughts and instructions. Later, I will be giving you instructions that allow you to learn to put yourself into hypnosis. When you do that, you always give yourself instructions aloud." [*I turn on an audio recorder at this point, so that the patient also may listen to the instructions at home.*]

"Now, please close your eyes [*at this point I close my eyes, modeling the behavior I want*] and listen carefully to what I say. Let yourself relax as best you can in your chair. Let the chair hold all of your weight, so all your muscles can relax. As you continue to listen to my voice, you may feel more or less wide awake than you do now. But no matter how deeply involved in hypnosis you become, you will at all times be able to hear me. You may be less or more aware of your surroundings than you are now. It doesn't matter. What does matter is that you listen to my voice, pay attention to my voice. You will always be able to hear me and to respond to suggestions that are good for you. Just let happen whatever you find is happening, even if it isn't what you expect."

Hypnotic Induction

[*Speaking more slowly.*] "Now focus on your right arm and hand and let all the muscles relax. Let the muscles in the upper arm become limp. The muscles around the right elbow and forearm becoming loose and comfortable. And all the muscles in the hand letting go, letting go. Completely letting go. [*Pause.*] Now feel into the left arm and hand. Let all the muscles in the upper arm become warm and heavy, soothed and comfortable. And let a feeling of deep relaxation flow down into the elbow, forearm, and wrist. Now the left hand and fingers are entirely relaxing as you move into a quiet, easy state of mind. [*Pause.*]

"You may find that the mind relaxes along with the body. It becomes possible to put all worries aside. Do that now. Let go of all concerns for now and just let your body and mind relax. Allow yourself to become more and more comfortable as you continue to listen to my voice. Just keep your thoughts on what I am saying. . . .[26]

more and more relaxed, perhaps even drowsy, but at no time will you have any trouble hearing me.

"*Blue mountain lake.*[27] Just some key words for entering hypnosis, a pleasant image to delineate this state of mind from other states. I'm going to count from one to twenty, and as I count, you go ever deeper into this quiet relaxed state of mind. You will be able to do all sorts of things that I suggest, things that will be interesting and acceptable to you. You will be able to do them without breaking the pattern of complete relaxation that is gradually coming over you. And you can move around to make yourself comfortable at any time without it disturbing you or breaking the pattern of relaxation. One . . . two . . . three. Let your legs relax now. Start with the right leg. Feel the muscles in the right thigh relaxing, easing and quieting, let the muscles around the knee and foreleg ease and relax as well. Now feel the muscles in the foot become warm and heavy, warm and heavy. [*Pause.*] Now feel that relaxation flowing through your left leg. The thigh . . . knee . . . foreleg . . . foot . . . toes, all the muscles relaxing and easing as you become calm and quiet, become eased and comfortable.

"Four . . . five . . . six. Now the trunk of your body, your shoulders and chest, all the muscles in your abdomen loosening and letting go. Letting go, letting go, fully letting go.

"Now the back. Imagine yourself breathing through every pore in your body, breathing in a million points of healing light with each breath. With each breath in, relaxation, ease, and health flow into your body. Focus for a moment on the muscles in your back. Imagine them opening to greet the air as it flows through your back directly into your lungs. And as you imagine this, the muscles in the back become easier and more relaxed and let you float more deeply into your chair. Now the muscles of your scalp and face and neck relax. The muscles around the jaw letting go. Letting go. [*Observe whether the mouth opens slightly. If so, the person is truly relaxing, as they are violating a norm about appropriate behavior in a public setting. Mouth opening isn't necessary, but it is nice when it happens.*] With each breath out, any remaining tension leaves you. This happens easily and naturally. There is nothing you have to do, no need to try, none to hurry. As you allow yourself to become deeply, fully, and completely relaxed, nothing will disturb you. You can move about to respond to suggestions or simply to make yourself more comfortable without disturbing your concentration or relaxation in the slightest.

"Seven . . . eight . . . nine . . . ten. . . . Now bring both arms straight out in front of you, arms about shoulder height, hands about a foot apart, palms facing inward toward each other. That's good. Hands about a foot apart. Palms facing inward. Now, please imagine a force, pulling your hands together. You can imagine that force any way you like. Perhaps you can imagine rubber bands pulling the hands together. Perhaps the force is like having magnets in each hand pulling your hands together. Whatever way you imagine it, imagine that force as fully as possible.[28] [*Pause.*]

"Now something may begin to happen. You may be able to begin to really feel the force pulling your hands together. Slowly at first, your hands may begin to slowly move together. [*Pause, then as soon as there is any movement, say "Good."*] More and more together. Coming slowly together. And as they come together, you go deeper and deeper into this quiet state of mind, deeper and deeper into hypnosis. Don't try to help. Just let happen whatever you find is happening, as the hands move together, more and more together and you go deeper and quieter. And when your hands touch, that will be our signal that the deeper parts of your mind are open and responsive to suggestions that are good for you. When the hands touch, that will be our signal that you are deep enough to benefit from the suggestions that you will receive. [*Continue to give suggestions that the hands are moving together. When they get close, say:*] Soon the hands will touch, soon they will touch. And when they do, that will be our signal of openness and readiness to receive and respond to suggestions that are good for you. Then the force will release, and the hands can return to their resting position in your lap. The hands will return to their resting position. Soon they will touch. . . . Now, the hands return to their resting position in your lap.[29] Remember, you will always hear me distinctly no matter how hypnotized you are."

[*At this point the formal induction of hypnosis has been completed.*][30]

Stress Reduction: Relaxation and General Healing Suggestions

"Eleven . . . twelve . . . thirteen . . . fourteen. *Magic garden, secret garden.* Please imagine a beautiful garden on a pleasant late spring or summer morning. The garden is a special place of enormous natural beauty. Look at the trees and flowers and grass. Imagine the slight pleasant breeze on your face. And somewhere in the garden you will see a couch or padded bench or especially soft patch of grass, someplace where you can lie down comfortably and absorb the calm and beauty and utter safety of this place. When you see the place to lie down, raise one finger of your right hand to let me know you are there. [*Wait for the signal*]. . . . Good. . . . Now, please go to that place and lie down on it and during the next long minute, spend the rest of that morning resting and relaxing in the utter beauty and safety of that garden. Take a long minute and spend the rest of the morning there relaxing and absorbing the essence of beauty in the garden. [*Allow 60 seconds to pass.*]

"Fifteen . . . sixteen . . . seventeen. Now you get up from the bench or couch and feel yourself drawn inexorably to the very center of the garden, where there is a body of water. Perhaps it is that mountain lake I spoke of, or perhaps it is a pond or a stream or a brook. Whatever it is, you are drawn toward it. And when you reach the water, just raise one finger of your right hand again to signal me that you are there. [*Wait for the signal*] . . . Good. . . . Now please make contact with the water. You might bathe in it, or swim in it, or drink some of it, or simply wash with it. Whichever you do, you will find that the water is a healing balm, a healing fluid that penetrates every solid and permeates every subtle part of your body and mind. The healing balm flows everywhere in you, giving health and strength and ease to every aspect of your mind and body. [*Pause.*]

"All right, now look around this place and find something to bring back with you, perhaps a twig or a leaf or a stone . . . something you can reach out and touch with your mind's eye that will connect you back to this place of peace and healing. And, please, safely tuck it away so that it comes back with you, so in the midst of other things you can always reach out and touch it and connect to this source of inner healing within yourself. And when you have done that, again raise one finger to let me know. [*Wait for the signal.*]. . . . Good.

"Eighteen . . . nineteen. . . . Twenty, Twenty, Twenty, Twenty. Fully relaxed and fully hypnotized. You are able to incorporate into the deeper parts of yourself any suggestions that are good for you. In this state, you will only be open to suggestions that are good for you, and the deeper parts of yourself easily discriminate such suggestions.

General Self-Esteem Suggestions (Paraphrased and Condensed from Hartland, 1971) and Specific Suggestions Designed for the Particular Patient

"You are now very deeply relaxed . . . and everything that I tell you . . . will make a deep and lasting impression on your mind and affect your thoughts . . . your feelings . . . and your actions.

"As a result of this deep relaxation . . . you are going to feel physically stronger and fitter in every way. You will find yourself to be more alert . . . more wide awake . . . more energetic. You will become and you will remain much less easily tired . . . much less easily fatigued . . . much less easily discouraged . . . much less easily depressed.

"Every day . . . your nerves will become stronger and steadier . . . your mind calmer and clearer . . . more composed . . . more peaceful . . . more tranquil. You will become and you will remain much less easily worried . . . much less easily agitated . . . much less easily agitated . . . much less fearful and apprehensive . . . much less easily upset.

"You will become and you will remain able to think more clearly . . . able to concentrate more easily and more fully. As a result . . . you will be able to see things in their true perspective . . . without magnifying them . . . without ever allowing them to get out of proportion.

[*Specific CBT instructions can be inserted here. This can be very useful if there are specific automatic thoughts that need to be corrected. For example, one might say:*] "The ability to see things in their true perspective will make you more effective and happier in a variety of situations. So, you will find that anytime you begin to feel disturbed by thoughts of making a mistake at work, you will remember all the good evaluations you have received and realize there is no evidence that you are in danger of getting fired. Rather, the reverse is true, and you will allow yourself to accept and appreciate the respect you have generated in others around you by your hard work." [*The following returns to Hartland, 1971.*]

"As you become and as you remain able and willing to keep things in their true perspective, you will be emotionally much calmer . . . much more at peace with yourself and with the whole world. So you find yourself developing more and more confidence in yourself and your abilities . . . you find yourself able to do the things each day . . . the things you want to do and the things you ought to do. You do these things without fear of failure . . . without fear of consequences . . . without unnecessary anxiety . . . without uneasiness.

"And everyday . . . you will feel a greater feeling of personal well-being . . . and a greater feeling of personal safety and security . . . than you have felt for a long, long time. Perhaps more than ever before. And all these things will begin to happen . . . exactly as I tell you they will happen . . . more and more rapidly, powerfully and completely each day. And as you continue to listen to these suggestions and do the self-hypnosis I'm about to teach you, that feeling of being at peace with yourself and the universe will grow stronger and stronger until it becomes as much a part of you as the air that you breathe."

Instruction in Self-Hypnosis and Termination of Hypnosis

"In a few minutes I am going to ask you to come back . . . come back awake, alert, with no headache or any other aftereffects . . . bringing back with you all the good things from this place. But before I do, please realize that you can enter this place on your own. You will have a recording of what we have done that you can play. But besides that you will be able to enter hypnosis on your own and give yourself any appropriate instructions that are *good for you*. Remember, in this state, you will only respond to suggestions that are good for you, and the deeper parts of you can clearly recognize them.

"When you want to enter a trance, you will simply shut your eyes and say aloud "*blue mountain lake*." Then say aloud, "*Hands together . . . one . . . two . . . three.*" Then, as you did before, raise your arms to about shoulder height so your hands are about a foot apart. Then, as you did, just imagine a force pulling your hands together and find that they come together until, when they touch, you are ready to receive suggestions that are good for you. When that happens and the hands touch, the force goes away, and your hands go back to a resting position. You then count *aloud* slowly from four to twenty and repeat "*blue mountain lake*." Then you can say "*magic garden*" and go to the garden to have several hours of rest during a long minute. Or you may wander to the pool or brook at the center of the garden and make contact with that healing fluid. Or you may give yourself any suggestion that is good for you. Remember, in self-hypnosis you give yourself all instructions aloud so as to distinguish between what you are suggesting to yourself and what you are merely thinking. You can say the suggestions softly, but you must say them aloud. Finally, you can come out of this state by doing just what we are about to do, count backward from ten to one and then say "out." At "three," not sooner, allow your eyes to open. When you reach "one" you follow it by saying "out," and you bring back all the good things that you have experienced during hypnosis.

"Now, I'm going to ask you to come back, out of hypnosis. I'm going to count from ten to one and then say "out." You will gradually come back. At "three," you allow your eyes to open. When I say "out," you will be fully awake, bringing back with you the good things from the place you have been. All right now, 10 . . . 9 . . . 8 . . . 7 . . . 6, halfway . . . 5 . . . 4 . . . 3, allow your eyes to open . . . 2 . . . 1 . . . OUT. Coming back relaxed and alert, feeling good." [*End the audio recording here.*]

Practicing Self-Hypnosis and the Posthypnotic Discussion

[*If I am working on teaching self-hypnosis, I will immediately segue into it, repeating the instructions I gave before.*] "Let's be sure the self-hypnosis part is set. Simply shut your eyes and say aloud "*blue mountain lake*." Then, as you did today, you raise your arms to about shoulder height so your hands are about a foot apart. Next, say aloud, "*Hands together . . . one . . . two. . . .* " And, as you did today, just imagine a force pulling your hands together and find that they come together until, when they touch, you are ready to receive suggestions that are good for you. Remember, in self-hypnosis you give yourself all instructions aloud so as to distinguish between what you are suggesting to yourself and what you are merely thinking. You can say the suggestions softly, but you must say them aloud. [*Closing my own eyes, I say:*] Do that now. Put yourself in a self-hypnotic state and bring yourself back just as I did with you, except this time the whole thing shouldn't take more than a couple of minutes altogether."

I will then prompt patients to say "*Hands together,*" "*magic garden,*" and so on and repeat specific CBT suggestions, helping them with both the phrasing and the timing of their suggestions.

Whether or not we practice self-hypnosis in this session or another session depends on the patient and the situation. For example, patients with severe depression may not be able to do much more the first week or two than to listen to the positive suggestions on a recording of the hypnotic session.

After the end of the hypnosis session, have patients describe what they experienced. People tend to elaborate hypnotic suggestions in idiosyncratic ways and often need reassurance that they are "doing it right." So, when asked to go to the magic garden, the patient may have gone to a specific garden that he or she has really been to and finds peaceful. That garden may have no water or water at a place other than the center of the garden. The patient needs to know that that is OK. It is important to emphasize that it is the essence of the experience that is important, not the wording of specific suggestions or how a suggestion is related to the person's own life. If the patient has been taught self-hypnosis, I usually communicate that self-hypnosis has been very useful in my life, citing an innocuous incident such as being stuck while writing a paper and using self-hypnosis to get past that point. Next, we schedule times for patients to play the audio for themselves. I will ask them to keep the audio in a safe place, out of the hands of children, thus emphasizing the power of the instructions. If they are to listen to it while going to bed, I explain that it will not do any harm to fall asleep with the recording playing. They will simply come out of hypnosis during sleep. Finally, I will usually suggest that the patient practice self-hypnosis once or twice a day, but for no more than 2–3 minutes at a time and once in 2 or 3 days listen and respond to the recording of our hypnotic session.

CASE EXAMPLE: HYPNOSIS AS PART OF STRESS MANAGEMENT AND CBT TREATMENT FOR SEVERE DEPRESSION

As noted previously, hypnosis is often used to facilitate CBT. The following case, involving long-term therapy, illustrates how the two can be used synergistically. Given the length of treatment, it is impossible to do this session by session. Rather, a broader overview should aid the reader in understanding how hypnosis is used in the framework of a fairly active and directive psychotherapy. Note that this patient, like the vast majority of clinical patients, was not highly hypnotizable. He was a moderate, compliant and willing to "go along," but not someone who was able to hallucinate with hypnosis. It was the context effects of hypnosis, not its ability to alter perception, memory, or cognition, that was important here.

Mr. A suffered from a quite severe and chronic unipolar depression. In the 9 months before seeing me, Mr. A had had a relatively lengthy course of electroconvulsive therapy (ECT) during psychiatric hospitalization with little, if any, lasting effect. He had been treated with a variety of antidepressants and, over the years, with a number of forms of psychotherapy. Like many others with severe, chronic depression, he arrived in my office strongly believing that any new treatment, like those in the past, would do him no good.

There were several major problem areas, but one key area was managing work stress. I briefly discussed cognitive therapy with him. He knew about it, had some minor experience with it, and dismissed it as "Pollyanna bullshit." Insisting on the utility of such techniques when this occurs has been shown to be countertherapeutic (cf. Castonguay, Goldfried, Wiser, Raue, & Hayes, 1996).

The one thing that created some hope was a discussion of hypnosis as a treatment modality. He felt unable to do anything for himself or to fight his depression any longer. Hypnosis, he believed, allowed him to be passive and listen to suggestions that might benefit him; he felt he could do that. Although it is clear that hypnosis does not require physical passivity (Banyai & Hilgard, 1976), the format of a standard relaxation induction supports such culture-wide beliefs.

In response to his view, I induced hypnosis using a format very close to that detailed above, including using the “hands together” suggestion to ensure that the situation was viewed as hypnotic. I gave him recordings of our hypnotic sessions to listen to at home, but at the beginning avoided self-hypnosis and specific CBT instructions. Passively listening to the recordings was in his repertoire. As his mood slightly improved, he felt encouraged that “something was working.” I was then able to address his belief that others at his job could sense how incompetent he was and therefore felt hostile toward him. In this regard, I used hypnosis to introduce (cognitively oriented) suggestions that he was becoming able to separate his interpretations of the events from the events themselves. He began to see that others were more indifferent to him than hostile. This led to an ability to employ rational disputation, using Beck’s (1996) version of the Dysfunctional Thought Record. Disputing the notion that “no one likes me,” he recalled that some people at work had engaged in positive overtures that he had turned away.[31]

Now more able to perceive others’ positive feelings, he began to socialize a little with his coworkers. These changes, and the sense that something was helping, made him willing to try other behavioral and cognitive procedures. We returned to behavioral activation and momentum-building strategies along with cognitive techniques. His Beck Depression Inventory (BDI) scores soon moved from the 40s and 50s to under 20. Throughout this part of our work, he continued to use recorded hypnotic instructions, asking for suggestions that directly addressed problems he was having at work.

Soon thereafter, the company decided that they did not have enough work in his specialty to keep him busy. They therefore assigned him a job negotiating contracts. His history suggested that this was a job for which he was entirely ill suited. Nevertheless, with the help of hypnotically framed suggestions and the cognitive and momentum-building techniques he had learned, he was able to manage the resultant stress and keep basically positive during the next year, with his mood largely reflecting environmental events. When he felt overwhelmed, disheartened, and too paralyzed to use other procedures, he used hypnosis to allow a temporary passivity that soon led to more active, and actually quite heroic, coping. Note that we would discuss the specific suggestions that he thought might be helpful. Thus for months he was able to do a job for which he was unsuited by training and temperament. His courage, hypnosis, and cognitive therapy all helped him continue to manage the stress and strive hard to do a good job during this period.

Ultimately, the misfit between my patient and the work available in his company resulted in his being laid off. Understandably, he became quite depressed (BDI score over 40). Again, he used hypnosis to feel slightly better and then was able to use cognitive and behavioral techniques to move himself past feelings of worthlessness, hopelessness, and futility. Within a month of losing his job, his BDI scores had returned to well within the normal range. During this period, he was actively searching for a job and prepared for and took a difficult professional examination that significantly enhanced his resume. Eventually, he found a permanent job in a difficult work environment. Despite the difficulties, he is doing reasonably well, and a promotion to vice president is quite possible. He continues to use hypnosis along with the cognitive and behavioral techniques he has learned. Hypnosis is seen as a safety net, as well as a current aid, something that is always there should he need it.

In this case, I used a variety of techniques, including direct advice (cf. Karlin, 2002; Woolfolk, 1998), cognitive and behavioral procedures, and others. But hypnosis, seemingly, made them all possible. Hypnosis was useful because of culture-wide beliefs about it, not because of this patient's specific ability to respond to hypnotic suggestions. Deep hypnosis was not required, simply the sense that something different was going on. Instruction in the role of hypnotic subject was not required; it is well known enough in our culture to seem a natural response to hypnotic induction. Cognitive notions framed as hypnotic suggestions were never subjected to the skeptical appraisal of someone with whom many therapies had failed. Rather, he could listen to and absorb them with faith in the notion that the "deeper parts of the mind" would be affected and that, at least at the beginning, only his passive attention was required for efficacy.

SUMMARY AND RECOMMENDATIONS

We are close to the end of this chapter. Let me suggest that we step back from hypnosis for a moment in order to put what we have learned from hypnosis in perspective. One of the most intriguing and frustrating findings in clinical psychology is the victory of the Dodo Bird verdict (Luborsky, 1995). It seems to be true that "Everyone has won and all shall have prizes." As a variety of contextualists have noted, different treatments whose differences are the products of entirely different theories are roughly similar in effectiveness across a variety of clinical syndromes (cf. Wampold & Imel, 2015). There are some possible exceptions (e.g., exposure treatments for specific phobias). However, in general, treatments presented and treated as legitimate psychotherapies have effect sizes somewhere near 0.6 or 0.7 when compared with no treatment. And hypnosis seems to add another effect size of about 0.5 to that. How can we understand that?

Imagine, if you would, that you are the only survivor of a small plane crash in the desert. Finding yourself alone, you set off to find food, water, and civilization. Twenty-four hours later, you are almost ready to lie down and die. Then, as you reach the top of one more barren hill, you see a large sign reading: DESERT GUIDE SERVICE. Immediately, any thought of lying down to die leaves your mind. You find hope surging through you. And it brings energy and resolution that you had thought exhausted.

When you get to the guide service a couple of hours later, you are handed a description of several types of guides. The guides who are available this week include a cognitive-behavior therapy guide, a hypnotic guide, a psychoanalytic guide, a client-centered guide, and a gestaltist. In each case, they start by asking for your story.

The CBT guide listens, then says that she wants to teach you some specific skills about how to survive in the desert. She also wants you to do daily aerobics to get ready for the rigors of your trip back to town. The hypnotic therapist may well do the same things that the CBT guide did, adding trance suggestions for self-confidence, perspective, physical and emotional strength, and endurance and pain relief.

The psychoanalytic guide wants to help you understand why you often choose airplanes that have a wing falling off. She may also ask, "Can you remember someone close to you making similar choices when you were younger?" Of course, therapists using hypnosis may do some of the same things our psychoanalytic therapist would do but use the context of hypnosis to aid them.

The client-centered guide slows you down and helps you become comfortable. Then she asks you for a very detailed version of your flight, especially right before and after the crash. She may then repeat what you said: "I saw a town with a railroad station just before the plane started to come apart." The client-centered therapist then wonders aloud whether you know the direction of that town. Somewhat confused, you point to your left, but the therapist fails to respond. Then, you point to your right. She says "ummm-hmmm."

Finally, you find the Gestalt guides napping in a comfortable chair. Waking them, you tell your story one more time. One Gestalt guide asks you why you left town in the first place. You reply, "I was bored." The guide then asks "Why are you rushing back to a life that bores you? Is it possible that here and now is the time and place toward which you were traveling?" The other Gestalt guide might ask: "Did you have a chance to spend an hour or so last night looking up at the stars in the desert sky?"

Each of these approaches can be helpful. *But note, the largest change occurred when the traveler saw the sign. That was when hope and energy returned.* That effect was probably magnified when the traveler walked in and saw that he had found credentialed desert guides with diplomas to prove it. Remember, outcome research has demonstrated that all the types of therapy have about the same record of helping people. However, we have also learned that individual guides may differ in competence.

Could it be that simple? Might it be that the heavy lifting in each case was done by the large neon sign and the credentials on the wall? Remember, all credentialed guides have been trained to create a relationship allowing them to work with differing travelers. Further, all these brand-name guides have been trained to provide an explanation of the problem at hand. The resulting narratives increase hope, energy, and self-confidence. Again, once hope and energy are renewed, the different approaches seemingly make little difference (cf. Frank, 1961; Wampold & Imel, 2015). The specific techniques may well add something to the basically beneficent procedure, with effect sizes on the order of 0.1 or 0.2. Leaving the metaphor, much of the rest might well be differences in therapists' expertise and ability to form relationships with patients.

This analysis suggests that much of the literature on empirically supported therapies is largely or entirely a function of inadequate and systematically biased experimental design. As Kirsch (2010) showed in regard to the large majority of effects of selective serotonin reuptake inhibitors (SSRIs) and Wampold and his colleagues have shown in regard to psychotherapy, active drugs and therapies have been compared to conditions that can be called pseudo-placebos or worse. In this vein, active conditions benefit from effects like allegiance, greater structure, beliefs, outcome expectancies, effort, plausibility effects, and similar subtle differences among the ways that participants in these non-blind trials are treated. Because of this bias, active psychotherapy groups should show better results than the control condition. Either they do or the study will (almost always) remain unpublished (cf., Kirsch, 2010).

As we have seen, hypnosis has two sets of specific effects in the laboratory, those related to the ability to hallucinate in response to simple verbal suggestion and those related to the setting created by and beliefs about hypnosis. In clinical settings, the large majority of patients are moderately hypnotizable; they are compliant and able to experience minor hypnotic phenomena but are incapable of real hallucinations. Work to date strongly suggests that high hypnotizability, the hallucinatory element available for highly hypnotizable participants, plays little part in real-world treatment. Instead, treatment depends on the ability to use beliefs about hypnosis, many of them both strongly rooted in the culture and factually incorrect, to create situations that are good for the patient. This is true whether stress management, pain, or the treatment of psychopathology is the therapist's concern. For example, patients with anxiety who have tried and failed to relax using breathing or progressive relaxation-oriented procedures will often relax during hypnosis because they don't have to *try* to relax. Instead, they naturally relax as they occupy the social role of hypnotized subject. Similarly, patients with severe depression may listen passively to instructions and expect large benefits despite their inactivity. Finally, wrapped in the mantle of hypnosis, positive self-statements and other CBT techniques may be perceived as stronger and have more credibility, a greater likelihood of being utilized, and greater effectiveness. Hypnosis is a wonderfully flexible procedure, utilizable in directive behaviorally oriented therapy and in evocative treatment. It is clearly effective with pain and stress. Overall, it is a powerful tool.

If this flexible tool is to be used well, it requires a good clinical hypnotist. Whereas hypnotic phenomena in the laboratory may be produced by simply reading a script or playing a recording, helping the patient experience the magic of increased self-control, increased access to the "powers of the mind," involves one's own belief in the suggestions made during hypnosis. The greatest clinical benefits can be gathered by conveying the notion that hypnosis and an ongoing self-management stance can help in major ways. Further, in cases of chronic pain, ongoing use of self-hypnosis may sometimes be a necessary part of successful treatment.

When I first learned to do hypnosis during an internship with Arnold Lazarus in 1971, one often had to invent suggestions. When I gave pain control instructions, they were largely my own creations. Certainly, by the late 1970s, I knew there was a difference between pain and suffering and that I should suggest decreases in each. However, I assumed that what I saw in the laboratory would be reflected in the clinic. I had no idea of the power of the technique for less hypnotizable patients that the research now suggests. Additionally, we now have a proliferation of hypnotic scripts almost unimaginable then. In general, the utility of hypnosis seems to steadily increase.

Hypnosis does require caveats. Naïve views about the effects of hypnosis on memory have led to its use in creating inaccurate memories of childhood sexual abuse, memories that have driven families apart. The inappropriate use of hypnosis has also resulted in a small epidemic of a quite malignant, iatrogenic form of dissociative identity disorder (c.f., Karlin & Orne, 1996). In forensic settings, hypnotic age regression has led to detailed, convincing, inaccurate testimony resulting in major and minor miscarriages of justice (c.f., Karlin, 1997). Thus the use of hypnosis to "refresh recollection" in both clinical and forensic settings should be approached very cautiously, if at all.

The other caveat has to do with the treatment of pain with psychotic patients in remission. If a patient has a history of hospitalization for schizophrenia, be cautious about taking away pain that has no verified pathophysiological origin. On very rare occasions such patients may be using the pain to focus and hold themselves together (cf. Karlin, 2018)

Lastly, a joint consideration of the hypnosis literature and the literature on the effectiveness of CBT yields some interesting directions for thought. Attempts to transport CBT into field settings have met with mixed success. Overall, obsessive–compulsive disorder and phobias have yielded the best effectiveness and transportability trials for CBT. These are disorders treated with exposure, and exposure works. Far less success has been seen with other disorders, with even the outcome of CBT for depression giving one reason to pause (cf. Elkin et al., 1989).

Similarly, as just noted, control of pain and stress in response to hypnotic suggestions does not depend on measurable hypnotizability for its effects. Cultural beliefs about the ritual of hypnosis as a credible means of reducing perceived pain and otherwise ameliorating stress make variables such as beliefs about the power of hypnosis and the clinician's diplomas do the heavy lifting. These add to the change in expectancies and attention concurrent with the use of any technique made credible by expert administration. Thus, for hypnotic analgesia and stress reduction and exposure-based CBT, we not only have RCTs, but we also have believable mechanisms that underlie the success of the procedure across settings. I have noted above my concerns about the evidence base for "evidence-based" psychotherapy. Going forward, perhaps we should require elucidation of such mechanisms before claiming that a treatment is evidence-based.

Alternatively, we might focus on the contextual elements that make most psychotherapies generally beneficent and on avoiding tempting collusions, such as blaming easy targets outside the therapy dyad, that may do harm. Moreover, an understanding of the power of context and of people's beliefs about our techniques (as illustrated by hypnosis) and a recognition of the logic of emotions, the realities of interpersonal problems, and the utility of common sense might be seen as directions for future progress.

ACKNOWLEDGMENT

This chapter could not have been completed without the able assistance of Sarah Coffin-Karlin.

NOTES

1. The distinctions between highs, moderates, and lows can be made in a variety of ways. The simplest is to define hypnotizability levels in terms of response to a standardized hypnosis scale, such as the Stanford Hypnotic Susceptibility Scales: Forms A, B, and C (SHSS:A, B & C; Weitzenhoffer & Hilgard, 1959, 1962) and related scales. These scales each comprise 12 standardized suggestions; each suggestion is scored pass or fail. Most frequently, high, moderate, and low hypnotizability are defined in terms of number of suggestions passed. For example, with the SHSS:A, low hypnotizability is usually defined as passing 0–4 of the suggestions, moderate hypnotizability passing 5–8 of the suggestions, and high hypnotizability as passing 9–12. A less frequently used method of distinguishing highs, moderates, and lows is to make diagnostic ratings of "plateau hypnotizability" over a number of sessions, using clinical techniques for relaxing participants and maximizing their responses (cf. Orne & O'Connell, 1967). Such ratings indicate five levels of response: (1) no response, (2) response to suggestions for unobstructed movement, (3) objective and subjective inability to perform a movement when challenged, (4) responses to suggestions to hallucinate, and (5) reversible amnesia and true posthypnotic response. Each level assumes success at the previous level. The distinction of most importance in this chapter involves the ability to respond to cognitive and hallucinatory suggestions using both behavioral and subjective criteria. Highs routinely respond positively to such suggestions; moderates and lows do not.

2. The usual rough estimate is that 15% are highs, 15% lows, and the remaining 70% moderates. There is evidence that a proportion of the lows are not willing to be fully tested (Karlin, Hill, & Messer, 2008) with standard scales. Such "unwilling" participants are rare in clinical settings.

3. For an opposing view about the modifiability of hypnotizability, see the work of Lynn and his colleagues (e.g., Gfeller et al., 1987) and the work of Spanos and his colleagues (e.g., Gorassini et al., 1991). It is clearly possible to alter responses to hypnotic scales so that relatively unhypnotizable participants can soon learn to pass far more items on standardized scales than they did before. The question is whether such individuals also respond in other ways similar to highly hypnotizable participants. For example, do they show brain function changes during reported hypnotic hallucinations similar to those seen among untrained highly hypnotizable participants? Given the extraordinary stability of hypnotizability over time, I doubt it. I think the situation akin to a preparatory course for an IQ test. Certainly, test performance can be improved with appropriate preparation, but it is not clear that one would still be measuring "G" when well-prepared participants are measured.

4. In suggesting a positive hallucination, one suggests the presence of stimuli that are not, in fact, present. For example, during the age-regression suggestion on the Stanford C scale, one suggests that a voice will be heard coming from a school loudspeaker. Alternatively, for negative hallucinations, one can ask participants to hallucinate the absence of stimuli that are present (e.g., seeing two colored boxes on a large, easily visible poster board that shows three colored boxes.) Both analgesia and amnesia suggestions are viewed as suggestions for negative hallucinations in this context.

5. Although a good deal of speculation and some research suggested that hypnosis was a right-hemisphere phenomenon, that view was disconfirmed. Rather, highly hypnotizable participants show more lateralized activity during both right- and left-hemisphere–related tasks (Labriola, Karlin, & Goldstein, 1984; McCloud-Morgan, 1984).

6. Highs, the 0.3–30% of the population who can clearly hallucinate in response to verbal suggestion, may or may not find that ability helpful in dealing with chronic pain or other severe problems. Hallucinations have their limits, and laboratory pains cannot equal clinical pain or agony in severity or chronicity.

7. Unfortunately, in an altogether different arena, during the last 40 years hypnosis has been used to "refresh recollection" in forensic and clinical settings. Done by police hypnotists, hypnosis resulted in eyewitness testimony that was certain and detailed. Juries are convinced by such eyewitness testimony. However, these memories were often based on fantasy, confabulation, and postevent information. They were often wrong both in detail and essence. For example, witnesses remembered faces that they physically could not have seen. Clinically, when used naively, hypnosis and similar procedures led to false memories of childhood sexual abuse and satanic ritual abuse. Both set the stage for a malignant, iatrogenic form of multiple personality disorder (unfortunately now called dissociative identity disorder; cf. McNally, 2003).

8. In a recent meta-analysis of the treatment of anxiety with hypnosis by Milling's group (Valentine, Milling, Clark, & Moriarty, 2019), only 3 of the 17 trials cited were published since 2000. Of these 3, 2 concerned anxiety occasioned by coronary bypass surgery, and the 3rd involved dental anxiety. However, it is noteworthy that over all 17 trials, the average weighted effect size was 0.79, and the seven longest follow-ups had a mean weighted effect size of 0.99.

9. In a study on willingness to be hypnotized, about half of the lows seemingly did not participate fully in hypnotizability testing (Karlin et al., 2008). But that was an experimental group comprising coerced volunteers who were fulfilling experimental requirements as part of an introductory psychology course. With rare exceptions, in the clinical situation, people are quite willing to be hypnotized.

10. Where D is effect size (d) weighted by sample size.

11. It is also similar to the effects of most psychotherapies when compared with no-treatment, wait-list controls, or even pseudo-placebo control conditions.

12. At several points in this discussion, I list a number of the contextual effects of hypnosis. These lists are meant to be illustrative, not comprehensive. I don't think we know enough to make a comprehensive list.

13. Many of the studies reviewed by Kirsch et al. (1995) were done in the 1970s and early 1980s. Thus methodological concerns are probable and have been expressed by one of Kirsch's colleagues (Schoenberger, 2000), who called for further RCTs to demonstrate the effectiveness of hypnosis as an adjunct to CBT. However, recent studies (e.g., Bryant et al., 2003) have basically supported the conclusions of Kirsch et al. (1995).

14. Flammer and Bongartz (2003) claim that their meta-analysis is very conservative and may well underestimate effect size because they included dependent variables that could not reasonably be expected to change with hypnosis. They are probably right, especially in regard to pain-related studies. Also, they excluded follow-up effect sizes (available from 22 of the 57 randomized studies). Interestingly, these effect sizes are often larger than those found immediately posttreatment.

15. After I spoke with their authors, two weight-loss studies with enormous effect sizes should almost certainly have been excluded. Their exclusion would leave the average effect size for nonrandomized trials using pre–post scores a little under 2.0, still a very large effect size.

16. Many areas of research start with small sample studies showing quite major differences between treatments. For example, we saw that pattern in CBT for phobias, OCD, and bulimia and in cognitive therapy and behavioral activation for depression. No one who has gone through the recent debates about the causes of specific effect sizes expects replication of these size differences among treatments. However, as in other successful treatments, further research provided almost unanimous positive support for hypnotherapy for IBS and other functional gastrointestinal disorders. However, the study of Dobbin et al. (2013) creates an important caveat. One must always ask, "Who designed the placebo condition?" If, as is most often the case, it is the authors, there well may be a basic design flaw in the study. A very recent multicenter trial with this kind of design flaw is discussed below.

17. In some ways, this was an attempt to scale up a successful treatment, as well as an efficacy trial. The former, important goal seems to have been met.

18. Ignoring hypnotizability measurement in the clinical context has been by far the most common solution to the problems with such measurement. In contrast to the strong effects found in the experimental literature, the view that hypnotizability measurement has little or no relationship with clinical outcome has received a good deal of support. The view that measuring hypnotizability in the clinic is not worth the trouble has become increasingly widespread (cf. Montgomery et al., 2011).

19. There are over a dozen questions that one can directly draw from Patterson (2014). These include: Where does the patient hurt? What has been the course of pain in the last 6 months? What makes the pain worse? Better? What is the time pattern of pain (daily, weekly, monthly)? Is the pain neuropathic or musculoskeletal? What is the cause of pain? What treatments has the patient tried? Is the patient's orientation more or less biomedical or self-management? Does the patient get regular exercise? What is the patient's spouse's response? How does the patient cope? What, if any, pacing difficulties are there? Has there been/will there be compensation for pain? What are the reinforcement contingencies, motivational factors, coping strategies?

20. The Jadad score has five components: 1 point each is given for (1) randomization, (2) the appropriateness of the randomization technique, (3) reporting an explanation of why participants withdrew or dropped out, (4) whether the staff and assessors were blind to treatment assignment, and (5) whether the person providing the treatment was blind. A point could be subtracted if the randomization technique was not appropriate. Blinding of the hypnotist and/or the staff in these studies was often seen as quite difficult, and so these scores used only the first four criteria (Kendrick et al., 2016). Scores ranged from 0–4, with a mean of 2.33.

21. Of the 29 studies reviewed by Kendrick et al. (2016), 4 were published in the 1980s, 11 were published in the 1990s, 13 between 2000 and 2009, and 1 more recently.

22. Having bone marrow sucked into the needle is momentarily excruciating. Lidocaine is no help with that.

23. Specific directions may be obtained from the author at rakarlin@psych.rutgers.edu.

24. Approaches using a mindfulness orientation and teaching the patient to carefully observe the pain may be useful in cases in which central sensitization has occurred (Kabat-Zinn, 2013; Shapiro & Carlson, 2009).

25. The suggestions include a large section condensed from Hartland's ego strengthening technique; some paraphrased versions of suggestions or techniques taught by Martin Orne, Arnold Lazarus, or Herbert Spiegel; those I ran across elsewhere and liked; as well as occasional phrasing of my own. When I am aware of the source of the paraphrased suggestion, I have noted it. But I have been doing this so long that it is certain that some of what I consider original was, in fact, a gift from others. I ask their pardon for not citing them.

26. The symbol . . . indicates a brief pause.

27. My arbitrarily chosen signal phrase, "blue mountain lake," is used to delineate hypnotic and nonhypnotic periods. Although I use this phrase in heterohypnosis, I consider the phrase most useful in the context of self-hypnosis. Using it allows the patient to specify that the instructions that follow are hypnotic.

28. There is a trade-off here that is very important to understand. Although one never wants to give a suggestion to which the patient does not respond, at the same time suggestions should ask that something unusual occur so that the patient will attribute the unusual event to entering hypnotic trance. Thus I tend to avoid inductions involving naturally occurring phenomena, events that the patient can reasonably expect to occur, trance or no trance. For example, I would not induce trance by having someone gaze fixedly at a point above eye level. The eye fatigue and eye closure that routinely follow are too easily (and correctly) attributed to simple muscle fatigue. Thus I routinely use a "hands being pulled together" suggestion to induce the experience of trance. There are a number of ways of minimizing the (quite minor) risks of having a patient not experience seemingly involuntary movement of the hands, but they are largely beyond the scope of the present discussion. However, let me mention one way to avoid the problem. One can give the hands-together suggestion without formally inducing hypnosis. Rather, the suggestion can be presented as a prehypnotic "test." One says, "Just let me see something. Allow your eyes to shut and hold both hands straight out in front of you," and so on. Because you can't fail at something you never tried, if the suggestion does not result in a positive response, the patient has not failed to respond to hypnosis. One then proceeds cautiously to determine the cause of the problem. However, such lack of response in a treatment setting is very rare in my experience. When a positive response occurs, as it does with the overwhelming majority of patients, one can easily segue from the nonhypnotic test into hypnosis with a patient who has already experienced an "involuntary" response. In my view, it is hard to overemphasize the importance of patients' perceiving themselves as hypnotized. Here is where you gain or lose the contextual effects of hypnosis for the large majority of patients.

29. When asking for a movement at any point in the procedure, repeat the last sentence or last few words of the sentence as needed, pausing between repetitions, until the movement occurs or it is clear that it isn't going to occur. Incidentally, at this point, we are leaving the Stanford Hypnotic Clinical Scale (Hilgard & Hilgard, 1975). What follows for a while is my phrasing.

30. Similar techniques, such as a hand levitation suggestion, are used to deepen hypnosis, if necessary. For example, "Your arm feels lighter and lighter, as if it were being pulled upward by a buoyant balloon. Feel your arm become lighter and lighter. Pulled upward. And as your hand and arm rise upward, you go deeper and deeper, deeper and deeper, until, when you have reached the proper depth for you in the here and now, your arm will return to its resting position in your lap. Its resting position in your lap." Incidentally, this is the type of suggestion advocated by Orne and O'Connell (1967) to maximize hypnotic responsiveness.

31. From my point of view, there were two critical elements here. First, my patient had "tried" cognitive therapy and "it didn't work." So he was unwilling to try to actively dispute his strongly held irrational cognitions. Moreover, he felt incapable of actively disputing them, even had he wanted to do so. Unlike most approaches, hypnosis allowed him to be passive and merely listen a number of times to the suggestion that he was becoming able to separate events and their interpretation. He could not repeatedly listen to that suggestion without understanding that his interpretations could be and should be separated from the events he was experiencing. Further, the suggestion said he was more and more capable of making this differentiation. It then became reasonable to ask him about what had occurred during the week and simply note that the behavior of his colleagues seemed more inattentive than hostile. Once hypnosis had planted the seed and discussion had paved the way, he came up with the (just noted) instances in which his colleagues had made positive overtures to him. At that point, he began to see his cognitions as irrational and became willing to dispute them in a standard CBT format enhanced by further hypnotic suggestions about emerging changes.

REFERENCES

Apkarian, A. (2015). *The brain adapting with pain: Contributions of neuroimaging technology to pain mechanisms*. Philadelphia: Wolters Kluwer Health.

Banyai, E., & Hilgard, E. (1976). A comparison of active-alert hypnotic induction with traditional relaxation induction. *Journal of Abnormal Psychology, 85,* 218–224.

Barber, T. (1969). *Hypnosis: A scientific approach*. New York: Van Nostrand Reinhold.

Beck, J. (1996). *Cognitive therapy worksheet packet*. Bala Cnwyd, PA: Beck Institute.

Bernheim, H. (1884). *De la suggestion dans l'état hypnotique et dans l'état de vielle*. Paris: Octave Doin.

Bryant, R., Moulds, M., Guthrie, R., Dang, S., & Nixon, R. (2003). Imaginal exposure alone and imaginal exposure with cognitive restructuring in treatment of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 71,* 706–712.

Castonguay, L., Goldfried, M., Wiser, S., Raue, P., & Hayes, A. (1996). Predicting the effect of cognitive therapy for depression: A study of unique and common factors. *Journal of Consulting and Clinical Psychology, 64,* 497–504.

Chambless, D., & Hollon, S. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology, 66,* 7–18.

Charcot, J., & Richer, P. (1978). *Contributions de l'hypnotisme chez les hysteriques*. In Krauss Reprints, *The origins of psychiatry and psychoanalysis: Pre-Freudian psychology series—France* (Vol. 28). Nendeln/Lichtenstein: Kraus-Thompson. (Original work published 1882)

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

De Pascalis, V., Cacace, I., & Massicolle, F. (2008). Focused analgesia in waking and hypnosis: Effects on pain, memory and somatosensory event-related potentials. *Pain, 134,* 197–208.

Dinges, D., Whitehouse, W., Carota Orne, E., Bloom, P., Carlin, M., Bauer, N., et al. (1997). Self-hypnosis training as an adjunctive treatment in the management of pain associated with sickle cell disease. *International Journal of Clinical and Experimental Hypnosis, 45,* 417–432.

Dobbin, A., Dobbin, J., Ross, S., Graham, C., & Ford, M. (2013). Randomized controlled trial of brief intervention with biofeedback and hypnotherapy in patients with refractory irritable bowel syndrome. *Journal of the Royal College of Physicians of Edinburgh, 43,* 15–23.

Elkin, I., Shea, T., Watkins, J., Imber, S., Sotsky, S., Collins, J., et al. (1989). National Institute of Mental Health treatment of depression collaborative research program: General effectiveness of treatments. *Archives of General Psychiatry, 46,* 971–982.

Elkins, G. (Ed.). (2017). *Handbook of medical and psychological hypnosis: Foundations, applications, and professional issues*. New York: Springer.

Elkins, G., Jensen, M., & Patterson, D. (2007). Hypnotherapy for the management of chronic pain. *International Journal of Clinical and Experimental Hypnosis, 55,* 275–287.

Ellenberger, H. (1970). *The discovery of the unconscious: The history and evolution of dynamic psychiatry*. New York: Basic Books.

Esdaile, J. (1957). *Hypnosis in medicine and surgery*. New York: Julian Press. (Original work published 1846)

Ewin, D. (1986). Emergency room hypnosis for the burned patient. *American Journal of Clinical Hypnosis, 29,* 7–12.

Ewin, D. (2009). *101 things I wish I'd known when I started using hypnosis*. Bethel, CT: Crown House.

Ewin, D., & Eimer, B. (2006). *Ideomotor signals for rapid hypnoanalysis: A how-to manual*. Spingfield, IL: Charles C Thomas.

Flammer, E., & Bongartz, W. (2003). On the efficacy of hypnosis: A meta-analytic study. *Contemporary Hypnosis, 20,* 179–197.

Flik, C., Laan, W., Zuithoff, N., van Rood, V., Smout, A., Weusten, B., et al. (2019). Efficacy of individual and group hypnotherapy in irritable bowel syndrome (IMAGINE): A multicenter, randomized controlled trial. *Lancet. Gastroenterology and Hepatology, 4,* 20–31.

Frank, J. (1961). *Persuasion and healing: A comparative study of psychotherapy*. Baltimore: Johns Hopkins University Press.

Franklin, B., de Bory, G., Lavoisier, A., Bailly, J., Majault, M., Sallin, J., et al. (1784). *Rapport des commissionaires charges par le roy de l'examen du magnetisme animal*. Paris: Bibliotheque Royale. (Reprinted in English in *Foundations of hypnosis: From Mesmer to Freud,* pp. 82–128, by M. Tinterow, Ed., 1970. Springfield, IL: Charles C Thomas).

Gfeller, J., Lynn, S., & Pribble, W. (1987). Enhancing hypnotic susceptibility: Interpersonal and rapport factors. *Journal of Personality and Social Psychology, 52, 586–595.*

Gonsalkorale, W. (2006). Gut-directed hypnotherapy: The Manchester approach for treatment of irritable bowel syndrome. *International Journal of Clinical and Experimental Hypnosis, 54,* 27–50.

Gonsalkorale, W., Miller, V., Afzal, A., & Whorwell, P. (2003). Long-term benefits of hypnovtherapy for irritable bowel syndrome. *Gut, 52,* 1623–1629.

Gorassini, D., Sowerby, D., Creighton, A., & Fry, G. (1991). Hypnotic suggestibility enhancement through brief cognitive skill training. *Journal of Personality and Social Psychology, 61,* 289–297.

Hammond, D. (Ed.). (1990). *Handbook of hypnotic suggestions and metaphors.* New York: Norton.

Hartland, J. (1971). Further observations on the use of "ego-strengthening" techniques. *American Journal of Clinical Hypnosis, 14,* 1–8.

Henchoz, Y., & Kai-Lik So, A. (2008). Exercise and nonspecific low back pain: A literature review. *Joint Bone Spine, 75, 533–539.*

Heutink, M., Post, M., Bongers-Janssen, H., Dijkstra, C., Snoek, G., Spijkerman, D., et al. (2012). The CONECSI trial: Results of a randomized controlled trial of a multidisciplinary cognitive behavioral program for coping with chronic neuropathic pain after spinal cord injury. *Pain, 153,* 120–128.

Hilgard, E. (1965). *Hypnotic susceptibility.* New York: Harcourt Brace Jovanovich.

Hilgard, E., & Hilgard, J. (1975). *Hypnosis in the relief of pain.* Los Altos, CA: Kaufmann.

Horton, J., & Crawford, H. (2004). Neurophysiological and genetic determinants of high hypnotizability. In M. Heap, R. Brown, & D. Oakley (Eds.), *The highly hypnotizable person: Theoretical, experimental and clinical issues* (pp. 133–151). London: Routledge.

Jadad, A., Moore, R., Carroll, D., Jenkinson, C., Reynolds, D., Gavaghan, D., et al. (1996). Assessing the quality of reports of randomized clinical trials: Is blinding necessary? *Controlled Clinical Trials, 17,* 1–12.

Jensen, M. (2011a). *Hypnosis for chronic pain management: Therapist guide.* New York: Oxford University Press.

Jensen, M. (2011b). *Hypnosis for chronic pain management: Workbook.* New York: Oxford University Press.

Jensen, M., & Barber, J. (2000). Hypnotic analgesia of spinal cord injury pain. *Australian Journal of Clinical and Experimental Hypnosis, 28,* 150–168.

Jensen, M., Barber, J., Hanley, M., Engel, J., Romano, J., Cardenas, D., et al. (2008). Long-term outcome of hypnotic-analgesia treatment for chronic pain in persons with disabilities. *International Journal of Clinical and Experimental Hypnosis, 56,* 156–169.

Jensen, M., Ehde, D., Gertz, K., Stoelb, B., Dillworth, T., Hirsh, A., et al. (2011). Effects of self-hypnosis training and cognitive restructuring on daily pain intensity and catastrophizing in individuals with multiple sclerosis and chronic pain. *International Journal of Clinical and Experimental Hypnosis, 59,* 45–63.

Jensen, M., Gianas, A., George, H., Sherlin, L., Kraft, G., & Ehde, D. (2016). Use of neurofeedback to enhance response to hypnotic analgesia in individuals with multiple sclerosis. *International Journal of Clinical and Experimental Hypnosis, 64,* 1–23.

Jensen, M., McArthur, K., Barber, J., Hanley, M., Engel, J., Romano, J., et al. (2006). Satisfaction with, and the beneficial side effects of, hypnotic analgesia. *International Journal of Clinical and Experimental Hypnosis, 54,* 432–447.

Jensen, M., & Patterson, D. (2005). Control conditions in hypnotic analgesia clinical trials: Challenges and recommendations. *International Journal of Clinical and Experimental Hypnosis, 53,* 170–197.

Jensen, M., & Patterson, D. (2006). Hypnotic treatment of chronic pain. *Journal of Behavioral Medicine, 29*(1), 95–124.

Jensen, M., & Patterson, D. (2014). Hypnotic approaches for chronic pain management: Clinical implications of recent research findings. *American Psychologist, 69,* 167–177.

Jensen, M., Smith, D., Ehde, D., & Robinsin, L. (2001). Pain site and the effects of amputation pain: Further clarification of the meaning of mild, moderate, and severe pain. *Pain, 91,* 317–322.

Kabat-Zinn, J. (2013). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain and illness* (rev. ed.). New York: Bantam Books

Karlin, R. (1997). Illusory safeguards: Legitimizing distortion in recall with guidelines for forensic hypnosis: Two case reports. *International Journal of Clinical and Experimental Hypnosis, 45,* 18–40.

Karlin, R. (2002). Advice and consent: Demand characteristics, ritual, and the transmission of practical wisdom in the clinical context. *Prevention and Treatment, 5,* Art. 44.

Karlin, R. (2018). Metaphor, *verstehen,* or neither: A reflection on hypnotic analgesia and active ingredients in psychotherapy. *Pragmatic Case Studies in Psychotherapy, 13,* Art. 4.3.

Karlin, R., Hill, A., & Messer, S. (2008). Responding and failing to respond to both hypnosis and a kinesthetic illusion, Chevreul's pendulum. *International Journal of Clinical and Experimental Hypnosis, 56,* 83–98.

Karlin, R., Morgan, D., & Goldstein, L. (1980). Hypnotic analgesia: A preliminary investigation of quantitated hemispheric EEG and attentional correlates. *Journal of Abnormal Psychology, 31,* 227–234.

Karlin, R., & Orne, E. (2001). Neural basis of hypnosis. In N. Smelser & P. Baltes (Series Eds.) & R. Thompson & J. McClelland (Vol. Eds.), *International encyclopedia of the social and behavioral sciences* (Vol. 10, pp. 7101–7105). New York: Elsevier Science.

Karlin, R., & Orne, M. (1996). Hypnosis, social influence, incestuous child abuse, and satanic ritual abuse: The iatrogenic creation of horrific memories for the remote past. *Cultic Studies Journal, 13,* 42–94.

Kendrick, C., Sliwinski, J., Yu, Y., Johnson, A., Fisher, W., Kekecs, Z., et al. (2016). Hypnosis for acute procedural pain: A critical review. *International Journal of Clinical and Experimental Hypnosis, 64,* 75–115.

Kiecolt-Glaser, J., Marucha, P., Atkinson, C., & Glaser, R. (2001). Hypnosis as a modulator of cellular immune dysregulation during acute stress. *Journal of Consulting and Clinical Psychology, 69,* 674–682.

Kihlstrom, J. (2013). Neuro-hypnotism: Prospects for hypnosis and neuroscience. *Cortex, 49,* 365–374.

Kirsch, I. (2010). *The emperor's new drugs: Exploding the antidepressant myth.* New York: Basic Books.

Kirsch, I., & Lynn, S. (1995). The altered state of hypnosis: Changes in the theoretical landscape. *American Psychologist, 50,* 846–858.

Kirsch, I., Montgomery, G., & Sapirstein, G. (1995). Hypnosis as an adjunct to cognitive-behavioral psychotherapy: A meta-analysis. *Journal of Consulting and Clinical Psychology, 63,* 214–220.

LaBriola, F., Karlin, R., & Goldstein, L. (1984). *Quantitated measure of right hemisphere involvement during hypnotic cognitive tasks.* Paper presented at the 36th annual meeting of the Society for Clinical and Experimental Hypnosis, San Antonio, TX.

Laurence, J., & Perry, C. (1988). *Hypnosis, will, and memory: A psycho-legal history.* New York: Guilford Press.

Liossi, C., & Hatira, P. (1999). Clinical hypnosis versus cognitive behavioral training for pain management with pediatric cancer patients undergoing bone marrow aspirations. *International Journal of Clinical and Experimental Hypnosis, 47,* 104–116.

Liossi, C., & Hatira, P. (2003). Clinical hypnosis in the alleviation of procedure-related pain in pediatric oncology patients. *International Journal of Clinical and Experimental Hypnosis, 51,* 4–28.

Liossi, C., White, P., & Hatira, P. (2006). Randomized clinical trial of local anesthetic versus a combination of local anesthetic with self-hypnosis in the management of pediatric procedure-related pain. *Health Psychology, 25,* 307–315.

Liossi, C., White, P., & Hatira, P. (2009). A randomized clinical trial of a brief hypnosis intervention to control venipuncture-related pain of paediatric cancer patients. *Pain, 142,* 255–263.

Loeser, J. Butler, S., Chapman, C. R., & Turk, D. (Eds.). (2001). *Bonica's management of pain* (3rd ed.). Philadelphia: Lippincott Williams & Wilkins.

Luborsky, L. (1995). Are common factors across different psychotherapies the main explanation for the Dodo Bird verdict that "Everyone has won so all shall have prizes"? *Clinical Psychology: Science and Practice, 2,* 106–109.

Lynn, S., Boycheva, E., & Barnes, S. (2008). To assess or not assess hypnotic susceptibility: That is the question. *American Journal of Clinical Hypnosis, 51,* 161–165.

Lynn, S., Kirsch, I., & Rhue, J. (1996). *Casebook of clinical hypnosis.* Washington, DC: American Psychological Association.

Lynn, S., & Rhue, J. (1991). *Theories of hypnosis: Current models and perspectives.* New York: Guilford Press.

McCloud-Morgan, C. (1984, October). *Laterality during tasks and hypnosis.* Paper presented at the 36th annual meeting of the Society for Clinical and Experimental Hypnosis, San Antonio, TX.

McGlashan, T., Evans, F., & Orne, M. (1969). The nature of hypnotic analgesia and placebo response to experimental pain. *Psychosomatic Medicine, 31,* 227–246.

McNally, R. (2003). *Remembering trauma*. Cambridge, MA: Harvard University Press.

Montgomery, G., Bovbjerg, D., Schnur, J., David, D., Goldfarb, A., Weltz, C., et al. (2007). A randomized clinical trial of a brief hypnosis intervention to control side effects in breast surgery patients. *Journal of the National Cancer Institute, 99,* 1304–1312.

Montgomery, G., David, D., Winkel, G., Silverstein, J., & Bovbjerg, D. (2002). The effectiveness of adjunctive hypnosis with surgical patients. *Anesthesia and Analgesia, 94,* 1639–1645.

Montgomery, G., DuHamel, K., & Redd, W. (2000). A meta-analysis of hypnotically induced analgesia: How effective is hypnosis? *International Journal of Clinical and Experimental Hypnosis, 48,* 138–153.

Montgomery, G., Hallquist, M., Schnur, J., David, D., Silverstein, J., & Bovbjerg, D. (2010). Mediators of a brief hypnosis intervention to control side effects in breast surgery patients: Response expectancies and emotional distress. *Journal of Consulting and Clinical Psychology, 78,* 80–88.

Montgomery, G., Schnur, J., & David, D. (2011). The impact of hypnotic suggestibility in clinical care settings. *International Journal of Clinical and Experimental Hypnosis, 59,* 294–309.

Morgan, A., & Hilgard, J. (1978a). The Stanford Hypnotic Clinical Scale for adults. *American Journal of Clinical Hypnosis, 21,* 134–147.

Morgan, A., & Hilgard, J. (1978b). The Stanford Hypnotic Clinical Scale for children. *American Journal of Clinical Hypnosis, 21,* 148–169.

Oneal, B., Patterson, D., Soltani, M., Teeley, A., & Jensen, M. (2008). Virtual reality hypnosis in the treatment of chronic neuropathic pain: A case report. *International Journal of Clinical and Experimental Hypnosis, 56,* 451–462.

Orne, M. (1959). The nature of hypnosis: Artifact and essence. *Journal of Abnormal and Social Psychology, 58,* 277–299.

Orne, M. (1970). Hypnosis, motivation, and the ecological validity of the psychological experiment. In W. Arnold & M. Page (Eds.), *Nebraska Symposium on Motivation* (pp. 187–265). Lincoln: University of Nebraska Press.

Orne, M., & O'Connell, D. (1967). Diagnostic ratings of hypnotizability. *International Journal of Clinical and Experimental Hypnosis, 15,* 125–133.

Palsson, O., Turner, M., Johnson, D., Burnett, C., & Whitehead, W. (2002). Hypnosis treatment for severe irritable bowel syndrome: Investigation of mechanisms and effects on symptoms. *Digestive Diseases and Sciences, 47,* 2605–2614.

Patterson, D. (2010). *Clinical hypnosis for pain control*. Washington, DC: American Psychological Association.

Patterson, D. (2014, February). *Hypnosis and mindfulness in pain management*. Paper presented at the Rehabilitation Psychology Conference, San Antonio, TX.

Patterson, D., Everett, J., Burns, G., & Marvin, J. (1992). Hypnosis for the treatment of burn pain. *Journal of Consulting and Clinical Psychology, 60,* 713–717.

Patterson, D., & Jensen, M. (2003). Hypnosis and clinical pain. *Psychological Bulletin, 129,* 495–521.

Patterson, D., Jensen, M., Wiechman, S., & Sharar, S. (2010). Virtual reality hypnosis for pain associated with recovery from physical trauma. *International Journal of Clinical and Experimental Hypnosis, 58,* 288–300.

Patterson, D., Wiechman, S., Jensen, M., & Sharar, S. (2006). Hypnosis delivered through immersive virtual reality for burn pain: A clinical case series. *International Journal of Clinical and Experimental Hypnosis, 54,* 130–142.

Perry, C. (1977). Is hypnotizability modifiable? *International Journal of Clinical and Experimental Hypnosis, 25,* 125–146.

Piccione, C., Hilgard, E., & Zimbardo, P. (1989). On the degree of stability of measured hypnotizability over a 25-year period. *Journal of Personality and Social Psychology, 56,* 289–295.

Rainville, P., Carrier, B., Hofbauer, R., Bushnell, M., & Duncan, G. (1999). Dissociation of sensory and affective dimensions of pain using hypnotic modulation. *Pain, 82,* 159–171.

Raz, A. (2004, November). *Multiple assays of highly- and less-hypnotizable individuals*. Paper presented at the 55th annual meeting of the Society for Clinical and Experimental Hypnosis, Santa Fe, NM.

Rhue, J., Lynn, S., & Kirsch, I. (1993). *Handbook of clinical hypnosis*. Washington, DC: American Psychological Association.

Schoenberger, N. (2000). Research on hypnosis as an adjunct to cognitive-behavioral psychotherapy. *International Journal of Clinical and Experimental Hypnosis, 48,* 154–169.

Shapiro, S., & Carlson, L. (2009). *The art and science of mindfulness: Integrating mindfulness into psychology and the helping professions*. Washington, DC: American Psychological Association.

Shor, R. (1979). The fundamental problem in hypnosis research as viewed from historic perspectives. In E. Fromm & R. Shor (Eds.), *Hypnosis: Developments in research and new perspectives* (pp. 15–41). Hawthorne, NY: Aldine.

Shor, R., & Orne, E. (1962) *The Harvard group scale of hypnotic susceptibility: Form A*. Palo Alto, CA: Consulting Psychologists Press.

Smith, J., Barabasz, A., & Barabasz, M. (1996). Comparison of hypnosis and distraction in severely ill children undergoing painful medical procedures. *Journal of Counseling Psychology, 43,* 187.

Soltani, M., Drever, S., Hoffman, H., Sharar, S., Wiechman, S., Jensen, M., et al. (2018). Virtual reality analgesia for burn joint flexibility: A randomized controlled trial. *Rehabilitation Psychology, 63,* 487–494.

Spanos, N. (1996). *Multiple identities and false memories: A sociocognitive perspective*. Washington, DC: American Psychological Association.

Spiegel, D. (1989). Cortical event-related evoked potential correlates of hypnotic hallucinations. In V. Gheorghiu, P. Netter, H. J. Eysenck, & R. Rosenthal (Eds.), *Suggestion and suggestibility: Theory and research* (pp. 183–189). New York: Springer-Verlag.

Tan, G., Rintala, D., Jensen, M., Fukui, T., Smith, D., & Williams, W. (2015). A randomized controlled trial of hypnosis compared with biofeedback for adults with chronic low back pain. *European Journal of Pain, 19,* 271–280.

Vaitl, D., Birbaumer, N., Gruzelier, J., Jamieson, G., Kotchoubey, B., & Kubler, A. (2005). Psychobiology of altered states of consciousness. *Psychological Bulletin, 131,* 98–127.

Valentine, K., Milling, L., Clark, L., & Moriarty, C. (2019). The efficacy of hypnosis as a treatment for anxiety: A meta-analysis. *International Journal of Clinical and Experimental Hypnosis, 67,* 336–363.

Wall, V., & Womack, W. (1989). Hypnotic versus active cognitive strategies for alleviation of procedural distress in pediatric oncology patients. *American Journal of Clinical Hypnosis, 31,* 181–191.

Wampold, B., & Imel, Z. (2015). *The great psychotherapy debate: The evidence for what makes psychotherapy work* (2nd ed.). New York: Routledge.

Weitzenhoffer, A., & Hilgard, E. (1959). *Stanford Hypnotic Susceptibility Scales: Forms A and B*. Palo Alto, CA: Consulting Psychologists Press.

Weitzenhoffer, A., & Hilgard, E. (1962). *Stanford Hypnotic Susceptibility Scales: Form C*. Palo Alto, CA: Consulting Psychologists Press.

Weitzenhoffer, A., & Hilgard, E. (1967). *Revised Stanford Profile Scales of Hypnotic Susceptibility: Forms I and II*. Palo Alto, CA: Consulting Psychologists Press.

Whitehouse, W., Dinges, D., Carota Orne, E., Keller, S., Bates, B., Bauer, N., et al. (1996). Psychosocial and immune effects of self-hypnosis training for stress management throughout the first semester of medical school. *Psychosomatic Medicine, 58,* 249–263.

Whorwell, P., Prior, A., & Colgan, S. (1987). Hypnotherapy in severe irritable bowel syndrome: Further experience. *Gut, 28,* 423–425.

Whorwell, P., Prior, A., & Faragher, E. (1984). Controlled trial of hypnotherapy in the treatment of severe refractory irritable bowel syndrome. *Lancet. Gastroenterology and Hepatology, 2,* 1232–1234.

Woolf, C., & Chong, M. (1993). Preemptive analgesia: Treating postoperative pain by preventing the establishment of central sensitization. *Anesthesia and Analgesia, 77,* 362–379.

Woolfolk, R. (1998). *The cure of souls: Science, values, and psychotherapy*. San Francisco: Jossey-Bass.

Zeltzer, L., & LeBaron, S. (1982). Hypnosis and nonhypnotic techniques for reduction of pain and anxiety during painful procedures in children and adolescents with cancer. *Journal of Pediatrics, 101,* 1032–1035.

SECTION E

OTHER METHODS

CHAPTER 20

Stress Concepts in Relation to Music

Töres Theorell
Ingrid Hammarlund

Stress can be defined in many ways. Originally, stress theories were focused on the acute stress reactions, whereas long-term consequences (chronic stress) of exposure to repeated acute stress reactions have been discussed during later years. There is some confusion regarding definitions of long-term stress. Even the acute stress reaction is defined in many different ways. Among laypeople, there may be as many definitions of acute stress as there are participants. To make a complicated discussion short, one might say that there are two kinds of definitions of acute stress:

1. Stress is the energy mobilization that is needed when an individual is exposed to an unusual threat or challenge. This definition is focused on the nonspecific reaction. It does not have any positive or negative connotation. This definition is close to Hans Selye's original definition of stress and has its origin in physiological theory.
2. Stress is the individual's response to a situation that taxes an unusually large amount of the individual's resources. This definition has its origin in psychology, mainly in coping theory (Richard Lazarus). According to this thinking, stress has a negative connotation.

The cognitive activation theory of stress (CATS; Ursin & Eriksen, 2010) is related to the second definition. It postulates that all reactions to challenges and threats are influenced by expectations. If a person has had positive experiences in such situations, he or she is likely to cope well, whereas the opposite is true of negative expectations. *Helplessness* means that the person does not expect him- or herself to be able to do anything to reduce the threat or manage the challenge. An even worse situation is *hopelessness,* in which the person expects the situation to become worse if he or she tries to do anything.

These definitions are important to remember when we discuss the use of music in relation to stress management. There are many immediate psychological, social, and biological effects of music while it is ongoing, and this has been studied extensively. A more difficult question is how repeated uses of music affect chronic stress, and this question has been examined scientifically to a much smaller extent.

As proposed by McEwen (1998), the crucial elements in chronic stress are (1) exposure for long periods of time (weeks/months/years) to repeated acute stress reactions and (2) constant energy mobilization with insufficient opportunity for recuperation. Such states give rise to disturbed regulation of stress reactions. These disturbances could be an inability to downregulate an alerted state (resulting, e.g., in insomnia) or an inability to mobilize energy when needed (as in chronic fatigue syndrome or burnout). However, a factor of equal importance to the possible negative consequences of chronic stress is the activity in the system that protects the cells from adverse effects. A sweeping label for this is the *regenerative system*. The hypothalamic–pituitary–adrenocortical (HPA) axis stimulates the release of cortisol with its crucial importance for the stress reaction. It is counterbalanced by the hypothalamic–pituitary–gonadal (HPG) axis, which stimulates the release of hormones such as oxytocin, human growth hormone, and sex steroids with its precursor DHEA, which help the body to repair and replace worn-out cells and to restore a healthy balance.

AROUSAL AND RELAXATION: A SPECTRUM OF MOMENTARY MUSIC EFFECTS RELEVANT TO STRESS MANAGEMENT

That music may have immediate effects on several organ systems in the body is becoming increasingly clear with the increasing variety of recording equipment for various organ systems. For instance, recordings of peristaltic movements in the stomach (Chen, Xu, Wang, & Chen, 2005), performed by means of a pressure recording device swallowed by the participant, showed that harmonic slow classical music stimulated well-coordinated slow peristaltic movements (supposedly good for alimentation!), whereas noise stimulated fibrillating uncoordinated peristaltics. More indirectly, assessment of the ability of the arterial wall to recuperate after compression (Miller, Mangano, Beach, Kop, & Vogel, 2010) has shown that participants who listen to happy music while an artery in the forearm is compressed, as in blood pressure assessment, have a better recuperation afterward, when the compression is released, than when they listen to anxiety-provoking music. This relates to the function of the endothelial cells on the inside of the artery and could theoretically be of significance in atherosclerosis.

Music is being used increasingly in medical settings, for instance, for pain management (Bernatzky, Presch, Anderson, & Panksepp, 2011), but also in treatment of depression, after surgery and other painful medical procedures, and in dementia care and Parkinson's disease, just to mention a few examples (Theorell, 2014; Jensen & Bonde, 2018). Piloerection is an interesting phenomenon that arises unexpectedly when people listen to music and become surprised by the music's strong effect. Piloerection is literally erection of hair. All fur animals have the ability to erect the straws in their fur, either because they need to increase their size or to increase insulation against cold temperatures. In an experiment (Vickhoff, Åström, Theorell, & von Scheele, 2012) that we performed, the listening study participant had an unexpected piloerection when piano music was improvised during a change from calm, slow, rhythmic, and harmonic music to disharmonic rhythm with irregular beat. Physiological recordings showed at first increasing heart rate and at the same time diminished heart rate variability, then increased sweating (skin conductance), and finally decreased finger temperature (decrease of 0.7° C during 40 seconds). The cold skin temperature coincided with piloerection. Again, this illustrates how strong our physiological reactions can be to musical stimuli, a well-known observation among music therapists.

In an experiment performed on a group of 37 young adults, the participants were asked to select two of their own favorite music pieces (Lingham & Theorell, 2009), one piece that, according to their own assessment, was "stimulating" and one that they considered "relaxing." While sitting quietly, they listened in random order to these pieces. Heart rate, breathing frequency, and emotional state were recorded, and recordings made while they were listening to their favorite pieces were compared with the preceding restful silence. During the stimulating piece, the average increase in heart rate was 7 beats per minute compared with the quiet condition. The relaxing music, on the other hand, did not produce the expected deceleration of heart rate in some individuals. Quite to the contrary, in 11 cases it was even associated with a small increase in heart rate. There were similar observations with regard to breathing. During the stimulating music, breathing frequency increased significantly, with an average increase of four breathing cycles per minute. During relaxing music, on the other hand, there was no significant change. Emotional self-recordings verified this in the psychological domain. The stimulating music quite clearly induced arousal feelings, while the relaxing piece triggered both relaxed and aroused feelings. The first conclusion to be drawn from this seems to be that these highly educated people were skillful in selecting their own stimulating music, but they were not as successful with regard to relaxing music, which frequently did not have the expected calming effect on heart rate and breathing. The second conclusion is that responses to self-selected music showed enormous interindividual variation. Despite the fact that the participants did not move, some of them had strong heart rate reactions. One participant's heart rate increased by 20 beats per minute while she was listening to the stimulating music and decreased by 20 beats when she listened to the relaxing music. Other participants' heart rates did not change at all during music listening.

Research has shown that specially adapted music may decrease both the subjective experience of physical effort and the physiological reactions to it. Szmedra and Bacharach (1998), for instance, did such an experiment several years ago. Young men were asked to do exactly the same physical work on the treadmill during two different conditions. One condition was carried out in silence, and the other while listening to special gym music. The two conditions were randomly ordered. The blood concentration of lactic acid was assessed. Heart rate and blood pressure were recorded, and the participants were asked to make a self-rating of effort. Despite the fact that the physical work was fixed and exactly the same in the two conditions, blood pressure and heart rate elevation were smaller during the music condition, and the same observation was made for subjective effort and lactic acid concentration.

THE RELATIONSHIP BETWEEN STRESS AND MUSIC EXPERIENCES

There is an extensive scientific literature supporting the idea that music experiences can reduce or amplify the acute stress response. It is harder to find published research supporting the corresponding idea that music experiences can reduce long-term stress.

What about the acute stress response (energy mobilization, according to terminology above) in relation to music? Essential components seem to be:

1. *Amplitude*. Forceful music volume is in itself a stressor, a fact that has been extensively shown in the scientific literature on physiological reactions to noise. On the contrary, low volume can be soothing.

2. *Rhythm*. When the music rhythm is faster than one's own heart rate, the pulse tends to increase in many participants, and vice versa. The mechanisms behind this are largely unknown. However, the parasympathetic system through the vagus nerve may play an important role, and one of the possible mediators is oxytocin. Ooishi, Mukai, Watanabe, Kawato, and Kashino (2017), for instance, showed that listening to slow tempo music induces an elevation of the saliva concentration of oxytocin. An increased release of oxytocin is related to an increased activity in the parasympathetic system. It is a well-established fact in physiology that deep, slow breathing increases heart rate variability and lowers the average heart rate. Both of these effects mirror changes in balance from more sympathetic to more parasympathetic activity. Chaotic, stumbling (e.g., periods of five or seven beats in the bars), and unpredictable rhythms could also be distressing for people with no advanced music training, whereas slow predictable rhythms may be soothing. *Sudden changes* in the music may, per se, induce drastic changes in physiological state (Bernardi & Sleight, 2007).

3. *Chords*. The harmonic landscape of the music may also be important. The diatonic chord "big third" is perceived, for instance, by most participants who lack professional music training as calming and "inviting." Krantz, Madison, and Merker (2006) showed that the majority of musically untrained nondancers who were randomly exposed to different kinds of chords and were asked to move when they heard the music spontaneously chose embracing movements when the big third—which is a harmonic chord—was played. By contrast, when the same people listened to a disharmonic diatonic chord (big septima), they spontaneously chose poorly coordinated movements. In another study, we found that a short period of irregular heartbeat followed exposure to the big septima significantly more often than expected (Krantz, Kreutz, Ericson, & Theorell, 2010).

4. *Pitch*. In general, high-pitched music is associated with arousal, and vice versa.

5. *Dominant movement in the music*. Although this has not been studied systematically, music with a dominance of movement downward from high to low pitch is more likely to induce feelings of sadness and threat, whereas movement upward may increase joy and pride.

Apart from the physical characteristics of the music piece described above, there are also other conditions surrounding the music experience that determine whether the total effect will be either relaxing or arousing:

1. *Cohesiveness* in the situation. Oxytocin may be involved in this as well (Gebauer et al., 2016). Research has shown that the experience of a singing lesson for a pupil who is accustomed to this situation is associated with a rising plasma concentration of oxytocin (Grape, Sandgren, Hansson, Ericson, & Theorell, 2002), and it has also been shown that half an hour of choir singing is associated with increased prevalence of positive and decreased prevalence of negative feelings, as well as rising saliva oxytocin concentration (proxy for plasma concentration). No such changes were observed in the same group when the participants had conversations in small groups (Kreutz, 2014). This means that the social component in mutual interactions may be strengthened by music and that this relates to the activation of oxytocin release that is associated with parasympathetic activation. A word of caution—the fluctuations in oxytocin into blood (and secondly to saliva) are rapid. In studies of blood and saliva oxytocin concentration in relation to

musical experiences, conflicting results have been found that could be due to differences in time sequences for collection of samples.

2. *Associations to previous music experiences*. The music that the individual hears may have been associated in previous experience with emotionally charged situations. If the emotion is anxiety, a stress reaction may arise. This could be paradoxical in the sense that a music piece that is soothing or calming for most people could be anxiety-provoking for a person who has heard the piece in an anxiety-provoking context before. In the same vein, other emotions, such as joy, anger, sadness, and pride, could be associated with the piece. Of course, ethnic factors are also important. Participants whose dominant childhood music has been classical music are likely to respond in a different way physiologically to a piece by Mozart than are those who grew up with Elvis Presley's recordings.

3. *Context*. Music listening may *induce* feelings or *amplify* feelings that the participant already experiences in the moment, depending on the characteristics of the music and the memories associated with it. However, the context surrounding the music experience may have a decisive role. Temperature, acoustics, presence of other people and their emotional states, light conditions, and so forth, are examples of such factors.

MUSIC PSYCHOPHYSIOLOGY AND LONG-TERM CONSEQUENCES ON CHRONIC STRESS

Gabrielsson (2011) recruited more than 900 participants for an interview study. He asked them to describe in their own words the most profound musical experience that they had had in their lives and then went on to categorize these experiences with regard to contents, context, and consequences. Gabrielsson stated that it is very difficult to do such a categorization because the experiences cover a very large area, asserting that music seems to comprise the "whole psychological reality." Many of these situations were seen as turning points in people's lives. He provided examples of participants who reported that they had been deeply depressed when they discovered a kind of music that they had never been interested in before. In their depressed mood, they became passionately engaged in listening to this particular kind of music. In retrospect, they believed that this experience helped them out of the depression. A strong acute reaction may have profound long-term effects on health, as a reorientation in life may take place as a result.

Other research in this field has shown that in long-distance cycling (10 kilometers), when the cyclists choose their own tempo, listening to fast dance music ("trance" with tempo 142 beats per minute) is likely to induce an elevated cycling tempo. The work is perceived as harder without music (Atkinson, Wilson, & Eubank, 2004). In another study, the effects of different types of music on heart rate, rating of perceived exertion, and time to exhaustion were studied. The participants performed their physical work on a treadmill, and the conditions were randomly allocated to "soft music," "loud, fast, and exciting music," and silence. The results showed that the soft music reduced physiological and psychological arousal during submaximal exercise and also increased endurance of performance (Copeland & Franks, 1991).

In many activities typical in the modern world, music is used in ways that may seem new but are, in fact, very old. A striking example is the music played in the gym. Special kinds of music have been developed for this (De Nora, 2000). One is reminded of all music that has been used in the history of man in order to facilitate physical work—for instance, "pulling the boat" songs, sailor songs for rowing or managing big sails, and march music for facilitating long troop walks (see also Merriam, 1964).

SCIENTIFIC EVIDENCE OF EFFECTS OF MUSIC IN STRESS MANAGEMENT

Scientific evidence could be defined in many ways. The term *evidence-based* mostly refers to evidence from multiple scientific evaluations using randomized controlled trials (RCTs). The most well-known strategy for the assessment of degree of scientific evidence following such traditions has been introduced by the Cochrane Institute. When there are many published studies following RCTs that uniformly point at positive results, the evidence grade is maximal (4 out of 4 possible levels using the so-called GRADE system). Ideally, RCTs should be double-blind, which means that neither the experimenter nor the participants know which group they belong to. This is, of course, not possible in studies of the effects of music therapy.

The Cochrane system builds upon several conditions. First of all, participants in ideal evaluation trials should have been randomly allocated either to the intervention to be evaluated (in this case, mostly some kind of music listening) or to a comparison intervention. The latter could be treatment as usual or, when we are dealing with population samples not suffering from any disease, no particular intervention at all. This means that the Cochrane evaluation system is more suitable for judging the effectiveness of pharmacological therapy—in which it is possible to use placebo pills. Following the Cochrane system strictly, it would therefore be impossible for evidence regarding effects of music therapy to reach level 4.

Several Cochrane reviews of the effectiveness of music therapy or use of music (listening or performing) in relation to the management of several stress-related conditions have been published during recent years. Thus such reviews exist for depression (Aalbers et al., 2017), insomnia (Jespersen, Koenig, Jennum, & Vuust, 2015), stress and anxiety for people with coronary heart disease (Bradt, Dileo, & Potvin, 2013), preoperative anxiety (Bradt, Dileo, & Shim, 2013), and cancer (Bradt, Dileo, Magill, & Teague, 2016). The general conclusion of these reviews is that the number of published strictly designed studies is small but that results are promising. The most established fields seem to be the use of music against preoperative anxiety and stress and anxiety in patients with coronary heart disease (CHD). In a similar vein, a recent meta-analysis based on 15 randomized trials showed convincing evidence that music listening reduces anxiety during cardiac catheterization (Jayakar & Alter, 2017). Not unexpectedly, these reviews have shown that more research has been published on amelioration of acute stress reactions than on long-term conditions such as depression.

However, a more detailed overview of Cochrane reviews of relevance to the use of music in stress-related situations is found in Table 20.1. These are the most frequently examined groups and situations for which music listening in any form has been used. In general evidence, level 3 is reached. The exception is stress and anxiety among patients with CHD, for whom the evidence level was rated lower, 2 out of 4. The reason that the evidence level is judged to be lower for that outcome is that the included studies are very heterogeneous and therefore difficult to summarize. A general difficulty in assessing the outcomes is that the music stimuli have been weak. Recorded music has been used, and in many cases the choice of music has not been individualized. Music therapy has not been used in many of these studies.

A general difficulty with the evaluations is that they have often been performed on too-small samples. A formal statistical power calculation in advance would have shown that the study samples were too small to be able to show significant effects. As has been pointed out by evaluation experts, it is to do a disfavor to the field to publish a negative evaluation that is based upon samples that are too small.

TABLE 20.1. Stress-Related Outcomes in Relation to the Use of Music (Based on the Most Recent Cochrane Reviews)

Evidence level	GRADE	Participants/ no. of studies	First author
Depression	3/4, better than TAU	421/9	Aalbers (2017)
Insomnia	3/4, improves Subj sleep	314/6	Jespersen (2015)
Stress and anxiety in CHD patients	2/4, reduces anxiety, BP, HR, RR, sleep problems, pain	1,369/26	Bradt (2013) (1)
Preoperative anxiety	3/4, reduces anxiety	2,051/26	Bradt (2013) (2)
Cancer	3/4, reduces anxiety, pain, fatigue, improves quality of life	528/7	Bradt (2016)
Cardiac catheterization	3/4, reduces anxiety	695/6	Jayakar (2017)

Note. TAU, treatment as usual; Subj sleep, assessments using self-administered questionnaires; BP, blood pressure; HR, heart rate; RR, respiratory rate; CHD, coronary heart disease.

DIFFERENT KINDS OF MUSIC THERAPY

> The meaning of the world can only be acquired in communication and collaboration with other people. There is no such thing as meaning found entirely by a single self. Meaning has to be communicated or communicable. (Trevarthen, 2003, p. 67)

Music is considered a powerful therapeutic tool because of its ability to simultaneously access physiological, psychological, cognitive, and aesthetic reactions. Music has, throughout history, been used as a healing force to alleviate distress and illness. Modern music therapy is a research-based health profession, a clinical practice, and an academic discipline. Music therapy is both a science and an art form. Clinical music therapy can be understood as the use of sounds and music within a therapeutic relationship to support and encourage health and well-being (Wigram, Saperston, & West, 1995).

Clinical and theoretical perspectives show how music therapy has been matching practice to theory and theory to practice over time in the process of defining and applying music therapy in different contexts and clinical fields. There are today many models and methods of music therapy.

At the Ninth World Congress of Music Therapy, held in Washington, DC, in 1999, *five models* of music therapy used in clinical practice, research, and training were introduced as internationally acknowledged models developed since the beginning of 1970.

1. The *Bonny method of guided imagery and music* was founded in the United States by Helen Bonny as a depth-oriented approach to music psychotherapy. It is an active music listening process to generate dynamic unfolding of inner experiences and images. Theory-based in humanistic and transpersonal psychology, guided imagery and music (GIM) is a receptive music therapy model based on music listening. It is used in a number of clinical settings, often in the somatic field for clients suffering from heart problems, cancer, or other life-threatening diseases and in palliative care. The music is carefully chosen to stimulate and sustain a dynamic unfolding of bodily experiences, sensations, feelings, and inner imagery. The music is introduced to the client after a focused

relaxation to release tension and facilitate the music experience. GIM has, through many years, developed as a specialized psychotherapeutic music therapy method, and practitioners of it enhance their therapeutic practice with concepts drawn from many theorists. There is an increasing number of clinical studies and research on this method in both effect and process, with qualitative, quantitative, and mixed-methods designs (Wrangsjö & Körlin, 1995; Körlin & Wrangsjö, 2001; Bonde, 2005; Grocke, 2010; Beck, 2012).

2. *Analytically oriented music therapy* was founded in the United Kingdom by Mary Priestley as a music psychotherapeutic method based on the symbolic use of musical improvisations, followed by an interpretive therapeutic dialogue between client and therapist. It is theory-based in the psychodynamic tradition.

3. *Creative music therapy* was founded by Paul Nordoff and Clive Robbins in the United States and the United Kingdom as an improvisational nondirective approach of music therapy, originally developed for children with severe handicaps and learning disabilities. It was theoretically inspired by humanistic psychology.

4. *Benenzon music therapy* was founded by Rolando Benenzon in Argentina as an eclectic model inspired by many different psychological and psychotherapeutic theories and psychodrama.

5. *Cognitive-behavioral music therapy* was originally founded in the United States by Clifford Madsen to stimulate cognitive and behavioral development for developmentally disabled children and adolescents. A modern, science-based treatment model for neurological rehabilitation was later founded by Michael Thaut, who also developed a specific training in "neurological music therapy" based on the principles of cognitive music therapy (Trondalen & Bonde, 2012).

Bruscia (1998) defines the concept of a music therapy model as a systematic and unique approach to method, procedure, and technique based on certain principles. Fundamental to all approaches is the emphasis on affording music experiences within a therapeutic relationship between client and therapist to promote health and well-being (Wheeler, 2015).

The World Federation of Music Therapy (*www.wfmt.info*) decided on a general definition: "Music therapy is the professional use of music and its elements as an intervention in medical, educational and everyday environments with individuals, groups, families or communities who seek to optimize their quality of life and improve their physical, social, communicative, emotional, intellectual and spiritual health and wellbeing. Research, practice, education and clinical training in music therapy are based on professional standards according to cultural, social and political contexts."

MUSIC THERAPY TRAINING

Music therapy is the professional practice component of a discipline informed by theory and research. Music therapy is an academic and clinical field with training programs at the bachelor's and master's levels, research centers and networks, doctoral programs all over the world, and an international consortium of research universities (Dileo, 2016).

A music therapy training program teaches how music can be adapted to suit the needs of various people and therapeutic purposes in a given clinical situation. Music is used to address physical, emotional, cognitive, and social needs based on proven experience and evidence. Music can release tension, stimulate movement, change mood, and promote social interaction. Music is a medium in which feelings can be articulated and shared. A music therapy session can be described as a reflexive process wherein the therapist supports and encourages the client to use music experiences and the relationship formed through them as an impetus for change, improvement, well-being, and quality of life.

Musical skills and musicianship are the ground from which the music therapist develops therapeutic competence. A music therapist needs to be flexible in his or her musical expression and to develop expertise in order to be able to choose appropriate procedures and approaches for different clients. The training in such a program usually involves music psychology, musical biography, listening skills and auditory training, musical form, structure and analysis, body and voice work, and accompaniment skills.

The therapeutic identity and ability is trained and developed through self-experience training, ethics, clinical practicum periods, and supervision for individual and group therapy. The theoretical and scientific studies in the training inform the students how to formulate objectives and goals of music therapy in both somatic and psychiatric care. Music therapists mostly work in multidisciplinary teams in institutions and hospitals. This requires a comprehensive interdisciplinary knowledge and dialogue.

Music, Health, and Well-Being

Music, health, and well-being has in the last 10 years developed as a broad interdisciplinary research field, opening for the exploration of the biological foundations of music's health effects; the psychological, philosophical, and cultural foundations of music; clinical music therapy; and music in medicine and public health (MacDonald, Kreutz and Mitchell, 2012).

Today, music therapy is contextualized within this broad framework of methods and theories that apply music experiences and music interventions for health and well-being purposes. Music therapy can be understood as one of many ways to promote health by using music to regulate emotional states, empower communication, and promote well-being.

Music Therapy in the Medical Context: Expressive and Receptive Methods

In clinical practice, music therapists work with a variety of methods: music listening, vocal and instrumental improvisation, movement, singing, composing, and performing. In clinical *improvisation*, the music develops within the patient's and therapist's immediate relationship. Music has the ability to move us both physically and emotionally. These experiences can be understood with concepts of dynamic forms of vitality, arousal, affect attunement, mirroring, matching, grounding, holding, and containing (Stern, 2010). The use of music therapy improvisation has been studied with patients having a wide range of clinical issues: neurorehabilitation (Magee & Baker, 2009), cancer (Pothoulaki, MacDonald, & Flowers, 2012), palliative care (Hartley, 2001), mental illness (Gold et al., 2013; Storz, Maes, von Kamp, Schumacher, & Martin, 2014), eating disorders (Trondalen, 2003; Robarts, 2000), forensic psychiatry care (Hakvoort, 2014), and dementia (Ridder, Stige, Qvale, & Gold, 2013).

Within music therapy, the "receptive method" of intentional listening to music in a relaxed state is used for children, adolescents, adults, and older people in hospitals and in palliative/hospice care. The purpose is mainly to relax, soothe, and reduce pain and anxiety in contexts that are stress-inducing (Grocke & Wigram, 2007). Receptive music therapy has been used for patients with burnout syndrome, which is regarded as a consequence of long-lasting exposure to constant energy mobilization without periods of relaxation and recuperation. A large, four-armed randomized trial with 150 patients has shown that two specific music programs reduced burnout symptoms significantly after 5 weeks and that these effects were maintained during a long follow-up period (Brandes et al., 2009). As far as we know, there are no Cochrane reviews examining the effects of the use of music in treatments of burnout syndrome, but this is a promising and potentially important field.

Music listening is used increasingly for anxiety relief in many medical contexts. A frequent situation in scientific evaluations is the use of music listening postoperatively. One of the randomized studies that have been described is a study of the use of music listening with special bedside equipment after open heart surgery (Nilsson, 2009). This was a study of 40 patients, who were randomized into two equal-sized groups. In the experimental group, the patients were allowed to select music preoperatively from a music menu, and when they woke up, they listened to this music. There was a significant difference in the release of plasma oxytocin, with increasing levels up to 30 minutes after waking up in the music group versus lowered levels compared with the preoperative concentration in the control group. In addition, oxygen saturation and subjective anxiety improved more in the music group than in the control group. The study is an illustrative example showing results from a rapidly growing research field. Oxytocin has anxiolytic effects and is also associated with decreased pain. The findings are therefore of practical relevance.

The applications of methods are dependent on clinical area, client needs, assessments, and therapeutic goals and are defined by music therapy theory and research. Music therapy can supplement other clinical fields and also offer something unique in itself.

During the past 10 years, music therapy has been implemented in new areas of medical clinical practice across the lifespan, both for individuals and groups: in neonatal care, palliative care for children, and for patients with stress-related and burnout diagnoses among adolescents and adults. Uggla and colleagues (Uggla et al., 2016; Uggla, Bonde, Hammar, Wrangsjö, & Gustafsson, 2018) conducted a randomized trial with children who underwent a very difficult medical procedure lasting for many weeks, namely, haematopoietic stem cell transplantation. Children in the music group received a highly individualized music therapy including both receptive and expressive components twice a week, whereas children in the control group received usual care. The findings showed that the children in the music group had lower heart rates at night, possibly reflecting lower stress levels, than the other children, and also that children in the music group had a better physical function score at the time of discharge.

APPLICATIONS FOR STRESS MANAGEMENT

Components of stress and anxiety are, in a broad perspective, frequently present in many music therapy clinical situations. Music therapists often meet children, adolescents, and adults before, during or after difficult medical procedures. Stress assessment is an important tool for music therapists to:

- facilitate breathing by singing
- promote recognition of bodily responses
- reduce tension and facilitate relaxation

- regulate emotions
- provide alternative coping strategies to deal with feelings of stress

When music experiences are combined with other stress reduction techniques, various factors have to be addressed. Can the music enhance the stress reduction technique? What is the perceptual role of the music: Is it to be mainly in the foreground or in the background? Does the music match and follow verbal instructions in form and content?

Grocke and Wigram (2007) write about relaxation methods in music therapy for adults. In medical settings, music and relaxation can:

- reduce stress and tension
- reduce anxiety prior to medical procedures
- alleviate pain and pain intensity during debridement procedures
- regulate breathing
- enhance music listening experience for well-being
- promote a positive experience within a stressful environment of the hospital

CASE EXAMPLE

The purpose of music therapy in treatment of work-related stress can be seen as a way to stimulate recovery and rehabilitation.

Peter was a 45-year-old man on part-time stress leave from his job as a nurse in a psychiatric clinic. He suffered from psychological symptoms related to stress, loss of motivation, depressive feelings, and insomnia. One month earlier, he had been referred to a weekly rehabilitation group focused on mindfulness.

After listening to a radio program about music therapy, he decided that he wished to try music therapy and contacted one of the music therapists from the program. They made an agreement regarding six music therapy sessions (each lasting for 60 minutes) over a period of 6 weeks.

Peter told the therapist that he wished to increase his self-care and coping ability: "I hope music therapy can help me to explore and integrate new aspects of myself. I feel that I have lost my ability to be warm and listening in contact with the patients in the clinic where I work. I experience myself as tired, stiff, and frozen. And I feel depressive after a long period of stress-related problems at work."

The music therapist initially asked Peter to move around in the music therapy room and explore the appearance and sound of the different instruments and to choose two instruments that he felt were interesting for him just now, in this moment. He slowly walked around and tried the sound of every instrument in the room. Finally, he chose a big hand drum and a long rainstick. The therapist asked him to play the instruments and tell something about his choice. "The sound of the rainstick makes me worried, there is no direction in the sound, no way for me to influence and move further. The drum gives me the possibility to explore and influence the sound. It makes me feel motivated and active in a playful way. That's what I long for."

The therapist proposed to go on in the session by playing music together, an improvisation with Peter on his drum and the therapist at the piano. It could perhaps give them information about how playing together can promote communication and support the therapeutic alliance. The musical interplay developed into a long and varied improvisation, in which the drum kept the process together and the piano formed a melodic narrative.

Music therapy improvisations are often used as a complementary assessment. It is a musical way to provide insight into the clients' coping mechanisms and ways of relating to self and others.

The theme for the second session grew from grounding "warming-up movements" when listening to drumming music from a CD that Peter himself brought into the session. "I was so inspired by using the drum in the first session. It gave me a good contact with a balanced form of energy inside me. For a long time I have thought that I had lost that feeling."

This session went on with an improvisation, this time with an articulated focus on playing with opposites, to try to find a good way to release rigid tension and to make an opening for the experience of a healing process of *ritardando* and *accelerando* in life.

During the sessions, the therapist had noticed that Peter's voice lacked energy and introduced short breathing exercises during the grounding process in the beginning of each session. Experimenting with the voice by mirroring and by playful dialogue often ended in mutual liberating laughs. Focus in the following sessions were different kinds of voice work, from toning without words to improvisations with sounds, words, and songs.

For the two last sessions, Peter felt prepared to explore inner images by listening to music. In the first listening procedure, he was sitting in a chair at a table with paper and crayons, which made it possible for him to draw something *during* the music listening. The artwork can help to "contain" the listening experience in a concrete way. The music piece, *The Enchanted Lake* by Anatolij Liadov, lasts for 8 minutes and was chosen by the therapist to evoke and stimulate images within a structured form. Peter's images during the music surprised him; he saw himself in a boat sailing on a lake. "I felt wind in my sail. A new experience, I never had a chance to sail in my real life! I have decided to take up my piano playing again. Yesterday I played for an hour at home. I felt happy and relaxed."

In the final music therapy session, Peter asked for the possibility to summarize the music therapy experiences in an improvisation for four hands at the piano, together with the therapist. They recorded the improvisation and listened, talked, and evaluated the process together. Peter named the improvisation "From Helplessness to Hope."

MUSIC-BASED EXPERIENCES

Entrainment means synchronization of the physiological rhythms of the body (pulse, heart frequency, breathing) with external rhythmical stimuli through music. The music therapist can "match" the client's heart rate with music and help transformative processes for the client. Drumming, toning, and grounding movements can resonate to inner aspects and stimulate self-awareness and resource-oriented refocusing.

NEGATIVE EFFECTS OF MUSIC

All powerful therapeutic methods may have side effects. This is true also of music. Music listening has been used as an amplifier of torture. The combination of being prevented from movements and listening to sudden disharmonic chords or to endless repetition of short simple melodies can be extremely damaging. As mentioned previously, a participant may have heard a music piece that most individuals would perceive as joyful or calming in an extremely traumatic situation. For this individual, that particular piece is likely, perhaps forever, to elicit an anxiety reaction and worsened stress, despite its joyful character. Accordingly, music must be used with caution and respect for the diversity in symbolic meaning that a music piece could have for various individuals. It is well known that music can also be used for manipulative purposes, for instance, in order to amplify political messages or cheating.

GENERAL DISCUSSION

The clinical course in music therapy is highly individual, and the psychological and biological effects of listening to or making music are also highly individual. Cochrane reviews of the effects of the use of music in relation to stress reduction could therefore be heavily criticized. It is our hope that Cochrane reviews in the future will be based on more elaborate research designs with well-defined music interventions, sufficiently large samples, and well-defined conditions and outcomes. The literature so far shows that music, when individualized and utilized in skillful ways, can be a very important addition to other principles in stress management.

There seem to be few contraindications. However, the importance of individualization and careful attention to the participants' reactions cannot be sufficiently emphasized. Used in the wrong way, music can worsen anxiety and stress.

CONCLUSIONS

The use of music in medical contexts for decreasing stress levels is becoming more and more popular. The number of evaluations of music interventions in such contexts is rapidly growing, and a number of Cochrane reviews indicate that there is added value of music in many of these situations. The use of music in long-term therapy in various forms of music therapy for stress-related conditions has been less extensively studied, but there is growing evidence that the treatment of conditions related to long-term stress such as coronary heart disease, depression, and burnout can benefit substantially from the addition of music therapy.

REFERENCES

Aalbers, S., Fusar-Poli, L., Freeman, R. E., Spreen, M., Ket, J. C. F., Vink, A. C., et al. (2017, November 16). Music therapy for depression. *Cochrane Database of Systematic Reviews, 11*(11), CD004517.

Atkinson, G., Wilson, D., & Eubank, M. (2004). Effects of music on work-rate distribution during a cycling time trial. *International Journal of Sports Medicine, 25*(8), 611165.

Beck, B. D. (2012). *Guided imagery and music (GIM) with adults on longterm sick leave suffering from chronic stress: A mixed methods study*. Unpublished doctoral dissertation, Aalborg University, Institute for Communication, Aalborg, Denmark.

Bernardi, L., & Sleight, P. (2007). Music and biological rhythms. In M. Klockars & M. Peltomaa (Eds.), *Music meets medicine: Proceedings Acta Gyllenbergiana VII*. Helsinki, Finland: Signe and Arne Gyllenberg Foundation.

Bernatzky, G., Presch, M., Anderson, M., & Panksepp, J. (2011). Emotional foundations of music as a non-pharmacological pain management tool in modern medicine. *Neuroscience and Biobehavioral Reviews, 35*(9), 1989–1999.

Bonde, L. O. (2005). *The Bonny method of guided imagery and music (BMGIM) with cancer survivors: A psychosocial study with focus on the influence of BMGIM on mood* *and quality of life*. Unpublished doctoral dissertation, Aalborg University, Aalborg, Denmark.

Bradt, J., Dileo, C., Magill, L., & Teague, A. (2016, August 15). Can music interventions benefit cancer patients? *Cochrane Database of Systematic Reviews, 8*, CD006911.

Bradt, J., Dileo, C., & Potvin, N. (2013, December 28). Music to reduce stress and anxiety for people with coronary heart disease. *Cochrane Database of Systematic Reviews, 12*, CD006577.

Bradt, J., Dileo, C., & Shim, M. (2013). Music interventions for preoperative anxiety. *Cochrane Database of Systematic Reviews, 6*, CD006908.

Brandes, V., Terris, D. D., Fischer, C., Schuessler, M. N., Ottowitz, G., Titscher, G., et al. (2009). Music programs designed to remedy burnout symptoms show significant effects after five weeks. *Annals of the New York Academy of Sciences, 1169*, 422–425.

Bruscia, K. (2014). *Defining music therapy* (2nd ed.). Gilsum, NH: Barcelona.

Chen, D. D., Xu, X., Wang, Z., & Chen, J. D. (2005). Alteration of gastric myoelectrical and autonomic activities with audio stimulation in healthy humans. *Scandinavian Journal of Gastroenterology, 40*(7), 814–821.

Copeland, B. L., & Franks, B. D. (1991). Effects of types and intensities of background music and art on health with ageing. *Arts and Health, 1*, 19–34.

De Nora, T. (2000). *Music in everyday life*. Cambridge, UK: Cambridge University Press.

Dileo, C. (Ed.). (2016). Envisioning the future of music therapy. Philadelphia: Temple University. Retrieved from *www.temple.edu/boyer/documents/ENVISIONING_THE_FUTURE.pdf*.

Gabrielsson, A. (2011). *Strong experiences with music: Music is much more than just music*. Oxford, UK: Oxford University Press.

Gebauer, L., Witek, M. A., Hansen, N. C., Thomas, J., Konvalinka, I., & Vuust, P. (2016). Oxytocin improves synchronisation in leader–follower interaction. *Science Reports, 6*, 38416.

Gold, C., Mössler, K., Grocke, D., Heldal, T. O., Tjemsland, L., Aarre, T., et al. (2013). Individual music therapy for mental health care clients with low therapy motivation: Multicentre randomized controlled trial. *Psychotherapy and Psychosomatics, 82*(5), 319–331.

Grape, C., Sandgren, M., Hansson, L.-O., Ericson, M., & Theorell, T. (2002). Does singing promote well-being?: An empirical study of professional and amateur singers during a singing lesson. *Integrative Physiological and Behavioral Science, 38*, 65–74.

Grocke, D. E. (2010). An overview of research in the Bonny method of guided imagery and music. *Voices, 10*(3).

Grocke, D., & Wigram, T. (2007). *Receptive methods in music therapy techniques and clinical applications for music therapy clinicians, educators, and students*. London: Jessica Kingsley.

Hakvoort, L. (2014). *Cognitive-behavioral music therapy in forensic psychiatry: Workable assumptions, empirical studies and theoretical foundations for primary goal-oriented treatment*. Arnhem, the Netherlands: ArtEZ Press.

Hartley, N. A. (2001). On a personal note: A music therapist's reflections on working with those who are living with a terminal illness. *Journal of Palliative Care, 17*(3), 135–141.

Jayakar, J. P., & Alter, D. A. (2017). Music for anxiety reduction in patients undergoing cardiac catheterization: A systematic review and meta-analysis of randomized controlled trials. *Complementary Therapy in Clinical Practice, 28*, 122–130.

Jensen, A., & Bonde, L. O. (2018). Deltagelse i kunst-og kulturaktiviteter og kunstbaserede interventioner have positiv effekt på somatiske sygdomme? [Can participation in artistic and cultural intervention have a positive effect on somatic illnesses?] *Ugeskrift for Læger, 180*, V06170481.

Jespersen, K. V., Koenig, J., Jennum, P., & Vuust, P. (2015, August 13). Music for insomnia in adults. *Cochrane Database of Systematic Reviews, 8*, CD010459.

Körlin, D., & Wrangsjö, B. (2001). Gender differences in outcome in guided imagery and music. *Nordic Journal of Music Therapy, 10*(2), 132–143.

Krantz, G., Kreutz, G., Ericson, M., & Theorell, T. (2010). Bodily movements influence heart rate variability (HRV) responses to isolated melodic intervals. *Music and Medicine, 3*, 108–113.

Krantz, G., Madison, G., & Merker, B. (2006, August). *Melodic intervals as reflected in body movement*. Paper presented at the Ninth International Conference on Music Perception and Cognition, Bologna, Italy.

Kreutz, G. (2014). Does singing facilitate social bonding? *Music and Medicine, 6*, 51–60.

Lingham, J., & Theorell, T. (2009). Self-selected "favourite" stimulative and sedative music listening: How does familiar and preferred music listening affect the body? *Nordic Journal of Music Therapy, 18,* 150–166.

MacDonald, R. A. R., Kreutz, G., & Mitchell, L. (Eds.). (2012). *Music, health, and wellbeing.* Oxford, UK: Oxford University Press.

Magee, W., & Baker, M. (2009). The use of music therapy in neuro-rehabilitation of people with acquired brain injury. *British Journal of Neuroscience Nursing, 5*(4), 150–156.

McEwen, B. S. (1998). Stress, adaptation and disease: Allostasis and allostatic load. *New England Journal of Medicine, 840,* 33–44.

Merriam, A. P. (1964). *The anthropology of music*. Evanston, IL: Northwestern University Press.

Miller, M., Mangano, C. C., Beach, V., Kop, W. J., & Vogel, R. A. (2010). Divergent effects of joyful and anxiety-provoking music on endothelial vasoreactivity. *Psychosomatic Medicine, 72*(4), 354–356.

Nilsson, U. (2009). Soothing music can increase oxytocin levels during bed rest after open-heart surgery: A randomised control trial. *Journal of Clinical Nursing, 18*(15), 2153–2161.

Ooishi, Y., Mukai, H., Watanabe, K., Kawato, S., & Kashino, M. (2017). Increase in salivary oxytocin and decrease in salivary cortisol after listening to relaxing slow-tempo and exciting fast-tempo music. *PLOS ONE,12*(12), e0189075.

Pothoulaki, M., MacDonald, R., & Flowers, P. (2012). An interpretative phenomenological analysis of an improvisational music therapy program for cancer patients. *Journal of Music Therapy 49*(1), 45–67.

Ridder, H. M., Stige, B., Qvale, L. G., & Gold, C. (2013). Individual music therapy for agitation in dementia: An exploratory randomized controlled study. *Aging and Mental Health, 17*(6), 667–678.

Robarts, J. Z. (2000). Music therapy and adolescents with anorexia nervosa. *Nordic Journal of Music Therapy, 9*(1), 3–12.

Stern, D. N. (2010). *Forms of vitality exploring dynamic experience in psychology, the arts, psychotherapy and development,* New York: Oxford University Press.

Storz, D., Maes, R., von Kamp, J., Schumacher, K., & Martin, E. (2014). *The music in music therapy: Psychodynamic music therapy in Europe: Clinical, theoretical and research approaches*. London: Jessica Kingsley.

Szmedra, L., & Bacharach, D. W. (1998). Effect of music on perceived exertion, plasma lactate, norepinephrine and cardiovascular hemodynamics during treadmill running. *International Journal of Sports Medicine, 49,* 142–147.

Theorell, T. (2014). *Psychological health effects of musical experiences*. London: Springer.

Trevarthen, C. (2003). Neuroscience and intrinsic psychodynamics: Current knowledge and potential for therapy. In J. Corrigall & H. Wilkinson (Eds.), *Revolutionary connections: Psychotherapy and neuroscience* (pp. 53–78). London: Karnac Books; Taylor & Francis.

Trondalen, G. (2003). Self-listening in music therapy with a young woman suffering from anorexia nervosa. *Nordic Journal of Music Therapy, 12*(1), 3–17.

Trondalen, G., & Bonde, L.-O. (2012). Music therapy: Models and interventions. In R. A. R. MacDonald, G. Kreutz, & L. Mitchell (Eds.), *Music, health, and wellbeing* (pp. 40–62). Oxford, UK: Oxford University Press,

Uggla, L., Bonde, L. O., Hammar, U., Wrangsjö, B., & Gustafsson, B. (2018). Music therapy supported the health-related quality of life for children undergoing haematopoietic stem cell transplants. *Acta Paediatrica, 107*(11), 1986–1994.

Uggla, L., Bonde, L. O., Svahn, B. M., Remberger, M., Wrangsjö, B., & Gustafsson, B. (2016). Music therapy can lower the heart rates of severely sick children. *Acta Paediatrica, 105*(10), 1225–1230.

Ursin, H., & Eriksen, H. R. (2010). Cognitive activation theory of stress (CATS). *Neuroscience and Biobehavioral Reviews, 34*(6), 877–881.

Vickhoff, B., Åström, R., Theorell, T., & von Scheele, B. (2012). Musical piloerection. *Music and Medicine, 4*(2), 82–89.

Wheeler, B. L. (Ed.). (2015). *Music therapy handbook*. New York: Guilford Press.

Wigram, T., Saperston, B., & West, R. (1995). *The art and science of music therapy*. Reading, UK: Harwood Academic.

Wrangsjö, B., & Körlin, D. (1995). Guided imagery and music (GIM) as a psychotherapeutic method in psychiatry. *Journal of the Association for Music and Imagery, 4,* 79–92.

CHAPTER 21

Walk–Talk Exercise, Stress Resilience, and Mental Health

Cristy Phillips
Wesley E. Sime

Stress is a ubiquitous experience for human beings and is associated with activation of stress response systems. Although the physiological effects of acute stress are fundamental to survival, the negative effects of chronic stress on brain health have been well demonstrated by the plethora of psychiatric conditions associated with it. Clearly, chronic stress mediates the breakdown of brain, muscle, immune, endocrine, and cardiac systems. Chronic stress is synonymous with the challenges in contemporary times and often exists in the form of cognitive and social dysfunction and even results in low-grade inflammation. Because of its ill effects, the management of chronic stress represents a global challenge to health care.

Fortunately, research focusing on effective stress management has revealed the benefits of numerous cognitive, as well as physical, techniques as described by various chapters in this volume, but each approach confers limitations. On the one hand, psychotherapy and relaxation-based approaches (e.g., meditation, breathing, and mindfulness) decrease the frequency and duration by which maladaptive thoughts trigger the stress response but fail to elicit key resilience mechanisms that are inherent with simply engaging musculoskeletal movement. On the other hand, aerobic exercise engages musculoskeletal resilience mechanisms acutely but fails to mitigate the chronic effects of psychogenic stressors. To address the aforementioned limitations, we present a unique form of exercise therapy called walk–talk therapy. This therapeutic technique combines exercise therapy with psychotherapy in an attempt to provide clients with a more holistic program for stress management.

Walk–talk is similar to traditional psychotherapy, except clients are walking with the therapist while exploring issues. This type of therapy permits clients to engage in a way that often feels more comfortable than talking face-to-face in an intense manner in a traditional office setting. The walk–talk approach appears to be highly cathartic (i.e., it aids in "getting it all out") when the issues are complicated or entangled (Hays, 1999). By activating neuroprotective processes in the body and brain, the "walk" component confers resilience by enhancing psychophysiological, skeletal, endocrine, and immune function benefits. By eliminating maladaptive psychological expectancies and learned associations that trigger the stress response, the "talk" component facilitates learning and stress coping resilience. Thus walk-talk therapy deploys both bottom-up and top-down techniques that modulate stress. Given the growing interest in nonpharmacological methods to mitigate stress, we have refined a protocol that will enable clinicians to modulate central and peripheral neural pathways implicated in stress-related disorders.

Central to our model is the distinction between "bad stress" and "good stress," as well as between chronic or intermittent stress, respectively. This requires a deeper analysis of the neural mechanisms underlying both aerobic exercise and psychological stress. Accordingly, we provide a brief summary of the neuromodulatory underpinnings of how aerobic exercise acts as an intermittent stressor to promote neuroprotection, coping, and resilience. We suggest that aerobic exercise primes the brain to benefit from psychotherapy by triggering homeostatic and neuroplasticity mechanisms. We discuss a number of factors that are implicated in the latter processes, particularly modulation of hippocampal neurophysiology and neurochemistry, neurogenesis, brain-derived neurotrophic factor (BDNF), tryptophan metabolism, and immune and endocrine function. Given space limitations, we merely acknowledge that psychotherapy facilitates cognitive restructuring to mitigate the effects of maladaptive learning and refer the reader to relevant chapters in this volume for a more thorough description on the topic. We attempt to inform clinical practice by discussing important factors that should be considered with walk–talk therapy clients. We also hope that the presentation of the mechanisms (or physiological mediators to follow) will help future researchers design and analyze experiments and training programs of many forms of exercise, including walk–talk therapy, so that unresolved elements may be studied more effectively.

HISTORY OF THE METHOD

Recognizing the potential to leverage exercise for therapeutic effect, Thaddeus Kostrubala, a psychiatrist in San Diego, California, took many of his patients walking or running on the beach with him in the mid-1900s (Sime, 1981). This was the first documented use of a form of walk–talk therapy for stress management and mental health. Kostrubala demonstrated that the metabolic effects of physical exertion seem to spin off to psychological catharsis as well, such that verbal expression of emotionally charged past trauma experiences came forth more freely to enable more effective processing. This, we believe, is one of the early connections to the benefits of movement theories. Subsequently, many other health care professionals began to recommend aerobic exercise for their clients, and some clinical researchers documented the objective benefits of using an exercise prescription for two major stress symptoms, depression and anxiety (Sime, 1981, 1987, 2002). Therein, exercise appeared to help clients who repressed anger to become more assertive in their conversations as they explored issues on a deeper level.

Walk–talk therapy is now acknowledged as a relatively convenient and safe method of conducting exercise during traditional psychotherapy (Sime, 2002; Sime & Hellweg, 2001). The act of walking alongside of the client during brisk walking or running eliminates barriers and subtly helps clients to engage in difficult topics by eliminating the pressure of continuous eye contact (Sime, 2002). If the client is having difficulty formulating responses, then the combination of an informal atmosphere and having the option to gaze at a point of scenery or to focus on pace or rhythm of the walking stride seems to help facilitate more free and spontaneous responses. The approach appears to be highly cathartic when the issues are complicated or entangled (Hays, 1999). Moving forward to the present, it will be helpful to the reader to understand the many complex physiological changes that come about as a result of systematic exercise training that includes the highlighted process of walk–talk therapy.

PHYSIOLOGICAL MEDIATORS

Aerobic exercise is a stimulus that disrupts the physiological status of an organism; thus it is regarded as a physical stressor. When the brain recognizes physical stressors, it triggers the release of molecules in a complex chain of events to facilitate immediate adjustments in brain and body function. These adjustments occur as a result of responses in the sympathetic–adrenomedullary system (SAM), the hypothalamic–pituitary–adrenal axis (HPA), and the sympathetic and parasympathetic arms of the autonomic nervous system (ANS). Within seconds, physical stressors elicit the secretion of epinephrine and norepinephrine from the SAM. Within minutes, the HPA axis triggers a hormonal response that involves immune system activity and cortisol release, an event that results in an amplified and more protracted stress response. Concomitantly, physical stressors such as exercise activate brain structures that regulate the autonomic response: The brainstem registers stressors by sensing increased activity in type III and IV muscle afferents and rhythmic shifts in heart rate that are mediated in part by the vagus nerve, changes that alter sympathetic and parasympathetic activities in the ANS. The body's response to stress prepares the body for physical exertion, whether it be fleeing a threat or exercising. Interestingly, many of the modulatory processes triggered by stress are similar for physical, cognitive, and emotional stress. By design, modulatory processes occur to restore basal homeostatic levels as soon as the presence of the stressor abates, and, therefore, the prestimulation activation level of the ANS tunes and predicts the net response to stress (Weybrew, 1963). Therein markers of autonomic resilience (e.g., vagally mediated heart rate variability) indicate the degree to which the top-down processes alter peripheral response to stress, with higher levels of stress resilience being positively correlated with higher heart rate variability, making the latter a key biomarker for prevention and treatment programs.

Aerobic exercise is regarded as beneficial to mental and physical health and often touted as a preventative or therapeutic to stress-related illness. Generally speaking, physically active people show reduced reactivity to life, physical, and mental stress. Moreover, regular physical activity contributes to a reduction in susceptibility for stress-related disorders. Partially underlying enhanced resilience is the fact that regular aerobic exercise attenuates cortisol, epinephrine, and norepinephrine responses and modulates other stress correlates. Studies that have shown such changes are listed in Table 21.1. Moreover, physically active individuals show reduced cardiovascular reactivity by mechanisms that involve increased parasympathetic nervous system activity.

Exercise Effects on the Hippocampus

The hippocampus is a region of the brain that modulates learning, memory, autonomic regulation, emotional function, and feeding behavior. It generates synchronous activity between its neurons and other regions in a way that promotes sensory binding, memory retrieval, and learning. Also, the hippocampus is a primary site for neuroplasticity in the brain.

The hippocampus is highly responsive to stress given its high receptor density (e.g., mineralocorticoid and glucocorticoid receptors) for stress hormones. Mineralocorticoid receptors are mostly found in the hippocampus, whereas glucocorticoid receptors are found in the hippocampus, amygdala, and prefrontal cortex. Functionally speaking, mineralocorticoid receptors in the hippocampus play a pivotal role in feedback regulation of the HPA axis, but glucocorticoid receptors in the hippocampus regulate genes related to development, metabolism, and immune function.

Generally speaking, mild to moderate levels of intermittent stress positively affect hippocampal structure and function, but severe or chronic stress adversely affects hippocampal structure and function. An inability of the hippocampus to function appropriately contributes to the persistent sympathetic nerve activity that is found in stress-related disorders. The scale of significance of excessive sympathetic nerve activity becomes clear when one considers the prevalence of mortality rates of stress-related conditions in industrialized nations (Lloyd-Jones et al., 2009), suggesting the hippocampus is a key factor for mitigating stress-related changes that contribute to ANS dysfunction and alterations in cortisol exposure. Evidence suggests that the modulatory insufficiency that results from ANS dysfunction or deleterious alterations in cortisol levels can produce autoimmune, allergic, asthmatic, or increased susceptibility to infection (Lehrer, personal communication, November 12, 2019).

By acting as an intermittent stressor, regular aerobic exercise of moderate intensity has been shown to exert robust, positive effects on the hippocampus and may mitigate or prevent autonomic dysfunction. Studies of the neuroprotective effects of regular aerobic exercise on the brain show robust effects in emotional function, cognitive function, and autonomic regulation—changes that can be attributed to exercise-induced changes in the structure and function of the hippocampus (also known as changes in neuroplasticity). The hippocampus is a primary site for neuroplasticity in the brain. Exercise positively affects neuroplasticity such that high levels of exercise result in greater hippocampal size and optimized function in rodents (van Praag, 2008) and humans (Firth et al., 2018), changes that putatively translate to enhanced regulation of autonomic function. One recent meta-analytic review of 737 participants showed that aerobic exercise was associated with greater left hippocampal volume in comparison to controls (Firth et al., 2018). Several neurophysiological changes underlie these neuroplastic changes, as highlighted in Figure 21.1.

Exercise and Brain-Wave Activity

Aerobic exercise alters gamma- (Chen et al., 2011) and theta-wave activity in the hippocampus in real time in a speed- and duration-dependent manner (Bland & Oddie, 2001; Buzsaki, 2002; Li, Kuo, Hsieh, & Yang, 2012). Gamma- and theta-wave activity interacts in the brain in a way that modulates neural activity in the cortex and hippocampus to facilitate attention, sensory binding, working memory, learning, and awareness. Preclinical work shows that mice that run faster show a dramatic increase in the strength of gamma rhythm (Chen et al., 2011), suggesting a correlation between movement and learning. Mechanistically speaking, it seems plausible that slower speeds favor a situation wherein synchronous activity between the cornu ammonis 3 (CA3) region of the hippocampus (that forms memories related to location awareness and episodic memory) and entorhinal cortex converge onto the CA1 region (the primary output region) to facilitate

TABLE 21.1. Summary of Research on the Effects of Exercise on Correlates of Stress

Outcome of exercise	Study	Design	Clinical significance	Population	Evidence
Decreased reactivity to life stress	Tucker, Cole, & Friedman (1986)	Correlational	Physically active people perceived less distress than unfit people.	4,628 men	Probably efficacious
Decreased reactivity to life stress	Luger et al. (1987)	Experimental wherein plasma ACTH, cortisol, and lactate responses were measured following treadmill exercise (50, 70, and 90% of maximal oxygen uptake) and administration of intravenous CRH hormone (1 microgram per kilogram of body weight)	Exercise-stimulated ACTH, cortisol, and lactate responses were attenuated in the trained participants when plotted against applied absolute workload.	21 men who were sedentary, moderately trained runners, or highly trained runners	
Decreased reactivity to life stress	Deuster et al. (1989)	Experimental intervention wherein the relation between graded exercise, sympathetic nervous system, and metabolic response were analyzed	Exercise intensity training related to sympathetic and metabolic response to stress in that hormonal and metabolic responses became more efficient. With increased training, greater workloads were attainable at equivalent increments in hormonal and metabolic indices.	21 untrained, moderately trained, and highly trained middle-aged men	
Decreased reactivity to life stress	Steptoe, Edwards, Moses, & Mathews (1989)	Experimental aerobic exercise program	Increases in level of physical activity resulted in reductions in mental tension and anxiety.	193 adults with low reported level of physical activity	
Decreased reactivity to mental stress	Claytor (1991)	Experimental procedure measuring cardiovascular function and plasma catecholamine levels	Trained individuals maintain lower heart rate in response to mental stress.	43 college-age males	Probably efficacious
Decreased reactivity to mental stress	Sothmann, Hart, & Horn (1991)	Experimental procedure measuring resting and absolute noradrenaline concentrations prior to and following aerobic exercise training program	Healthy men with relatively high noradrenaline experience a modest reduction in basal circulating plasma norepinephrine, resulting in lower absolute concentrations during an acute stress with exercise training.	16 middle-aged men	

Decreased reactivity to mental stress	von Haaren et al. (2016)	Randomized controlled trial examining the effects of aerobic exercise, heart rate variability, and perceived stress during academic examination	Exercise appears to be a useful preventive strategy to buffer the effects of stress on the ANS.	61 inactive male young adults	
Decreased autonomic and cardiovascular reactivity after mental stress	Spalding, Lyon, Steel, & Hatfield (2004)	Experimental procedure examining effects of aerobic training, weight training, or a no-treatment condition to determine whether training lowers systolic and diastolic blood pressure, heart rate, and rate-pressure product during rest, psychological stress, and recovery periods	Aerobic training lowered cardiovascular activity levels during psychological stress and recovery in healthy young adults, implying a protective role against age-related increases in coronary heart disease.	45 sedentary young adult males	
Decreased cardiovascular reactivity after mental stress	Rimmele et al. (2007)	Experimental intervention in which participants were exposed to psychological stress and salivary free cortisol levels, heart rate, and psychological responses were repeatedly measured before and after stress exposure	Trained men exhibited significantly lower cortisol and heart rate responses to the stressor compared with untrained men. In addition, trained men showed significantly higher calmness and better mood and a trend toward lower state anxiety during the stress protocol.	44 young adults trained and untrained males	
Decreased autonomic and cardiovascular reactivity after mental stress	Sinyor, Schwartz, Peronnet, Brisson, & Seraganian (1983)	Experimental intervention in which participants were exposed to psychological stress and heart rate, catecholamines, cortisol, and self-reports of arousal and anxiety were measured during and after stress exposure	Trained participants showed higher levels of norepinephrine early in the stress period, more rapid heart rate recovery following stress, and lower levels of anxiety.	30 young adult trained and untrained males	
Optimized cortisol levels in response to stress	Rimmele et al. (2007)	Experimental intervention in which participants were exposed to psychological stress and salivary free cortisol levels, heart rate, and psychological responses were repeatedly measured before and after stress exposure	Trained men exhibited significantly lower cortisol and heart rate responses to the stressor compared with untrained men. In addition, trained men showed significantly higher calmness and better mood and a trend toward lower state anxiety during the stress protocol.	44 young adult trained and untrained males	Probably efficacious

(continued)

TABLE 21.1. *(continued)*

Outcome of exercise	Study	Design	Clinical significance	Population	Evidence
Optimized cortisol levels in response to stress	Traustadottir, Bosch, & Matt (2005)	Experimental intervention wherein participants were exposed to psychological stress and heart rate, blood pressure, and cortisol levels were measured	Aging is associated with greater HPA axis reactivity to psychological stress, and higher aerobic fitness among older women can attenuate these age-related changes as indicated by a blunted cortisol response to psychological stress.	36 women classified as young-unfit, older-unfit, and older-fit	
Optimized cortisol levels in response to stress	Corazza et al. (2014)	Systematic review	Regular exercise appears to have a positive influence on serum cortisol level in older people, causing a decrease in concentration, which may help prevent comorbidities.	Older adults aged ≥ 60 years	
Hippocampal size	Erickson et al. (2011)	Randomized controlled trial wherein aerobic exercise training increased the size of the anterior hippocampus	Moderate aerobic exercise increased size of hippocampus by 2% in contrast to controls, a reversal that mitigated 1–2 years of age-related loss in hippocampal volume.	120 healthy community-dwelling older adults	Probably efficacious
Hippocampal size	Makizako et al. (2015)	Correlational study wherein participants were instructed to wear the accelerometer on an elastic band on their hips at all times for 2 weeks. Average daily duration of light, moderate, and total exercise (min/day) was calculated.	Moderate exercise was associated with hippocampal volume after controlling for age, but light exercise and total exercise were not. Moreover, hippocampal volume loss was significantly and directly associated with poor memory performance, suggesting that hippocampal volume was the link between moderate exercise and memory augmentation in people with MCI and that longer durations of moderate exercise could result in increased hippocampal volume and improved memory.	310 older adults with mild cognitive impairment who completed neuropsychological tests of memory, and structural magnetic resonance	

Hippocampal size	Pajonk et al. (2010)	Randomized controlled trial in which participants were exposed to aerobic exercise training and/or playing table football (control group) for a period of 3 months	Following exercise training, hippocampal volume increased significantly in patients with schizophrenia (12%) and healthy subjects (16%), with no change in the nonexercise group of patients (-1%). Changes in hippocampal volume in the exercise group were correlated with improvements in aerobic fitness measured by change in maximum oxygen consumption.	24 males, 8 of whom were controls, 8 patients with schizophrenia who exercised, and 8 patients with schizophrenia who were nonexercisers	
Hippocampal size	Siddarth et al. (2018)	Correlational examination of activity level with neuropsychological test and magnetic resonance imaging results	The higher exercise group had a thicker parahippocampal cortex compared to the lower exercise group. The higher exercise group also exhibited superior performance in attention and information-processing speed and executive functioning.	26 patients aged ≥ 60 years, with memory complaints	
Hippocampal size	Ott, Johnson, Macoveanu, & Miskowiak (2019)	A systematic review that analyzed clinical trials that investigated the effects of interventions on hippocampal measurements taken with functional magnetic resonance imaging	Hippocampal volume improvements after various treatments were associated with cognitive improvement, suggesting hippocampal size as a biomarker for pro-cognitive effects.	5 studies that included 10–40 participants with a diagnosis of depression, bipolar disorder, and schizophrenia	
BDNF	Szuhany, Bugatti, & Otto (2015)	Meta-analysis that examined the effects of exercise and increased BDNF levels in humans across multiple exercise programs	Results demonstrated a moderate effect size for increases in BDNF following a single session of exercise; regular exercise intensified the effect of a session of exercise on BDNF levels; and regular exercise exerted a small effect on resting BDNF levels.	1,111 individuals who were studied after participation in a single session of exercise, a session of exercise following a program of regular exercise, or at rest following regular activity	Probably efficacious

(continued)

TABLE 21.1. *(continued)*

Outcome of exercise	Study	Design	Clinical significance	Population	Evidence
BDNF	Dinoff et al. (2016)	A meta-analysis that examined the effects of exercise interventions on peripheral BDNF	Evidence suggests an increase in resting peripheral BDNF concentrations after aerobic exercise training interventions but not after resistance training interventions.	910 individuals who completed an exercise intervention ≥ 2 weeks and at ≥ 50% of peak oxygen uptake, or if exercise intensity was not reported, exercise described as running, cycling, or resistance training	
BDNF	Huang, Erlichman, & Dean (1997)	A review of the effects of exercise on peripheral BDNF or cardiorespiratory fitness	Peripheral BDNF concentrations were elevated significantly in response to acute and chronic aerobic exercise.	417 healthy adults	
BDNF	Knaepen, Goekint, Heyman, & Meeusen(2010)	A review of the effects of an exercise intervention (i.e., an acute aerobic, an acute strength, regular aerobic, or regular strength training program) or cardiorespiratory fitness status on peripheral BDNF levels	69% of the studies in healthy participants and 86% of the studies in persons with a chronic disease or disability showed a transient increase in serum or plasma BDNF concentration following acute aerobic exercise.	680 adults who were diagnosed as depressed, obese, having multiple sclerosis or mild cognitive impairment, or healthy	
Kynurenine metabolism	Lewis et al. (2010)	Experimental study designed to assess the effects of exercise on kynurenine metabolism in humans	Endurance exercise in form of marathon run led to an increase of kynurenic acid in peripheral blood.	25 healthy adults	Probably efficacious
Kynurenine metabolism	Schlittler et al. (2016)	Experimental study designed to assess the effects of exercise on kynurenine metabolism in humans	Endurance exercise of 150 km road cycling increased the expression of KATs and PGC-1α in muscles and led to an increase of kynurenic acid in peripheral blood, which was observed 1 h after the exercise, but returned to baseline levels as quickly as 5 hours later.	17 active males, half of whom were participating in endurance training	

Kynurenine metabolism	Strasser et al. (2016)	Experimental study that examined the effect of exhaustive aerobic exercise on biomarkers of tryptophan metabolism in trained athletes	Intense exercise was associated with a 6% increase in serum kynurenine after an incremental cycle ergometer exercise test until exhaustion.	33 trained adults	
PGC-1α	Pilegaard, Saltin, & Neufer (2003)	Experimental study that examined whether PGC-1α transcription is regulated by acute exercise and exercise training in human skeletal muscle. Participants performed 4 weeks of one-legged knee extensor exercise training. At the end of training, participants completed 3 h of two-legged knee extensor exercise. Biopsies were obtained from the vastus lateralis muscle of both the untrained and trained legs before exercise and after 0, 2, 6, and 24 hours of recovery	Exercise induced a marked transient increase in PGC-1α transcription (10- to > 40-fold) and mRNA content (7- to 10-fold), peaking within 2 hours after exercise.	7 healthy physically active adult males	Probably efficacious
PGC-1α	Vargas-Ortiz et al. (2015)	Experimental study that analyzed the effect of aerobic training on PGC-1α in skeletal muscle of overweight adolescents without change in caloric intake	Aerobic training increased PGC-1α expression levels in sedentary, overweight, or obese adolescents.	14 overweight or obese male adolescents	
PGC-1α	Lysenko, Popov, Vepkhvadze, Lednev, & Vinogradova (2016)	Gene expression and protein level were evaluated in samples from m. vastus lateralis before, 40 min, 5h, and 22 h after the aerobic exercise (performed 70-min bicycle intermittent exercise with both legs, followed by one-leg strength exercise (ES: 4 bouts of knee extensions at 75% MVC till exhaustion))	Expression of PGC-lα increased in both legs.	9 amateur endurance-trained young adults	
PGC-1α	Lysenko, Vepkhvadze, Lednev, Vinogradova, & Popov (2018)	Performed a cycling bout with and without branched chain amino acids	Expression of PGC-lα increased, but postexercise ingestion of branched-chain amino acids partially suppresses endurance exercise-induced expression of PGC-1α mRNA.	9 endurance-trained young adults	

(continued)

TABLE 21.1. *(continued)*

Outcome of exercise	Study	Design	Clinical significance	Population	Evidence
Skeletal muscle plasticity	Dickinson et al. (2018)	Experimental study that identifies the unique transcriptome response of skeletal muscle to acute aerobic exercise (40 min of cycling, 70% maximal heart rate)	48 genes were differentially expressed following aerobic exercise, including ones involved in angiogenesis and epigenetic responses.	6 healthy, recreationally active young men	Probably efficacious
Skeletal muscle plasticity	Lundberg, Fernandez-Gonzalo, Tesch, Rullman, & Gustafsson (2016)	Experimental study that explored the effects of an acute aerobic exercise bout on the transcriptional response to subsequent resistance exercise	176 genes were up- (127) or down-regulated (49) with a combination of aerobic and resistance exercise in comparison to resistance exercise only.	10 moderately trained men	
Skeletal muscle plasticity	Konopka, Trappe, Jemiolo, Trappe, & Harber (2011)	Experimental study that assessed the influence of aerobic training on muscle size and function after 3 months of training	Training elevated plasticity in muscle such that there was a shift toward an oxidative phenotype that appears beneficial for metabolic and functional health in older individuals.	8 sedentary older women	
Skeletal muscle plasticity	Harber et al. (2009)	Experimental study that assessed the influence of aerobic training on muscle size and function after 3 months of cycle ergometer training	Quadriceps muscle volume, determined by magnetic resonance imaging, was 12% greater after training, and knee extensor power increased 55% and remodeling of fibers occurred at the myofiber level, showing that aerobic exercise reduced age-related sarcopenia.	7 older women	
Optimized neurotransmitter level and function	Strasser et al. (2016)	Experimental study that examined the effect of exhaustive aerobic exercise on biomarkers of tryptophan metabolism in trained athletes	Exhaustive aerobic exercise is associated with alterations in monoamine metabolism.	33 trained adults	Probably efficacious
Optimized neurotransmitter level and function	Melancon, Lorrain, & Dionne (2014)	Experimental intervention wherein participants exercised for 1 hour (67–70% peak oxygen consumption)	Both pre- and posttraining exercise challenges markedly increased elevations in plasma tryptophan	16 healthy senior men	

		at baseline and following 16 weeks of aerobic training	availability to the brain in older men, a change known to impact serotonin synthesis.		
Optimized neurotransmitter level and function	Legakis et al. (2000)	Experimental investigation of the effect of a 20-min exercise on plasma human galanin. Postexercise levels were recorded after termination of the stressful test and 15 min thereafter.	Galanin hyperpolarizes noradrenergic neurons and inhibits locus coeruleus neuronal firing, leading to a suppression of noradrenaline release. Peripheral levels of blood concentration of galanin increased after acute exercise, a change thought to suppress noradrenaline release.	13 middle-aged healthy volunteer adults	
Anti-inflammatory	Tartibian, Hajizadeh Maleki, Kanaley, & Sadeghi (2011)	Experimental investigation of the effect of aerobic exercise on immune markers in comparison to usual aerobic activity levels	Increasing aerobic exercise for 3 months resulted in a lowering of IL-6, IL-1, and TNF-α.	38 older adults	Efficacious
Anti-inflammatory	Abdollahpour, Khosravi, Eskandari, & Haghighat (2017)	Experimental investigation of the effect of aerobic exercise on immune markers in comparison to usual aerobic activity levels	Increasing aerobic exercise to recommended public health dosage (150 minutes for 6 months at 70–80% max resting heart rate) lowered basal levels of IL-6.	41 sedentary postmenopausal women	
Anti-inflammatory	Tartibian, FitzGerald, Azadpour, & Maleki (2015)	Experimental investigation of the effect of aerobic exercise on immune markers in comparison to usual aerobic activity levels	Increasing aerobic exercise for 4 months resulted in a lowering of IL-6, IL-1, and TNF-α.	28 sedentary postmenopausal women	
Anti-inflammatory	Hayashino et al. (2014)	Meta-analytic review that evaluated the ability of exercise to reduce C-reactive protein levels in patients with type 2 diabetes	Exercise interventions reduced inflammatory cytokine (C-reactive protein and IL-6).	824 patients with diabetes	
Anti-inflammatory	Meneses-Echavez et al. (2016)	Meta-analytic review that evaluated the ability of exercise to reduce IL-6, TNF-α, IL-8, and IL-2	Exercise reduced the serum concentrations of IL-6, TNF-α, IL-8, and IL-2.	478 patients with a history of breast cancer	

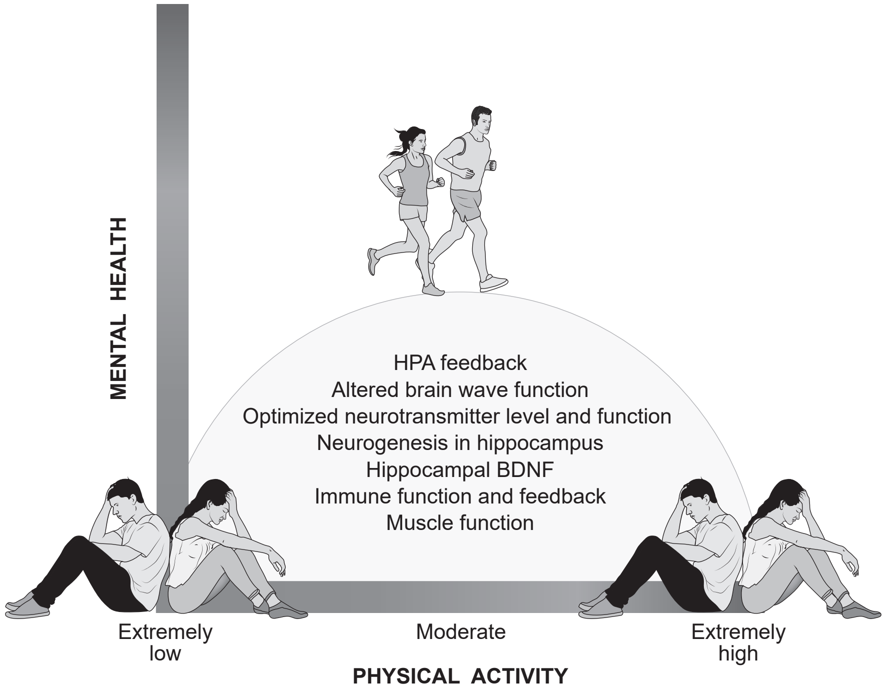


FIGURE 21.1. Regular aerobic exercise optimizes brain and mind function. Moderate-intensity aerobic exercise alters neuropsychophysiological processes in a dose-dependent manner. Acutely, aerobic exercise boosts mood by altering neurotransmitter signaling and brain-wave function. Over time, aerobic exercise optimizes feedback of the stress axis and, in turn, reduces exposure of the brain and body to circulating stress hormones. Consequently, aerobic exercise optimizes common neuroplasticity substrates and confers resiliency to stress-related diseases, particularly mental illness.

learning (Chen et al., 2011), which may explain why some people think better while walking, particularly when walking facilitates a range of physical stress that is within the mild to moderate range. Nevertheless, running at faster speeds requires faster processing and learning. Thereby, speed-associated increases in gamma-wave activity more efficiently synchronize brain activity to promote rapid thinking and perception (Bartsch, Dohring, Rohr, Jansen, & Deuschl, 2011; Chen et al., 2011) by processes that involve the synchronization of hippocampal activity with the frontal cortex (Li, Long, & Yang, 2015). A key assumption of the latter notion is that activity-induced alterations in gamma activity in the hippocampus interact with different types of theta waves in the frontal cortex in a process called *coupling* to alter information separation and retrieval (Alekseichuk, Turi, Amador, Antal, & Paulus, 2016). Ultimately, different patterns of coupling between brain regions represent various patterns of functional connectivity in the brain and, in turn, result in different behaviors. Thus it seems plausible that speed-induced alterations in gamma activity in the hippocampus can produce different coupling patterns with the frontal cortex to yield different thinking patterns during walking and running.

Movement-induced changes in brain-wave activity result in part from alterations in neurotransmitter level and function. Aerobic exercise optimizes neurotransmitter level and function in the hippocampus by altering the synthesis, metabolism, and release of serotonin, norepinephrine, dopamine, glutamate, gamma-aminobutyric acid (GABA), and acetylcholine. Serotonin is an excitatory neurotransmitter that is implicated in mood, cognition, arousal, and autonomic function; its concentration levels in the brain increase with acute stress but decrease with chronic stress. Similarly, norepinephrine and dopamine participate in the sympathetic response to stress, whereas acetylcholine participates in the parasympathetic response. Glutamate and GABA modulate the HPA axis via their excitatory and inhibitory influences, respectively. Optimization of neurotransmitter level and function is important for keeping the balance between neuronal excitation and inhibition that is required for neuronal health. Moreover, the optimization of neurotransmitters promotes more efficient communication between neurons, particularly in those that comprise emotional, cognitive, and motor circuits via interactions with BDNF.

Effects of Exercise on BDNF

Belonging to a family of growth factors, BDNF plays a vital role in the maintenance of synaptic function, metabolic programming, and neuronal health—functions that are particularly important in the hippocampus (Autry & Monteggia, 2012). Studies suggest that peripheral levels of BDNF correlate with brain health and decrements correlate with psychiatric (Phillips & Fahimi, 2018) and metabolic (Krabbe et al., 2007) disorders. Notably, aerobic exercise increases BDNF levels in the hippocampus and periphery (Huang, Larsen, Ried-Larsen, Moller, & Andersen, 2014; Noble et al., 2014), changes that contribute to synaptogenesis and prevent neuronal loss. The latter effect is modulated by changes in the GABAergic system, which alters excitability of neurons (Chen et al., 2011; Rico, Xu, & Reichardt, 2002) and decreases the propensity for excessive neuronal activation and damage during times of high stress.

"Aftereffects" of Exercise

Following exercise cessation, there is an abrupt removal of control by higher brain centers and abolished feedback from muscle mechanoreceptors that reset the arterial baroreflex to a lower level to cause an initial decrease in heart rate. As recovery continues, a more gradual cardio-deceleration is observed, likely mediated by parasympathetic reactivation and sympathetic withdrawal. These autonomic adjustments appear to be elicited by the gradual clearance of postexercise metabolites and the reduction of circulating catecholamines and cortisol, as well as thermoregulatory factors. Lingering neurotransmitter and neurotrophic changes in the brain gradually alter the rate of hippocampal neurogenesis in a factor-specific manner. Modulation of neurogenesis by these factors constitutes a key form of neuroplasticity in the adult brain.

Exercise and Neurogenesis

Neurogenesis refers to the brain's ability to make new neurons in the adult hippocampus. Aerobic exercise coaxes the division of stem cells in the hippocampus during the process of neurogenesis. Following division, some of the newly born neurons make their way into parts of the brain that regulate memory, cognition, emotion, and stress regulation. This process is important because it increases neural reserve in the hippocampus to promote resilience or recovery from injury. Just 4 weeks of regular aerobic exercise can reverse deficits in neurogenesis found in persons with chronic stress, inflammation, and depression—changes that reify as increased hippocampal volume and function and improved stress regulation. Improved hippocampal health and function is important because hippocampal neurons that project to the hypothalamus inhibit the release of regulatory stress hormones such as corticotropin-releasing hormone (CRH) to dampen the stress response when the stressor abates (Radley & Sawchenko, 2011); in turn, alterations in CRH release alter the release of corticosteroids. An inability of the hippocampus to function appropriately causes persistent sympathetic nerve activity found in stress-related disorders. The scale of significance of excessive sympathetic nerve activity becomes clear when one considers the prevalence and mortality rates of stress-related conditions in industrialized nations (Lloyd-Jones et al., 2009), suggesting the hippocampus is a key factor for mitigating stress-related changes that contribute to ANS dysfunction and excess cortisol exposure.

Notably, aerobic exercise also influences neurogenesis levels by increasing proliferation of microglia levels in the hippocampus (Ehninger & Kempermann, 2003). In vitro experiments show that the viability of newly born neurons in the hippocampus is dependent upon microglial function and exposure to proinflammatory cytokines (Vukovic, Colditz, Blackmore, Ruitenberg, & Bartlett, 2012). Microglia taken from exercising mice stimulate stem cell activities in cell culture preparations derived from sedentary mice (Vukovic et al., 2012) to increase levels of neurogenesis.

Exercise and Tryptophan Metabolism

Another key mechanism by which aerobic exercise exerts neuroprotection involves modulation of tryptophan metabolism. Serotonin is made from tryptophan, and, therefore, alterations in tryptophan metabolism can alter serotonin levels in the brain. This is problematic because decreased serotonergic signaling in the brain contributes to morbidity symptoms of depression (e.g., low mood, loss of pleasure, weight loss or gain, low energy, and feelings of hopelessness), specifically because these are manifestations of stress-induced depression. Cortisol causes the breakdown of tryptophan to the metabolite kynurenine during times of stress and inflammation. The breakdown of tryptophan decreases serotonin synthesis in the brain to contribute to mood, sleep, and behavioral abnormalities. Also, the breakdown of tryptophan produces several neuroactive metabolites that contribute to excessive neuronal activity, energy depletion, and neuronal damage.

Activation of the kynurenine pathway is one mechanism by which stress and stress-induced inflammation interfere with tryptophan metabolism and alter brain function. Aerobic exercise prevents the shunting of tryptophan toward kynurenine metabolism and prevents excessive neuronal activity and brain dysfunction by increasing the expression of an enzyme in skeletal muscle called peroxisome proliferator-activated receptor-gamma coactivator (Pgc)-1α. The Pgc-1α enzyme creates conditions in the body that protect the brain against stress-related conditions.

Effects of Prolonged, High-Intensity Exercise

Mechanistically, aerobic exercise that requires prolonged and high metabolic demand increases the PGC1α-dependent expression of kynurenine aminotransferase (KAT) enzymes in muscles (Agudelo et al., 2014; Ruas et al., 2012; Schlittler et al., 2016). KATs work to clear kynurenine from circulation, partly by shifting peripheral metabolism toward the production of kynurenic acid, a metabolite that cannot cross the blood–brain barrier. Specifically, aerobic endurance exercise causes a transient increase in the neuroprotective metabolites of kynurenine (i.e., kynurenic acid) by 63% and a reduction in the neurotoxic metabolites (i.e., quinolinic acid) by 19% (Schlittler et al., 2016). Increased production of kynurenic acid positively affects mental health by preventing kynurenine accumulation in the brain and reducing stress-induced neuropathology.

Concomitantly, KATs increase the expenditure of adipocytes (i.e., fat storage cells) and promote an anti-inflammatory bias in the periphery (Agudelo et al., 2018), changes that can accrue rather quickly. Translational studies show that exercise induces shifts in skeletal gene expression that improve stress resilience, with changes in KATs occurring after a 3-month training regimen (Mahoney & Tarnopolsky, 2005; Ruas et al., 2012; Schmitt et al., 2003).

Exercise and Interleukin-6

Another neuroprotective factor modulated by aerobic exercise is interleukin (IL)-6. During exercise, muscle contractions elicit the production of cell signaling molecules called myokines. These myokines mediate their exercise effects by communicating with the cells that produce them (autocrine), with nearby cells (paracrine), and with distant organs (endocrine). Thereby, myokines regulate brain structure and function, stress resilience, learning and memory, locomotor activity, and feeding behavior. IL-6 is a well-known myokine that is released by skeletal muscles and paradoxically acts as a pro- and anti-inflammatory factor depending upon the stimuli, the profile of other immune factors, and cellular context.

Persistently elevated levels of basal IL-6 levels are proinflammatory and play a role in the pathologies associated with physical inactivity, obesity, metabolic syndrome, and neurodegenerative and neuropsychiatric conditions. However, the transient release of IL-6 (up to 100-fold) by trained muscles elicits the breakdown of fats and facilitates insulin sensitivity—aggregate effects that can lower basal levels of inflammation over time and prevent neuronal damage or neurodegeneration in the brain.

Because IL-6 is modulated by the amount of carbohydrate availability, it appears to function as an energy sensor. Thus IL-6 levels also increase in the presence of chronic stress, pathogenic invasion, and tissue injury. Persistently elevated levels of IL-6 contribute to allostatic overload in stress-related illness, whereas aerobic exercise transiently elicits an increase in IL-6 in the short term to power adaptive mechanisms that confer stress resilience.

Exercise and Cognitive Deficits

The ability of aerobic exercise to attenuate HPA dysregulation, kynurenine shunting, and inflammation is important for preventing hippocampal atrophy and reversing cognitive deficits in persons with stress-related disorders such as mental illness, changes that negatively affect cognitive function. Neurons that are persistently exposed to inflammation and elevated glucocorticoids retract their dendrites and exhibit fewer dendritic spines. This effect is significant given that persons with stress-related illness are vulnerable to hippocampal atrophy and dysfunction. Similarly, persons who are aging tend to exhibit higher levels of inflammation and, by corollary, increased cortisol exposure to the detriment of neuronal structure and function in the hippocampus. Notwithstanding, the degree of dendritic branching in hippocampal neurons and the overall number of dendritic spines increases when animals are exposed to voluntary wheel running (Eadie, Redila, & Christie, 2005), suggesting that exercise can reverse stress-related hippocampal deficits that contribute to cognitive dysfunction. Further underscoring the importance of these effects is the fact that antidepressant medication optimizes dendritic tree structure and enhances neuroplasticity. As such, exercise in conjunction with this medication may optimize benefits well beyond previous clinical understanding.

So might aerobic exercise mitigate hippocampal exposure to stress hormones to prevent neuropathology in the hippocampus? One translational study revealed that 8 weeks of exercise improved depressive symptoms and levels of urinary cortisol (Nabkasorn et al., 2006). Another demonstrated that 12 weeks of high-intensity aerobic exercise enhanced mood and optimized responsiveness of the HPA to the dexamethasone responsiveness test in persons experiencing chronic pain (Chatzitheodorou, Mavromoustakos, & Milioti, 2008). Altogether, these studies suggest that moderate levels of exercise at sufficient intensity do, in fact, mitigate skeletal, immune, and neuroendocrine dysregulation in persons under stress and, thereby, hippocampal protection, an adaptive state that is likely characterized by increased vagal activity, neurotransmitter optimization, and antipain factors.

Thus exercise triggers the whole system in a moderated way to promote a stronger adaptive response to physical stress and make it more sensitive over time, promoting mental and physical resilience. Conversely, repeated bouts of extreme exercise over time can serve as a chronic stressor and deplete allostatic resources, contributing to stress-related illness.

CLINICAL DISORDERS AMENABLE TO EXERCISE THERAPY BENEFITS

We have concluded that the listing of clinical disorders that have valid evidence to support the use of exercise therapy is voluminous. However, we have set the bar high for the level of empirical evidence needed to support advocating for exercise and, in particular, for walk–talk therapy. This evidence is summarized in Table 21.2 wherein we provide a rationale and impetus for evaluating the therapeutic effects of individualized structured therapy for patients with the following challenges: major depressive disorder, bipolar disorder, generalized anxiety disorder, substance abuse disorder, eating disorders, schizophrenia, and attention deficit/hyperactivity disorder (ADHD).

Depression

Depression, in a clinically significant form known as major depressive disorder, is a common psychiatric condition characterized by low mood, helplessness, self-devaluation, lack of interest in activities that were once pleasant, and alterations in energy, appetite, and cognition—changes that have occurred for a minimum of 2 weeks. Moreover, depression is associated with adverse cardiovascular and metabolic outcomes, as well as an impaired stress response. The two main treatments for depression include administration of antidepressants and psychotherapy. Although helpful, extant treatments fail to provide full relief in a significant proportion of individuals.

A bevy of evidence shows that supervised aerobic exercise is efficacious and specific when used as a first-line treatment or adjunctive for people with mild to moderate depression. A report on the effects of exercise in depression conducted by the Mental Health Foundation in England provides evidence that exercise can be as successful at treating mild and moderate depression as antidepressants in the short term (4 months) and more effective in the long term (10 months; Halliwell, 2005). A meta-analysis by Mead and colleagues (2009) showed that the effect of exercise was as efficacious as cognitive-behavioral therapy (Mead et al., 2009), but failed to articulate that mechanistic differences exist between the two methods. Whereas aerobic endurance exercise causes a transient increase in KATs and reduces stress-related excitotoxic damage to neurons in the brain, CBT aims to eliminate thoughts that trigger the stress response, suggesting a need for skilled analysis of depressive causation and therapeutic application of rehabilitative technique. This data supports the National Institute for Clinical Exercise guideline that recommends that clinicians treating depression in primary and secondary care advise patients of all ages of the benefits of a structured and supervised exercise program, typically up to three sessions per week of moderate duration (45–60 minutes) for between 10 and 12 weeks (National Institute for Clinical Excellence, 2004), but acknowledges the fact that skilled handling of the stress response is a lifelong process that necessarily considers both mental and physical strategies. Thus, we recommend that patients of all ages be apprised of the latter, as failure to help patients adopt a more comprehensive strategy may predispose them to relapse. Interestingly, the notion that people with depression are lethargic and disinclined to exercise has been challenged by work showing that exercise adherence rates are often as high as the rates for adherence to medication (Halliwell, 2005). However, given the difficulty that individuals with depression have in getting started with an exercise program, our proposed walk–talk therapy model makes good clinical sense. Focus should also include encouraging exercise habits over time, given the cyclical nature of the disorder and the fact that exercise diminishes risk of relapse (Babyak et al., 2000).

Bipolar Depression

Bipolar disorder is a prevalent, chronic mental illness that consists of recurrent mood disturbances ranging from elevated (manic) to low (depressive) moods and cognitive dysfunction. People with bipolar disorder commonly exhibit a sedentary lifestyle (Chwastiak, Rosenheck, & Kazis, 2011), reduced functional exercise capacity (Vancampfort, Sienaert, et al., 2015), and a higher incidence of inflammation (Drago, Crisafulli, Calabro, & Serretti, 2015). They also show increased rates of hyperglycemia, dyslipidemia, hypertension, and metabolic disorder (Guan et al., 2010), underscoring the fact that this diagnostic group struggles to maintain both mental and physical health. Current treatment strategies for bipolar disorder include pharmacological management with antidepressants, antipsychotics, and mood stabilizers, along with psychotherapy. Despite the positive effects of these therapies, bipolar disorder is characterized by high rates of relapse and functional impairment, particularly during times of stress. Fortunately, mechanistic knowledge of the disorder suggests that aerobic exercise can improve mood states and brain health in affected individuals.

Emerging evidence suggests that exercise may exert effects on mental and physical health in bipolar disorder. Ng, Dodd, and Berk (2007) studied the effects of an adjunctive walking group for inpatients with bipolar disorder and reported reduced stress, depression, and anxiety symptoms among participants. Hays and colleagues (2008) conducted an interventional study wherein persons diagnosed with bipolar 1 or bipolar 2 disorder walked or ran on a treadmill at 70% of maximum heart rate for 20 minutes. Their findings revealed a significant increase in perceptions of well-being after exercise (Hays et al., 2008). Sylvia, Ametrano, and Nierenberg (2010) showed that a multimodal intervention consisting of nutrition, wellness, and 100 minutes of exercise improved measures of stress and depression.

TABLE 21.2. Summary of Research for the Effects of Exercise on Mental Illness

Condition	Study	Design	Clinical significance	*N*	Level of evidence
Depression	Mead et al. (2009); Schuch et al. (2016); Kvam, Kleppe, Nordhus, & Hovland (2016); Wegner et al. (2014); Lawlor & Hopker (2001); Morres et al. (2019); Bailey, Hetrick, Rosenbaum, Purcell, & Parker (2018); Carter, Morres, Meade, & Callaghan (2016)	Systematic review and meta-analytic reviews on the effects of aerobic exercise and depression	Moderate to large, meaningful improvements in exercise group compared to controls in inpatient and outpatient groups; similar effects for exercise or cognitive-behavioral therapy in some studies; generally larger effects found for moderate intensity, aerobic exercise programs. and interventions supervised by exercise professionals.	53,213 children, adolescents, young adults, and older adults	Efficacious and specific
Anxiety	Wipfli et al. (2008); Ensari, Greenlee, Motl, & Petruzzello (2015); Aylett, Small, & Bower (2018); Stubbs et al. (2017); Stonerock, Hoffman, Smith, & Blumenthal (2015)	Meta-analysis and systematic reviews to determine effects of exercise on anxiety	Exercise exerted small to large reductions in anxiety in comparison to no-treatment controls; showed similar to greater reductions in anxiety compared to other treatments and placebo in some trials; high-intensity exercise programs showed greater effects than low-intensity programs.	4,830 adults	Efficacious
Schizophrenia	Dauwan, Begemann, Heringa, & Sommer (2016); Firth, Cotter, Elliott, French, & Yung (2015); Firth et al. (2017); Vancampfort, Rosenbaum, Ward, & Stubbs (2015); Vancampfort et al. (2017)	Systematic review and meta-analytic reviews investigating the effect of any type of exercise interventions in schizophrenia	Exercise exerted a robust treatment effect as an add-on, particularly for improving clinical symptoms such as cognitive and depressive symptoms, quality of life, global functioning, cognitive function, and cardiorespiratory fitness. Specifically, supervised exercise increased physical fitness and reduced both positive and negative symptoms in persons participating in ≥ 90 minutes of moderate intensity programs.	2,770 adults	Efficacious and specific as an add-on treatment for positive, negative, cognitive, and cardiorespiratory fitness impairments
ADHD	Vysniauske Verburgh, Oosterlaan, & Molendijk (2016); Cerrillo-Urbina et al. (2015); Neudecker	Systematic reviews and meta-analysis examined analyze	Aerobic exercise had a moderate to large effect on core symptoms such as attention, hyperactivity, and impulsivity and related	≥ 3,224 children & adolescents	Probably efficacious

	et al. (2019); Ng, Ho, Chan, Yong, & Yeo (2017); Suarez-Manzano, Ruiz-Ariza, De La Torre-Cruz, & Martinez-Lopez (2018); Christiansen et al. (2019)	the efficacy of exercise interventions in ADHD	symptoms such as anxiety, executive function, and social disorders, with mixed interventions and longer exercise interventions and durations consistently associated with larger effect sizes.		
Bipolar depression	Melo, Daher Ede, Albuquerque, & de Bruin (2016); Hays et al. (2008); Kycyi, Alsuwaidan, Liauw, & McIntyre (2010); Wright, Everson-Hock, & Taylor (2009); Ng et al. (2007); Sylvia, Nierenberg, Stange, Peckham, & Deckersbach (2011)	Systematic reviews and experimental designs that analyzed the efficacy of exercise interventions in bipolar disorder	Exercise was associated with improved health measures, including depressive symptoms, neurocognitive functioning, quality of life, well-being, and weight management, but future randomized controlled trials are needed to phase-specific guidelines.	15,801 adults	Possibly efficacious
Eating disorders	Ng, Ng, & Wong (2013); Hausenblas, Cook, & Chittester (2008); Cook et al. (2016); Weiss (2018); Moola, Gairdner, & Amara (2013); Blanchet et al. (2018); Vancampfort et al. (2014)	Systematic and meta-analytic reviews that analyzed efficacy of exercise interventions in eating disorders (e.g., anorexia nervosa, bulimia, and binge eating)	Supervised exercise may increase treatment compliance and improve a range of outcomes (e.g., depressive symptoms, quality of life, strength, laxative use, vomiting, binge eating, body mass index, comorbidities, and obligatory exercise) in medically approved patients. Strength and cardiovascular fitness were also shown to improve. Supervised exercise may increase treatment compliance and is positively related with weight restoration.	≥ 1,876 adolescents and adults with a mean age of 14 to 29	Probably efficacious as an add-on as long as nutritional needs are met and clients are medically cleared for activity
Substance use disorders	Hallgren, Vancampfort, Giesen, Lundin, & Stubbs (2017); Ussher, Taylor, & Faulkner (2008); Wang, Wang, Wang, Li, & Zhou (2014)	Systematic and meta-analytic reviews analyzing the effects of exercise for people with substance use disorders (e.g., alcohol, tobacco, & smoking)	Exercise did not appear to reduce alcohol consumption but had significant effects on depression and physical fitness measures. Exercise appears to reduce tobacco withdrawal and cravings. Some evidence suggests that moderate- to high-intensity aerobic exercise is needed to increase the abstinence rates, ease withdrawal symptoms, and reduce depressive symptoms.	8,517 adults	Possibly efficacious as an add-on

Another study reported that nearly 50% of surveyed patients with bipolar disorder used exercise to regulate symptoms, with the rhythmic nature of the activity providing a calming effect (Wright, Armstrong, Taylor, & Dean, 2012), possibly by decreasing allostatic load, although these effects may be state-dependent. A systematic review by Wright and colleagues (2012) underscored the fact that state-dependent knowledge of exercise effects on mania, hypomania, and mixed episodes awaits investigation. Taken together, this work suggests that exercise is possibly efficacious as an adjunct treatment to improve function in persons with bipolar disorder. On the one hand, the judicious use of aerobic exercise can reduce correlates of stress that impinge upon kynurenine cascades and, thereby, prevent excitotoxity of neurons in the brain. Also, regular aerobic exercise optimizes function in key regions important for sleep (e.g., suprachiasmatic nucleus), further reducing systemic stress. On the other hand, inappropriate timing of aerobic exercise or excess use can contribute to a person's overall stress profile, worsen symptoms, and destabilize moods, establishing a warrant for close monitoring. Moreover, aerobic exercise will not alleviate ongoing psychological stress, underscoring the need for skilled application.

Anxiety

Anxiety disorders make up the largest group of mental problems in Western society and are characterized by excessive and enduring fear and stress, avoidance of perceived threats, anxiety-related cognitions, and ANS activation. Although anxiety can be adaptive in the context of an objective threat, it becomes maladaptive when severe and pervasive. Current options for treatment of anxiety include psychotherapy and pharmacology (i.e., anxiolytics). Notwithstanding, many patients refuse pharmacotherapy due to side effects (e.g., nausea, hyperglycemia, weight gain, hyperlipidemia, high blood pressure, and sexual dysfunction) in lieu of lifestyle modification.

A large body of studies show that anxiety symptoms may be reduced by one-quarter to one-half standard deviation in response to acute exercise (Morgan, 1979; Petruzzello, Landers, Hatfield, Kubitz, & Salazar, 1991), with the greatest effects being seen in those experiencing high levels of anxiety. Clinical and epidemiological studies show that regular aerobic exercise conveys a transient reduction in anxiety symptoms on the order of 30–55% (Goodwin, 2003; Strohle et al., 2007). To date, four meta-analytic and one systematic review provide additional evidence for the anxiolytic effects of exercise (Aylett, Small, & Bower, 2018; Ensari, Greenlee, Motl, & Petruzzello, 2015; Firth et al., 2017; Stonerock, Hoffman, Smith, & Blumenthal, 2015; Wipfli, Rethorst, & Landers, 2008). Wipli and colleagues (2008) evaluated 49 randomized controlled trials and showed large anxiolytic effects for adults over 31 years of age, with the effect size increasing as exercise approached 150% or 12.5 kcal·kg–1·week–1 of the recommended public health dosage (Wipfli et al., 2008). Another, more recent meta-analytic review of 36 randomized controlled trials showed a small but statistically significant improvement following a single episode of acute exercise (Ensari et al., 2015).

Nevertheless, some evidence suggests the effects of adjunctive aerobic exercise on various anxiety disorders may vary (e.g., specific phobia, social anxiety disorder, generalized anxiety disorder, panic disorder, obsessive–compulsive disorder, and posttraumatic stress disorder), particularly when used as an adjuvant to psychotherapy. As such, logic would bode for walk–talk therapy as we describe it herein.

Merom and colleagues (2008) showed that combinations of cognitive-behavior therapy and exercise were significant for those with social phobia but less effective for panic disorder or generalized anxiety (Merom et al., 2008). Other work suggests that the physiological effects of exercise can induce anxious feelings in some individuals, but that exposure to this sensation may increase tolerance to anxiety sensitivity overall (Meyer, Broocks, Bandelow, Hillmer-Vogel, & Ruther, 1998). Mental Health Foundation guidelines recommend exercise referral for patients with anxiety who report low levels of physical activity (Edmunds, Biggs, & Goldie, 2013). Altogether, the body of evidence shows that exercise is efficacious for mitigating symptoms of anxiety in clinical and nonclinical populations, particularly when used as an add-on therapy.

Substance Use Disorders

A substance use disorder, also known as a drug use disorder, is a common medical condition wherein the abuse of one or more substances leads to clinically significant functional mental, emotional, and physical impairments. Many who develop substance abuse issues have comorbid mental health issues. A stressful and chaotic lifestyle contributes to substance abuse disorders among vulnerable individuals. Common treatments for substance addiction involve psychotherapy and drug replacement therapy (i.e., buprenorphine and methadone), with the latter approach aiming to relieve craving behavior, suppress abstinence symptoms, reduce or eliminate the use of substances, and decrease substance abuse–associated infective disease transmission (Joseph, Stancliff, & Langrod, 2000; Mattick, Breen, Kimber, & Davoli, 2014). Additionally, aerobic exercise may be a potential add-on treatment, particularly in the later phases of treatment wherein reintegration into society and maintenance of positive lifestyle changes become important goals.

Exercise reduces cravings and improves physical, emotional, and quality-of-life measures in those with substance abuse disorders (Giesen, Zimmer, & Bloch, 2016; Hallgren, Vancampfort, Giesen, Lundin, & Stubbs, 2017). Some evidence suggests that physical exercise improves abstinence by reducing cravings, with programs initiated prior to cessation programs showing greater effectiveness than programs initiated after attempts to quit (Linke, Ciccolo, Ussher, & Marcus, 2013; Ussher, Taylor, & Faulkner, 2008). Wang and colleagues (2014) showed that physical exercise reduced withdrawal, anxiety, and depressive symptoms and increased abstinence rates in those who abuse nicotine, alcohol, and illicit drugs but that the reduction of depressive symptoms was limited to alcohol and illicit drug abusers (Wang, Wang, Wang, Li, & Zhou, 2014). Parallel work showed that aerobic exercise training caused a reduction in daily use and craving for cannabis (Buchowski et al., 2011) and alcohol (Brown et al., 2009, 2010) in adults. Notably, Giesen and colleagues (2016) showed that people with mild to moderate alcohol use disorders are willing to engage in exercise interventions. Although the evidence is still somewhat limited, it appears that structured exercise is probably efficacious as an add-on treatment for those with substance use disorders who need to withdraw from addictive drugs and prevent relapse by improving stress resilience.

Eating Disorders

By definition, eating disorders (e.g., anorexia nervosa, bulimia, and binge eating) are complex mental health disorders characterized by excessive concerns about body image and weight that negatively affect self-esteem. Other hallmark traits include maladaptive perfectionism, dysfunctional emotional regulation, impulsivity, hyperactivity, and social cognitive deficits. These traits lead to abnormal behaviors related to diet restriction, uncontrolled overeating with or without purging, vomiting, laxative or diuretic misuse, and excessive exercise. In regard to the latter, Beumont, Arthur, Russell, and Touyz (1994) described a group of individuals who titrated exercise energy expenditures against caloric expenditure to ensure adequate energy "debts" to achieve weight loss as exertional bulimics. Notably, this group experienced withdrawal symptoms (e.g., depression, irritability, guilt, and anxiety) when their ability to exercise was thwarted, a trend that occurs in approximately 39% of affected individuals (Shroff et al., 2006). Knowledge of this exertional bulimia phenomenon tended to obscure the potential for the positive effects of exercise in this population until recently.

Nevertheless, contemporary guidelines now acknowledge the value of exercise as an add-on for the treatment of eating disorders. Aggregating evidence shows that exercise decreases the compulsive drive for activity and tendency for body dissatisfaction while simultaneously increasing strength, quality of life, psychological well-being, patient satisfaction, and autonomy (Bratland-Sanda et al., 2009; Cook et al., 2016; Hausenblas, Cook, & Chittester, 2008). Parallel evidence shows that supervised exercise interventions do not adversely affect weight gain for patients with anorexia nervosa (Ng, Ng, & Wong, 2013). In adjunctive treatment for binge eating, it has been shown that 6 months of walking at moderate intensity reduced weight and depression scores (Levine, Marcus, & Moulton, 1996). Another study showed sustained reductions in binge-eating episodes, body mass index, and depressive scores when exercise was paired with psychotherapy (Pendleton, Goodrick, Poston, Reeves, & Foreyt, 2002), which is another reason to advocate walk–talk therapy.

Thus experts now recommend exploration of a patient's attitudes, thoughts, and motives regarding exercise instead of avoidance (Danielsen, Ro, & Bjornelv, 2018). The hope is to help patients understand their symptoms, resist compulsive tendencies, and integrate healthy lifestyle attitudes (e.g., regular and adequate meals, rest, sleep, and exercise) into their repertoire while simultaneously addressing physical complications and targeting underlying psychological issues (e.g., body image and self-esteem; Beumont et al., 1994; Danielsen et al., 2018). Thus exercise may reduce bodily tensions and negative mood while building resilience to everyday stress to reduce the urge to binge and purge. Altogether, this evidence suggests that supervised exercise (such as walk–talk therapy) is probably efficacious as an add-on treatment for eating disorders as long as nutritional needs are met and the program is medically approved (Cook et al., 2016).

Schizophrenia

Schizophrenia is a chronic illness characterized by positive symptoms (experiences that add to one's usual experiences, such as hallucinations and delusions), negative symptoms (experiences that detract from one's usual experiences, such as social withdrawal, reduced ability to feel pleasure, affective flattening, poverty of speech, and decreased motivation for purposeful activities), and cognitive deficits that present during adolescence or early adulthood. Also, persons with schizophrenia demonstrate an impaired response to physical and psychosocial stressors (Jansen et al., 1998; Kudoh, Ishihara, & Matsuki, 1999). Current first-line treatments include psychotherapy (Galletly et al., 2016) and antipsychotic pharmacotherapies that target positive symptoms but that have minimal to no effects on negative or cognitive symptoms (Gold, 2004).

Problematically, atypical antipsychotics and lifestyle behaviors appear to promote weight gain and metabolic impairments, leading to reduced cognitive and physical capacity in the short term (Vancampfort et al., 2019; Friedman et al., 2010) and a diminished life expectancy in the long term (Walker, McGee, & Druss, 2015). Accordingly, there has been significant interest in deploying physical activity to positively alter the cardiometabolic profile and functional outcomes in this population, particularly given that only 56% of patients with schizophrenia achieve the recommended 150 minutes of moderate-intensity exercise per week (Stubbs et al., 2016).

Fortunately, an increasing number of studies show that exercise improves physical and psychological outcomes for persons with schizophrenia. Systematic and meta-analytic reviews showed that exercise improved physical fitness and reduced both positive and negative symptoms of schizophrenia (Dauwan, Begemann, Heringa, & Sommer, 2016; Firth, Cotter, Elliott, French, & Yung, 2015). Bolstering the latter findings, another study showed that aerobic exercise improved negative symptoms and general psychopathology during intervention and at 3-month follow-up in patients receiving antipsychotic treatment and that those who experienced the most severe symptoms experienced the greatest benefit (Wang et al., 2018). Other work showed that exercise improved cognitive function in a dose-dependent manner (Firth et al., 2018) and positively affected quality of life, global functioning, and depressive symptoms in persons with schizophrenia (Dauwan et al., 2016). Together, this evidence suggests that, if you can get these patients to participate, supervised exercise is efficacious as an add-on treatment to usual care in persons with schizophrenia.

Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is characterized by impairments in attention, impulsivity, self-monitoring, and cognitive flexibility. Hallmark symptoms often contribute to impairments in social, academic, and occupational functioning. As a result, affected individuals are at higher risk for developing anxiety, depression, and learning disorders. Cornerstone treatments include psychotherapy and stimulants, but 30% of people do not respond favorably to medication (e.g., side effects include insomnia, headaches, and appetite suppression). Moreover, treatment effects dissipate once active administration of medication ceases, prompting researchers to consider alternatives that target underlying neural mechanisms.

Several lines of evidence suggest that aerobic exercise may be a promising treatment alternative or add-on given the fact that it optimizes cognitive, emotional, and physical function via modulation of dopaminergic and noradrenergic systems (Gapin, Labban, & Etnier, 2011; Wigal, Emmerson, Gehricke, & Galassetti, 2013), effects that may mitigate stress and core symptoms. Results from meta-analytic reviews show that aerobic exercise positively affects cognitive and emotional outcomes for children and adolescents with ADHD, with longer interventions yielding stronger results (Cerrillo-Urbina et al., 2015; Vysniauske, Verburgh, Oosterlaan, & Molendijk, 2016). For example, Verburgh and colleagues examined the effects of exercise on 994 healthy individuals ranging in age from 6–35 years and found that acute exercise improved inhibition and executive function (Verburgh, Konigs, Scherder, & Oosterlaan, 2014). Another systematic review by Neudecker and colleagues showed a moderate to large effect on executive function after 20- to 30-minute aerobic exercise sessions and small to large effects on proxy measures for dopaminergic activity (i.e., spontaneous eye blinks and acoustic startle eye blinks; Neudecker, Mewes, Reimers, & Woll, 2019). Together, this evidence suggests that exercise is probably efficacious as an add-on or alternative, specifically because the overactive brain state that generates ADHD symptoms is functionally quieted, so to speak, after exercise such that a relaxed, attentive state of mind and behavior is facilitated, although there is a paucity of studies probing different exercise parameters across the lifespan.

CLINICAL APPLICATION OF EXERCISE THERAPY FOR MENTAL HEALTH

Although an extensive literature base supports the use of exercise as a first-line treatment for mild to moderate depression, evidence is more limited for other mental health disorders. Generally speaking, current evidence provides a clinical rationale for recommending exercise as an effective add-on treatment for the management of anxiety, schizophrenia, eating disorders, ADHD, and substance use disorders. Also, emerging evidence suggests that exercise may be a useful adjunct to the treatment of bipolar disorder, although knowledge on phase-specific effects remains lacking. Regardless of specific diagnosis, it appears that social support is a crucial element for exercise adherence for those with mental health issues (Moore, Moore, & Murphy, 2011), but cost-efficacy estimates for supervised exercise remain outstanding. Mechanistically, it seems plausible that the effects of indoor versus outdoor exercise may provide differential effects on mood state. Because those with mental health issues display a high comorbidity of respiratory, inflammatory, metabolic, cardiovascular, and neurological issues, patients should receive medical clearance from their physicians prior to the initiation of an exercise program.

Dose–Response Effectiveness of Exercise Therapy

How much exercise is needed to be effective in those with mental health disorders who receive medical clearance? A review of the literature suggests that compliance with public health dosage recommendations for intensity and duration tends to be more likely to yield clinically significant outcomes than would noncompliance with the recommendations. Thus it seems that a reasonable starting point for exercise prescription in those who are medically cleared is to attempt to adhere to the guidelines of the American College of Sports Medicine (ACSM) (American College of Sports Medicine, 2020), which recommend either (1) moderate-intensity aerobic exercise (50–85% maximum heart rate) for ≥ 20–30 minutes/day for ≥ 5 days/week, for a total of ≥ 150 minutes/week; or (2) vigorous activity ≥ 20 minutes/day on ≥ 3 days/week. Also, the ACSM standard acknowledges a commensurate need for (1) major muscle group strength exercise training 2 days/week at a 60–85% maximum, with 8–12 repetitions per set and (2) flexibility exercise of 15–30 seconds' duration following every exercise session. Remember to use caution and seek assistance if necessary to establish reasonable goals and appropriate adjustments to allostatic load (duration, intensity, and frequency), remaining mindful of the fact that exercise prescriptions are based on the client's age, fitness level, and medical condition. Consultation with a physical therapist may prove helpful in establishing standards for prescription in those with more complex medical presentations. Also, remain mindful that exercise may need to be integrated alongside standard psychotherapeutic practices and that supervision is paramount (as demonstrated in walk–talk therapy), particularly during the initial phases of treatment in those with complex comorbidities. As the therapeutic process progresses, Internet and telecommunication support may provide a helpful means to transition exercise habits from the direct treatment context to maintenance therapy.

Research on How Much Exercise Is Necessary to Be Effective

To determine optimal thresholds for exercise, one 12-week randomized controlled study examined 80 sedentary adults ages 20–45 who were diagnosed with depression (Dunn, Trivedi, Kampert, Clark, & Chambliss, 2005). Participants were entered into one of four different aerobic exercise conditions that varied total energy expenditure: low-energy expenditure (7.0 kcal/kg/week) training 3 days/week; high-energy expenditure training (17.5 kcal/kg/week) 3 days/week; low-energy expenditure training (7.0 kcal/kg/week) 5 days/week; and high-energy expenditure training 5 days/week. Specifically, total energy expenditures were divided into frequencies of 3 or 5 days/week. The results for the training participants were compared with a stretching and flexibility control group with activity of 15–20 minutes' duration. Results showed that exercise conducted at the high-energy expenditure dose (consistent with public health recommendations) was effective in reducing depressive symptoms 47% from baseline over the 12-week treatment period, whereas the low-energy expenditure group only showed a 30% decline, which was not statistically different from the control condition that showed a 29% decline. Regarding frequency of training, no significant difference in treatment response was found between those participants who exercised 3 days/week relative to those who exercised 5 days/week. The authors concluded that higher energy expenditure was critical to remission of major depressive disorder.

In another large-scale epidemiological study of the dose–response relationship between exercise and depression (Kim et al., 2018), 99,846 middle-aged adults, 4802 of which were depressed and had no medical contraindications to activity, were surveyed with a physical activity questionnaire, and the responses were then converted into metabolic equivalents (METs). Analysis showed that those exercising 1-15 times the minimum exercise recommendation, or 600-9000 METs-minutes/week, exhibited a significantly lower risk of depression compared with the sedentary group (0-600 METs-minutes/week). Moreover, middle-aged men who trained in the 6,000-9,000 METs-minutes/week range derived maximal benefit, whereas middle-aged women who trained in the 1,200-1,800 METs-minutes/ week derived maximal benefit. The upper limit of exercise-related antidepressant effects was much higher in men (37.5 hours) than in women (12.5 hours), suggesting that men have to participate in exercise longer and harder to get the same antidepressant effects.

Amid questions about appropriate dosage, we point out the fact that a clinician who treats one person with mental illness does simply that: He or she treats one unique person! Because of this, it behooves the evidence-based clinician to remain cognizant of the fact that every individual presents with a constellation of genetic, biological, environmental, and social background characteristics and to personalize the treatment prescription accordingly. Just recommending exercise to the client is not nearly as safe and effective as embarking on a walk–talk program with the client to gradually ease him or her into the habit of exercising for enjoyment and healthy outcomes.

Limitations and Contraindications of Exercise

Exercise, like medication, is a method of getting immediate (though temporary) stress reduction and symptom relief. As such, medication and exercise are similar in that they allow the person to get through a particularly difficult period of pain or discomfort (either physical or emotional), after which insight-oriented counseling may be appropriate for the prevention of escalating problems. Notably, some evidence suggests that the combination of exercise and talk therapy can be more effective than an equivalent experience of cognitive therapy alone (Kazemi et al., 2018; McNeil, LeBlanc, & Joyner, 1991; Sime, 1987).

Even though it appears that more potential benefits than risks are associated with exercise, it seems unlikely that benefits from exercise are universal. Some estimate that approximately 2% of the Western population experiences exercise-induced anaphylaxis (Barg, Medrala, & Wolanczyk-Medrala, 2011), a condition that can present alongside food-dependent anaphylaxis (Jiang, Wen, Li, & Yin, 2018), with common triggers involving wheat, shellfish, peanuts, corn, and tomatoes. As such, the presence of comorbid issues that result in hives and allergic reactions to exercise of sufficient intensity (Larson & Uwagbai, 2016) may preclude some from fully experiencing the full gamut of exercise routines. Yet with medical clearance, guidance, and monitoring, it may be possible for those with exercise allergies to benefit with specific cautions in place that may involve (1) avoiding precipitating factors (e.g., extreme temperatures, alcohol, nonsteroidal anti-inflammatory drugs, food consumption 6–8 hours prior to activity, or wearing a mask to avoid air allergens), (2) pursuing alternate activities such as swimming, and/or (3) prophylactic use of antihistamines or an EpiPen (an emergency injectable of adrenaline to stop an adverse reaction) during times of acute flare-up.

Undoubtedly, exercising near air pollution from traffic fumes cancels the beneficial effects of exercise for those both with allergies and without, and so the selection of green spaces is generally advised. Others may struggle with different types of physical issues. Some experience ongoing pain in muscles or joints with weight bearing as a result of old injuries or arthritis. For those with injuries or arthritis, exercise has been shown to increase muscle mass and improve strength and physical functioning without exacerbation of disease activity or joint damage (Cooney et al., 2011). Still others may struggle with balance or sensory issues, a segment of the population for whom the fear of falling or being struck by an automobile in the course of walking or jogging in a public area may initially outweigh any potential psychological benefits. For them, a combination of therapeutic rehabilitation activities may precede or co-occur alongside a structured exercise program.

In contrast to the unsuccessful intenders, the inclined abstainers may avoid exercise as a result of past adverse experiences or memories of discouragement, memories that they may not even be cognizant of but that can serve as a source of stress. For this hard-to-motivate group, consideration of self-efficacy may prove beneficial. Still others may be suffering from life-threatening suicidal ideation, a state that may preclude the immediate application of exercise interventions and negate the physiological effects of fitness (Gronningsaeter, Hyten, Skauli, & Christensen, 1992; Paffenbarger, Lee, & Leung, 1994). We have never addressed a suicidal person with the prospects of a walk–talk session, but we believe there is merit in doing so.[1]

Other diagnosis-specific issues also warrant mention. For instance, those with depressive symptoms may find it difficult to engage in unsupervised exercise routines initially, given deficits in motivation and energy. In contrast, those with bipolar disorder may use exercise to offset excess energy and normalize routines but progress to using exercise as a goal-striving activity with addiction-like qualities during a manic period (Wright et al., 2012). Such trends may be countered by adherence to consistent levels of daily exercise. Persons with bipolar disorder often demonstrate reduced exercise tolerance (Shah et al., 2007). Therefore, clinicians must be alert to the possibility of increased risk of exhaustion, dehydration, and injury during such times, particularly for those taking lithium and exercising in hot environments.

Specific precautions also exist for those with eating disorders. Loss of bone density, osteoporosis, stress fractures, damage to joints, and neuropathy may limit the ability of those with eating disorders to engage in activity; yet once medical clearance is given, weight-bearing exercises delivered in the context of adequate nutrition may be able to reverse deficits in bone deposition (Mehler, Cleary, & Gaudiani, 2011; Mehler & MacKenzie, 2009; Solmi et al., 2016). Parallel work shows that cardiovascular complications of anorexia and bulimia nervosa may induce mitral valve prolapse, bradycardia, hypotension, and cardiac arrhythmia (Schocken, Holloway, & Powers, 1989), changes likely related to electrolyte disturbances in emaciated individuals who vomit or purge (Hall & Beresford, 1989). Thus monitoring is required to ensure that perseverance of exercise in the presence of dizziness and palpitations does not occur. Moreover, it is important for those with eating disorders to consider different motives for exercise, particularly as they relate to weight, shape, and appearance (Dalle Grave, Calugi, & Marchesini, 2008), and regulation of emotions, compulsivity, and rigidity (Bruch, 1962; Meyer & Taranis, 2011). Some patients with depression also have bulimia and may use exercise as a means of purging (Dittmer et al., 2018), thereby using exercise to excess as a risky substitute for dealing with deeper psychological issues (Sime, 1987).

Individuals with a history of substance abuse may experience peripheral nerve damage, or neuropathy, which produces pain, numbness, and muscle extremity weakness. Although it is rare, some heavy drinkers exhibit reduced numbers of red blood cells, a state that triggers symptoms of fatigue, shortness of breath, and lightheadedness. People experiencing more severe withdrawal symptoms may require a period of specialized treatment prior to the initiation of a supervised exercise program.

MANUAL FOR TRAINING

Today, walk–talk therapy is acknowledged as a relatively convenient and safe method of conducting exercise during traditional psychotherapy (Sime, 2002; Sime & Hellweg, 2001). Therapeutically, the act of walking to the side of the client during brisk walking or running eliminates barriers and subtly helps clients to engage in difficult topics by eliminating the pressure of continuous eye contact (Sime, 2002). The informal atmosphere often proves especially helpful for clients who have difficulty formulating responses in that it provides an opportunity to focus on the scenery or the pace or rhythm of walking to mitigate stress. Moreover, the approach appears to be highly cathartic when the issues are complicated or entangled.

Therapists working with clients in the process of walk–talk therapy should follow general guidelines. Initially, when taking a client's history, it is necessary to establish his or her baseline activity patterns for both work and leisure physical activity. This task can be accomplished by tracking activity with a calendar, an online tracking system, or personal technology gadgets such as smartphones or a Fitbit, Garmin, or Google smartwatch. Other standardized measures that enable assessment include the Global Physical Activity Questionnaire, Pfaffenberg Physical Activity Questionnaire, and Yale Physical Activity Survey. Determining the client's typical work and leisure patterns offers an opportunity to scaffold upon existing activity to increase fitness over time. Also, document the effects of sleep and mood that result from increased levels of physical activity. As the process of walk–talk therapy progresses, encourage the client to record his or her daily exercise type, duration, and mood estimate using a scale of 1–10, with 10 indicating the worst mood and 1 the best mood.

It may also be advantageous to help the client to become aware of the sensory and aesthetic pleasures associated with exercise during the process. For example, the part of the brain that is responsible for food-pleasure cravings may be more active after exercise, and so clients may note that healthy foods such as fruits and vegetables taste much better following a vigorous bout of exercise. Similarly, drinking cold water during and after exercise provides a refreshing sensation while enabling rehydration. After sweating abates during the recovery period (in about 20–30 minutes), a cool shower provides a soothing reward. Moreover, many people notice more rapid sleep onset and restful sleep patterns with continuous sleep maintenance after participating in a pattern of regular aerobic exercise. These sensory and aesthetic rewards of exercise can be very powerful, immediate reinforcers. Take note of these as you revise the exercise program based on client feedback at 2- to 4-week intervals.

Because physical activity is not without risk, clients should receive medical clearance from their physicians prior to the initiation of an exercise program. For those without formal training in exercise, it is advisable to seek professional assistance from a physical therapist or exercise physiologist to offer assistance with the ongoing exercise prescription process. Also remain mindful of the fact that just as too little exercise is disadvantageous for mental and physical health, so too is extreme exercise.

Presentation to the Client

Given that movement is inherently pleasurable, most clients can recall having had a sense of satisfaction in completing a difficult physical task or the enjoyment of having accomplished a significant achievement in sport or recreational activity. Regardless of the specifics, a recall of the enjoyment of exercise is important, and so it becomes necessary to help the client identify a motivating event. When it feels timely to explore the walk–talk therapy option, it is helpful to facilitate a warm recall of exercise, perhaps with questions such as those given in Table 21.3. Then introduce the concept by asking the client, "Do you feel that you might enjoy walking a little while we talk in our next session?" If the client appears receptive to engaging in walk–talk therapy, do give the client some advance warning of appropriate preparations, such as wearing comfortable shoes, a warm coat, and gloves. Also let the client know that you intend to pick a route after considering both environmental circumstances (e.g., a route with minimal traffic or access to aesthetically appealing environments such as a park or garden) and privacy.

TABLE 21.3. Interview Questions for Walk–Talk Therapy Candidate

1. "How much physical exercise do you get right now, either at work or in recreational activities?" Options: none; yard work only; walk to work 1–2 miles daily; tennis once a week; etc.
2. "Do you recall a time in your life when you might have been active in sports or work such that you were physically fit?" Options: sports in school; biking; working construction; etc.
3. "Do you recall feeling a sense of relief on getting into a soothing and invigorating shower (or bath) immediately after a workout or exercise?"
4. "Do you recall enjoying the good taste of a cold drink and a healthy meal sometime after sport or physical work?"
5. "Do you recall a time in your life when you may have slept better and felt better in association with having been involved in a regular program of vigorous activity?"
6. "Do you recall a time in your life when you felt exceptionally happy, stress-free, or tolerant of stressful circumstances? If so, were you more or less active physically at that time in your life?"
7. "Is there a particular sport or recreational activity (hiking, gardening, etc.) that you enjoy a great deal but have not been engaged in during the past few years? If so, would you consider starting up once again? What obstacles stand in your way?"
8. "Do you have the opportunity to get more activity, either by commuting to work (walking or bicycling) or at work by taking on more effort (e.g., using stairs instead of elevator)?"
9. "Do you have a friend or relative (spouse) with whom you would enjoy walking or engaging in other forms of exercise? If so, please explore that option."
10. "Do you have any injuries or physical limitations in which exercise might cause aggravating symptoms?" Options: ankle, foot, knee, hip or back pain, or arthritis.

Session 1

Prior to the session, suggest that the client find a secure place for personal belongings (purse or briefcase locked in a secure place) so that he or she does not have to carry it or worry about having something stolen. Logistics aside, then encourage the client to help set the pace of walking from the beginning of the session, emphasizing the fact that there is no need to hurry. If the activity encourages client interaction, then continue within tolerance. Remain mindful that safety and relative comfort are paramount so as to avoid health risks such as straining muscles, falling, or causing chest pain due to cardiac ischemia (angina). It may be prudent to limit the duration of the initial walking session to no more than 10–20 minutes and to stay close to the vicinity of office resources and ancillary help in case of any difficulties for older clients, those with comorbid conditions, and those with marginal disabilities. Then the goal is to increase the pace and challenge of walking gradually. A reasonable target is to establish a pace that can be maintained comfortably while talking for 20–40 minutes. However, if the walk appears intimidating or becomes a strain, do return to the counseling office and process the effects of exercise and content of the session.

Session 2

At the outset of the second session, inform the client that activity options exist depending on how he or she responded to the first exercise session. Consider varying the session in some way by changing location or extending the duration of the walking component by 5–10 minutes if the process facilitated more verbal interactions. While doing so, monitor the client's response to exercise and remain mindful that the task of talking while walking is more physically demanding than the singular task of walking.

During the session, explore with the client whether the walk–talk experience is pleasant and invigorating (or aversive) and whether she or he discovers some degree of refreshing aftereffects during recovery. Share with the client several explanations for the potential benefits of exercise, including those related to aesthetic and kinesthetic aspects. Point out that the choice to actively take measures that restore and protect one's health promotes a sense of control over personal circumstances, as well as feelings of success after walking a considerable distance. Encourage the client to recapitulate these positive feelings by repeating the exercise session at home daily or every other day. Do point out that the antidepressant and anti-anxiolytic effects of exercise are temporary, further bolstering the rationale for daily exercise. Educate the client to the fact that the activity should become less effortful and fatiguing over a span of days, particularly as her or his fitness levels increase. The client should know by observation that the therapist him- or herself uses exercise for preventative and rehabilitative purposes.

Session 3

During the third session, the goal is to have the client engage in cognitive catharsis while walking. The goal is to use the activity to help clear out rumination, worry, and anxiety, as well as to free-associate and embrace creative thinking. In this way, the session can constitute an opportunity for introspection and the development of more lucid thinking. Acknowledge the fact that both the client and therapist may come up with spontaneous questions and comments in the midst of engaging in walk–talk conversation. For some clients, the side-by-side conversation (in contrast to face-to-face therapy) elicits more candor, as though the movement, together with the informal nature of the activity, can break down cognitive barriers to exercise (Kendzierski & Johnson, 1993) and at the same time facilitate greater emotional release.

Session 4

Dealing with thoughts that underlie decisions and choice is a critical issue in all health behavior change, but especially so with exercise (Kendzierski & Johnson, 1993). Eliminating barriers and establishing exercise as a way of being in the world, not a "stairway to heaven" per se, is vitally important (Fahlberg, Fahlberg, & Gates, 1992). Thus, during the fourth session, explore the client's thoughts about the meaning of exercise, particularly how exercise might be thought of as a self-affirming way of living in the world, not necessarily a compliance behavior or a measure of striving to acquiesce to society's dictates. Consider exploring various public exercise environments—which may include the beach, hills, wooded areas, stairs, streets, or malls in cold weather—as well as the home exercise environment (e.g., walking in a scenic neighborhood)—to increase the potential aesthetic pleasure of exercise and, ultimately, to eliminate excuses for not being active. Help the client remove barriers that might impede this form of self-realization by introducing the idea that wearing more comfortable, casual clothing for leisure and work makes it more feasible to include exercise during the daily routine and can be encouraged as a reward or added incentive for accomplishment. Also, clients may benefit from personal role model anecdotes, such as, "Regular exercise was much easier to maintain when I was younger and did not have the luxury of a car (or second car for the family) because I chose to commute to work on a bicycle, thus saving substantially on bus and train fare. Do you have any similar opportunities for commuter exercise?" Other options to make exercise functional include walking to the store for milk, mowing the lawn, raking leaves, and gardening, all of which could raise a sense of satisfaction when the task is completed. Do talk about the need for safety awareness with exercise (e.g., shoveling snow or lifting bulky boxes), particularly as they relate to age, health, and physical ability—after all, the idea is to improve the client's quality of life.

Session 5

After several weeks of walk–talk therapy, evaluate progress with the client. Breathing should be monitored periodically and especially after more intense segments (e.g., climbing a hill or a flight of stairs) to observe whether the client is using more efficient breathing (diaphragmatic or abdominal), which has relaxing effects, versus less efficient breathing (chest), which can cause tension in the neck and shoulders and contribute to feelings of anxiety. Should problems be detected, then muscle relaxation training and biofeedback can likely prove helpful (see Gevirtz, Chapter 6; McGuigan & Lehrer, Chapter 7; and Lehrer, Chapter 10, in this volume).

For some clients, walking and/or stair climbing may be uncomfortable because of joint strain associated with past injuries or simply being overweight. If so, consider alternatives such as aquatics or alternate machines (e.g., such as stationary bicycle, rowing machine, or low-impact exercise machines). If boredom is a problem with home exercise sessions, consider suggesting the use of a radio, television, audiotapes, podcasts, or apps for entertainment and engagement. Some clients enjoy dancing, which provides a healthy and refreshing alternative mode of activity while gradually building up fitness and tolerance for exertion. Regardless of the activity, reinforce the idea that becoming absorbed in movement can be meditatively beneficial.

Session 6

It is always important to evaluate the influence of family and friends on the daily habits of exercise. If the client receives little or no support from significant others, or, worse yet, if those significant others show disdain for exercise, the client will have a bigger hurdle to overcome in maintaining exercise habits. Then it becomes important to facilitate activities that bring the client into positive interaction with others during exercise. Also consider encouraging the client to exercise regularly to serve as a role model for children, family, and friends. Point out that exercising with another person while engaged in pleasant conversation becomes an extension of the walk–talk therapy. Underscore the fact that the benefits of voluntary exercise independent of therapy are vitally important for maximizing the impact of walk–talk therapy in the long term.

Session 7

Help the client initiate structured behavior-modification strategies to reinforce the activity. For example, clients might make a pact whereby they will watch television only while simultaneously exercising on a bicycle ergometer, minitrampoline, stair-stepper, Cardio-Glide, elliptical machine, or treadmill. Alternatively, the client might agree not to watch more than 1 hour of TV without engaging in at least 10–20 minutes of exercise in between programs. In this way the TV becomes the reinforcement for exercise behavior, especially if the exercise can be done while watching TV. Set up a log as part of a system of accountability for the client such that he or she can review progress in accomplishments (e.g., distance walked; consecutive days; change in mood, alertness, or sleep quality; appetite regulation and reduced fatigue; or relief from nagging joint or muscle pain). A system of record keeping often promotes compliance and continuity in the training regimen, factors that are critical for effective stress management and desired psychological outcomes.

Session 8

Recognize individual differences in ability, motivation, and preferences. Some clients may be unwilling to try the approaches described here. Some are endowed with the burning desire to stay involved in exercise, and others may have lethargy built into their genetic makeup. See the citation from Kenneth Cooper—the man with heart disease who, after exercising, chose to live instead of die—discussed in endnote 1. This case story is not surprising given that very intense exercise (e.g., marathon running) is, in fact, associated with more dramatic increases in mental time travel (remembering oneself as a child, adolescent, and adult), imagination, problem solving, and positive images of the future (Masters, 1992) than are reported at lower intensities such as those found in walk–talk therapy. Nevertheless, most therapists and clients are not prepared for such vigorous activity during therapy.

Session 9

Anticipate the first bout of recidivism, as nearly every client will have occasional lapses, reverting to sedentary living due to job pressures, family emergencies, illness, injury, and so forth. Planning ahead helps to counter feelings of failure and can defuse negative emotions that result (e.g., guilt, remorse, or low self-esteem). Help your client anticipate the problem, plan for the adjustment, and welcome the return to activity following a lapse. To prevent future relapse, seek to identify the client's immediate rewards for exercise, which may include (1) a break from the hassles of the day, (2) an opportunity to enjoy the fresh air out of doors, (3) the chance to talk with you, or with a friend, about problems in life while walking, (4) the refreshing sensation of the cool air or a warm shower, depending on the existing weather conditions, or (5) the pleasant culinary satisfaction of a good meal or fluid replacement with a favorite nonalcoholic drink immediately after the exercise session.

Session 10

In the tenth session, some clients may be ready to advance to running. This obviously challenges the therapist to be fit enough to keep up. For those clients who seek to transition from walking to running, the best advice is to run as slowly as possible at first until becoming fatigued or short of breath, slow to a walk until recovered, then return to running. Running for 1–2 minutes followed by a 1-minute rest is a good ratio to target in the initial sessions. Obviously, vigorous running compromises the ability to talk somewhat, but communication is still feasible when running at a slow speed. A rule of thumb about moderate-intensity running is that it should never compromise one's ability to carry on a conversation.

Regardless of whether choosing to run or walk, take the opportunity to problem-solve how the client might overcome future obstacles that get in the way of exercise, particularly those related to inclement weather. There will always be times when cold and wind make it difficult to enjoy being outdoors in northern regions. Under these conditions, it is important to dress warmly and to stay relatively close to indoor access to warm up periodically. In other circumstances, heat can make outdoor exercise difficult. In both circumstances, public buildings and shopping malls, if accessible, can be good locations for dealing with inclement weather.

Finally, evaluate the progression of overall physical activity levels in relation to psychological health using well-accepted standardized tests. Also evaluate sleep habits, nutritional status and intake, and any increasing gratitude for the simple pleasures in life.

Conclusion of 10-Session Walk–Talk Manual

The aspiring exercise walk–talk therapist should follow ethical practices involving responsibility to and communication with other health care professionals. It becomes important to regularly communicate with other relevant professionals about progress or concerns (e.g., the client's primary care physician, psychiatrist, physical therapist, social worker, or counselor). The walk–talk therapist should observe other ethical guidelines and medical or psychological licensing requirements. Attention should be paid to maintain clearly established boundaries, and statements about the benefits of exercise should be congruent with the evidence base (Sime, 1987). During the process, the therapist must ensure that he or she does not abdicate responsibility for therapeutic progress by letting the sessions become overly social. The therapist's responsibility is to help the client initiate a sustainable evidence-based exercise routine while also adhering to basic tenets of counseling. The therapist must remain cognizant that the program oftentimes will not supersede other lifestyle patterns. Also, care should be taken to make exercise functional while ensuring that the therapist does not feel overly responsible for the client's motivation or personal accomplishments.

The walk–talk therapist is encouraged to be bold in the use of innovative strategies but cautious in the presence of extenuating circumstances that might be risky for the client. Remain mindful that exercise independent of conjoint counseling can be an effective modality at times. Yet the judicious use of walk–talk therapy may be a particularly powerful technique when both top-down and bottom-up processes need targeting.

Admittedly, the use of walk–talk therapy is still in the pioneering stage, and a great deal of research is needed to refine parameters in many mental health conditions.

CASE EXAMPLE: LIFE HISTORY OF A DEVOTED EXERCISER

Willard grew up on a farm in the Midwest, where he was ingrained with an ethic of hard work. He had plenty of opportunity for exercise, even though it was experienced as drudgery at times. Therein, he developed a serendipitous appreciation for how sweat and work heightened the rewarding effects of food, water, and deep sleep. During his teenage years, Willard increasingly applied the work ethic he learned on the farm toward competitive sports. With time, he learned that he preferred the predictability and reward of competitive training to farm work, a tendency that followed into college and beyond into early adulthood.

When Willard took his first professional job as a research assistant in the Chicago Health Research Foundation, he collected data on fitness levels in middle-aged men. While doing so, he assessed their motivation and persistence as they attempted to train at least three times/week for 45–60 minutes at a moderately high intensity. In keeping with the standards set forth by the Research Foundation, Willard soon established that it was practically feasible to maintain intense, prolonged, and regular exercise. Translating this knowledge to his own life, he began to commute to and from work on a bicycle so that he could squeeze a good workout into his day while effectively managing his time and money, a practice that became essential once he was married and in graduate school.

After he transitioned to the professoriate, Willard prided himself at inspiring students and young professionals to remain fit and adhere to stress management practices by active modeling. Having only one family car and living 5 miles away from the university, he derived a convenient solution to deal with a daily commute to work. His neighbor left for work every morning at 7:30 and generously offered a ride. In the afternoon, the neighbor left work for home earlier than Willard, and so he opted to run the 5 miles home, which took about 40–45 minutes, the optimum recommended training duration. No extra decisions or extra motivation was required. If he wanted to partake in a nice dinner at home, Willard had to hustle to get there. This practice provided ancillary training benefits for his new recreational endeavors: racquetball and the occasional 10K run on weekends.

At about the same time period, Willard found a new friend who lived nearby. On weekends, the two of them would meet at a trail and run 5–6 miles for enjoyment and camaraderie. Willard discovered that the social enjoyment of running and talking was very satisfying and reinforcing to this continuing habit pattern of regular exercise. As such, leisure running in local competitions (10K), occasionally supplemented with longer training workouts for sport, also made the 5-mile running commute to work easier. Occasional and very rare 26.2-mile marathons punctuated that time period as well (the Pikes Peak Marathon, which he did at age 36, was one extreme example). The camaraderie associated with mixing at the starting line and suffering en masse at the end of such a race was the parallel equivalent to competing in sports from years past.

During this time period, Willard became intrigued with the concept of walk–talk therapy. He knew the personal benefits of the endorphin high immediately after a run and that the literature indicated that regular exercise had antidepressant effects. Willard had made a career change to become a health psychologist, so it was not outside his purview to begin using walk–talk therapy with his clients. He found that the cathartic effect of side-by-side walking brought forth more emotionally charged topics to explore and process. In essence, it simply brought out a flow of conversation that was also saturated with the mild antidepressant effects of brisk walking. Voilà—a breakthrough for many clients suffering from depression or a myriad of difficult life challenges presented, as attested to in this chapter.

NOTE

1. Here is an anecdote from Kenneth Cooper (Cooper, 1968). A middle-aged cardiac patient was so depressed because of his dismal prognosis for the future that he decided to end his life. In his mind, the easiest and least problematic way to do that was to go out and run until he had a heart attack. The first day, he left his front door to end his life (self-destructively) by running as far as he could, as long as he could. He ended up fully out of breath and collapsed on the ground but nowhere near his death. He tried again the next day, thinking he had to be more serious about this terminal experience. Once again he ran as hard as he could, as long as he could, only to find himself totally out of breath but not requiring a 911 call. He repeated this process every day for over a week, to no avail in mortality terms. But much to his amazement, after the 8th, 9th, and 10th days, he noticed that his depression had dissipated so much so that he no longer wanted to end his life.

REFERENCES

Abdollahpour, A., Khosravi, N., Eskandari, Z., & Haghighat, S. (2017). Effects of six months of aerobic exercise on plasma interleukin-6 and tumor necrosis factor-alpha as breast cancer risk factors in postmenopausal women: A randomized controlled trial. *Iran Red Crescent Medical Journal, 19*(1), 1–7.

Agudelo, L. Z., Femenia, T., Orhan, F., Porsmyr-Palmertz, M., Goiny, M., Martinez-Redondo, V., et al. (2014). Skeletal muscle PGC-1alpha1 modulates kynurenine metabolism and mediates resilience to stress-induced depression. *Cell, 159*(1), 33–45.

Agudelo, L. Z., Ferreira, D. M. S., Cervenka, I., Bryzgalova, G., Dadvar, S., Jannig, P. R., et al. (2018). Kynurenic acid and Gpr35 regulate adipose tissue energy homeostasis and inflammation. *Cell Metabolism, 27*(2), 378–392.

Alekseichuk, I., Turi, Z., Amador, de L., Antal, A., & Paulus, W. (2016). Spatial working memory in humans depends on theta and high gamma synchronization in the prefrontal cortex. *Current Biology, 26*(12), 1513–1521.

American College of Sports Medicine. (2020). Essential resources and downloads related to ACSM physical activity guidelines. Retrieved from *www.acsm.org/blog-detail/acsm-certified-blog/2020/05/07/acsm-guidelines-resources-and-downloads.*

Autry, A. E., & Monteggia, L. M. (2012). Brain-derived neurotrophic factor and neuropsychiatric disorders. *Pharmacological Reviews, 64*(2), 238–258.

Aylett, E., Small, N., & Bower, P. (2018). Exercise in the treatment of clinical anxiety in general practice" A systematic review and meta-analysis. *BMC Health Services Research, 18*(1), 559.

Babyak, M., Blumenthal, J. A., Herman, S., Khatri, P., Doraiswamy, M., Moore, K., et al. (2000). Exercise treatment for major depression: Maintenance of therapeutic benefit at 10 months. *Psychosomatic Medicine, 62*(5), 633–638.

Bailey, A. P., Hetrick, S. E., Rosenbaum, S., Purcell, R., & Parker, A. G. (2018). Treating depression with physical activity in adolescents and young adults: A systematic review and meta-analysis of randomised controlled trials. *Psychological Medicine, 48*(7), 1068–1083.

Barg, W., Medrala, W., & Wolanczyk-Medrala, A. (2011). Exercise-induced anaphylaxis: An update on diagnosis and treatment. *Current Allergy and Asthma Reports, 11*(1), 45–51.

Bartsch, T., Dohring, J., Rohr, A., Jansen, O., & Deuschl, G. (2011). CA1 neurons in the human hippocampus are critical for autobiographical memory, mental time travel, and autonoetic consciousness. *Proceedings of the National Academy of Sciences of the USA, 108*(42), 17562–17567.

Beumont, P. J., Arthur, B., Russell, J. D., &

Touyz, S. W. (1994). Excessive physical activity in dieting disorder patients: Proposals for a supervised exercise program. *International Journal of Eating Disorders, 15*(1), 21–36.

Blanchet, C., Mathieu, M. E., St-Laurent, A., Fecteau, S., St-Amour, N., & Drapeau, V. (2018). A systematic review of physical activity interventions in individuals with binge eating disorders. *Current Obesity Reports, 7*(1), 76–88.

Bland, B. H., & Oddie, S. D. (2001). Theta band oscillation and synchrony in the hippocampal formation and associated structures: The case for its role in sensorimotor integration. *Behavioural Brain Research, 127*(1–2), 119–136.

Bratland-Sanda, S., Rosenvinge, J. H., Vrabel, K. A., Norring, C., Sundgot-Borgen, J., Ro, O., et al. (2009). Physical activity in treatment units for eating disorders: Clinical practice and attitudes. *Eating and Weight Disorders, 14*(2–3), e106–e112.

Brown, R. A., Abrantes, A. M., Read, J. P., Marcus, B. H., Jakicic, J., Strong, D. R., et al. (2009). Aerobic exercise for alcohol recovery: Rationale, program description, and preliminary findings. *Behavior Modification, 33*(2), 220–249.

Brown, R. A., Abrantes, A. M., Read, J. P., Marcus, B. H., Jakicic, J., Strong, D. R., et al. (2010). A pilot study of aerobic exercise as an adjunctive treatment for drug dependence. *Mental Health and Physical Activity, 3*(1), 27–34.

Bruch, H. (1962). Perceptual and conceptual disturbances in anorexia nervosa. *Psychosomatic Medicine, 24,* 187–194.

Buchowski, M. S., Meade, N. N., Charboneau, E., Park, S., Dietrich, M. S., Cowan, R. L., et al. (2011). Aerobic exercise training reduces cannabis craving and use in non-treatment seeking cannabis-dependent adults. *PLOS ONE, 6*(3), e17465.

Buzsaki, G. (2002). Theta oscillations in the hippocampus. *Neuron, 33*(3), 325–340.

Carter, T., Morres, I. D., Meade, O., & Callaghan, P. (2016). The effect of exercise on depressive symptoms in adolescents: A systematic review and meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry, 55*(7), 580–590.

Cerrillo-Urbina, A. J., Garcia-Hermoso, A., Sanchez-Lopez, M., Pardo-Guijarro, M. J., Santos Gomez, J. L., et al. (2015). The effects of physical exercise in children with attention-deficit/hyperactivity disorder: A systematic review and meta-analysis of randomized control trials. *Child: Care, Health, and Development, 41*(6), 779–788.

Chatzitheodorou, D., Mavromoustakos, S., & Milioti, S. (2008). The effect of exercise on adrenocortical responsiveness of patients with chronic low back pain, controlled for psychological strain. *Clinical Rehabilitation, 22*(4), 319–328.

Chen, A. I., Nguyen, C. N., Copenhagen, D. R., Badurek, S., Minichiello, L., Ranscht, B., et al. (2011). TrkB (tropomyosin-related kinase B) controls the assembly and maintenance of GABAergic synapses in the cerebellar cortex. *Journal of Neuroscience, 31*(8), 2769–2780.

Christiansen, L., Beck, M. M., Bilenberg, N., Wienecke, J., Astrup, A., & Lundbye-Jensen, J. (2019). Effects of exercise on cognitive performance in children and adolescents with ADHD: Potential mechanisms and evidence-based recommendations. *Journal of Clinical Medicine, 8*(6). Available at *www.ncbi.nlm.nih.gov/pmc/articles/PMC6617109.*

Chwastiak, L. A., Rosenheck, R. A., & Kazis, L. E. (2011). Association of psychiatric illness and obesity, physical inactivity, and smoking among a national sample of veterans. *Psychosomatics, 52*(3), 230–236.

Claytor, R. P. (1991). Stress reactivity: Hemodynamic adjustments in trained and untrained humans. *Medicine and Science in Sports and Exercise, 23*(7), 873–881.

Cook, B. J., Wonderlich, S. A., Mitchell, J. E., Thompson, R., Sherman, R., & McCallum, K. (2016). Exercise in eating disorders treatment: Systematic review and proposal of guidelines. *Medicine and Science in Sports and Exercise, 48*(7), 1408–1414.

Cooney, J. K., Law, R. J., Matschke, V., Lemmey, A. B., Moore, J. P., Ahmad, Y., et al. (2011). Benefits of exercise in rheumatoid arthritis. *Journal of Aging Research.* Retrieved from *www.hindawi.com/journals/jar/2011/681640.*

Cooper, K. H. (1968). *Aerobics.* New York: Bantam Books.

Dalle Grave, R., Calugi, S., & Marchesini, G. (2008). Compulsive exercise to control shape or weight in eating disorders: Prevalence, associated features, and treatment outcome. *Comprehensive Psychiatry, 49*(4), 346–352.

Danielsen, M., Ro, O., & Bjornelv, S. (2018). How to integrate physical activity and exercise approaches into inpatient treatment for eating disorders: Fifteen years of clinical experience and research. *Journal of Eating Disorders, 6,* 34.

Dauwan, M., Begemann, M. J., Heringa, S. M., & Sommer, I. E. (2016). Exercise improves clinical symptoms, quality of life, global functioning, and depression in schizophrenia: A systematic review and meta-analysis. *Schizophrenia Bulletin, 42*(3), 588–599.

Deuster, P. A., Chrousos, G. P., Luger, A., DeBolt, J. E., Bernier, L. L., & Trostmann, U. H. (1989). Hormonal and metabolic responses of untrained, moderately trained, and highly trained men to three exercise intensities. *Metabolism, 38*(2), 141–148.

Dickinson, J. M., D'Lugos, A. C., Naymik, M. A., Siniard, A. L., Wolfe, A. J., & Curtis, D. R. (2018). Transcriptome response of human skeletal muscle to divergent exercise stimuli. *Journal of Applied Physiology, 124*(6), 1529–1540.

Dinoff, A., Herrmann, N., Swardfager, W., Liu, C. S., Sherman, C., & Chan, S. (2016). The effect of exercise training on resting concentrations of peripheral brain-derived neurotrophic factor (BDNF): A meta-analysis. *PLOS ONE, 11*(9), e0163037.

Dittmer, N., Voderholzer, U., von der Muhlen, M., Marwitz, M., Fumi, M., Monch, C., et al. (2018). Specialized group intervention for compulsive exercise in inpatients with eating disorders: Feasibility and preliminary outcomes. *Journal of Eating Disorders, 6,* 27.

Drago, A., Crisafulli, C., Calabro, M., & Serretti, A. (2015). Enrichment pathway analysis: The inflammatory genetic background in bipolar disorder. *Journal of Affective Disorders, 179,* 88–94.

Dunn, A. L., Trivedi, M. H., Kampert, J. B., Clark, C. G., & Chambliss, H. O. (2005). Exercise treatment for depression: Efficacy and dose response. *American Journal of Preventive Medicine, 28*(1), 1–8.

Eadie, B. D., Redila, V. A., & Christie, B. R. (2005). Voluntary exercise alters the cytoarchitecture of the adult dentate gyrus by increasing cellular proliferation, dendritic complexity, and spine density. *Journal of Comparative Neurology, 486*(1), 39–47.

Edmunds, S., Biggs, H., & Goldie, I. (2013). Let's get physical: The impact of physical activity on well-being. Retrieved from *https://cdn-flightdec.userfirst.co.nz/uploads/sites/danz/files/PDFs/2018/lets-get-physical-report.pdf.*

Ehninger, D., & Kempermann, G. (2003). Regional effects of wheel running and environmental enrichment on cell genesis and microglia proliferation in the adult murine neocortex. *Cerebral Cortex, 13*(8), 845–851.

Ensari, I., Greenlee, T. A., Motl, R. W., & Petruzzello, S. J. (2015). Meta-analysis of acute exercise effects on state anxiety: An update of randomized controlled trials over the past 25 years. *Depression and Anxiety, 32*(8), 624–634.

Erickson, K. I., Voss, M. W., Prakash, R. S., Basak, C., Szabo, A., Chaddock, L., et al. (2011). Exercise training increases size of hippocampus and improves memory. *Proceedings of the National Academy of Sciences of the USA, 108*(7), 3017–3022.

Fahlberg, L. L., Fahlberg, L. A., & Gates, W. K. (1992). Exercise and existence: Exercise behavior from an existential–phenomenological perspective. *The Sport Psychologist, 6,* 172–191.

Firth, J., Cotter, J., Elliott, R., French, P., & Yung, A. R. (2015). A systematic review and meta-analysis of exercise interventions in schizophrenia patients. *Psychological Medicine, 45*(7), 1343–1361.

Firth, J., Stubbs, B., Rosenbaum, S., Vancampfort, D., Malchow, B., Schuch, F., et al. (2017). Aerobic exercise improves cognitive functioning in people with schizophrenia: A systematic review and meta-analysis. *Schizophrenia Bulletin, 43*(3), 546–556.

Firth, J., Stubbs, B., Vancampfort, D., Schuch, F., Lagopoulos, J., Rosenbaum, S., et al. (2018). Effect of aerobic exercise on hippocampal volume in humans: A systematic review and meta-analysis. *NeuroImage, 166,* 230–238.

Friedman, J. I., Wallenstein, S., Moshier, E., Parrella, M., White, L., Bowler, S., et al. (2010). The effects of hypertension and body mass index on cognition in schizophrenia. *American Journal of Psychiatry, 167*(10), 1232–1239.

Galletly, C., Castle, D., Dark, F., Humberstone, V., Jablensky, A., Killackey, E., et al. (2016). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the management of schizophrenia and related disorders. *Australian and New Zealand Journal of Psychiatry, 50*(5), 410–472.

Gapin, J. I., Labban, J. D., & Etnier, J. L. (2011). The effects of physical activity on attention deficit hyperactivity disorder symptoms: The evidence. *Preventive Medicine, 52*(Suppl. 1), S70–S74.

Giesen, E. S., Zimmer, P., & Bloch, W. (2016). Effects of an exercise program on physical activity level and quality of life in patients with severe alcohol dependence. *Alcoholism Treatment Quarterly, 34*(1), 63–78.

Gold, J. M. (2004). Cognitive deficits as treatment targets in schizophrenia. *Schizophrenia Research, 72*(1), 21–28.

Goodwin, R. D. (2003). Association between physical activity and mental disorders among adults in the United States. *Preventive Medicine, 36*(6), 698–703.

Gronningsaeter, H., Hyten, K., Skauli, G., & Christensen, C. (1992). Improved health and coping by physical exercise or cognitive behavioral stress management training in a work environment. *Psychology and Health, 7,* 147–163.

Guan, N., Liu, H., Diao, F., Zhang, J., Zhang, M., & Wu, T. (2010). Prevalence of metabolic syndrome in bipolar patients initiating acute-phase treatment: A 6-month follow up. *Psychiatry and Clinical Neurosciences, 64*(6), 625–633.

Hall, R. C., & Beresford, T. P. (1989). Medical complications of anorexia and bulimia. *Psychiatric Medicine, 7*(4), 165–192.

Hallgren, M., Vancampfort, D., Giesen, E. S., Lundin, A., & Stubbs, B. (2017). Exercise as treatment for alcohol use disorders: Systematic review and meta-analysis. *British Journal of Sports Medicine, 51*(14), 1058–1064.

Halliwell, E. (2005). Up and running?: Exercise therapy and the treatment of mild to moderate depression in primary care. Retrieved from *www.mentalhealth.org.uk/sites/default/files/up_running_report.pdf.*

Harber, M. P., Konopka, A. R., Douglass, M. D., Minchev, K., Kaminsky, L. A., Trappe, T. A., et al. (2009). Aerobic exercise training improves whole muscle and single myofiber size and function in older women. *American Journal of Physiology Regulation, Integration and Comparative Physiology, 297*(5), R1452–R1459.

Hausenblas, H. A., Cook, B. J., & Chittester, N. I. (2008). Can exercise treat eating disorders? *Exercise and Sport Sciences Reviews, 36*(1), 43–47.

Hayashino, Y., Jackson, J. L., Hirata, T., Fukumori, N., Nakamura, F., Fukuhara, S., et al. (2014). Effects of exercise on C-reactive protein, inflammatory cytokine and adipokine in patients with type 2 diabetes: A meta-analysis of randomized controlled trials. *Metabolism, 63*(3), 431–440.

Hays, A., Goss, F. F., Aaron, D. J., Abt, K. L., Friedman, E. S., Gallagher, M., et al. (2008). Hormonal and perceptual changes in bipolar subjects after acute aerobic exercise. *Medicine and Science in Sports and Exercise, 40*(S17).

Hays, K. F. (1999). *Working it out: Using exercise in psychotherapy.* Washington, DC: American Psychological Association.

Huang, R. Q., Erlichman, J. S., & Dean, J. B. (1997). Cell–cell coupling between CO2-excited neurons in the dorsal medulla oblongata. *Neuroscience, 80*(1), 41–57.

Huang, T., Larsen, K. T., Ried-Larsen, M., Moller, N. C., & Andersen, L. B. (2014). The effects of physical activity and exercise on brain-derived neurotrophic factor in healthy humans: A review. *Scandinavian Journal of Medicine and Science in Sports, 24*(1), 1–10.

Jansen, L. M., Gispen-de Wied, C. C., Gademan, P. J., De Jonge, R. C., van der Linden, J. A., & Kahn, R. S. (1998). Blunted cortisol response to a psychosocial stressor in schizophrenia. *Schizophrenia Research, 33*(1–2), 87–94.

Jiang, N. N., Wen, L. P., Li, H., & Yin, J. (2018). A new diagnostic criteria of wheat-dependent, exercise-induced anaphylaxis in China. *Chinese Medical Journal (English), 131*(17), 2049–2054.

Joseph, H., Stancliff, S., & Langrod, J. (2000). Methadone maintenance treatment (MMT): A review of historical and clinical issues. *Mt Sinai Journal of Medicine, 67*(5–6), 347–364.

Kazemi, M., McBreairty, L. E., Chizen, D. R., Pierson, R. A., Chilibeck, P. D., & Zello, G. A. (2018). A comparison of a pulse-based diet and the therapeutic lifestyle changes diet in combination with exercise and health counselling on the cardio-metabolic risk profile in women with polycystic ovary syndrome: A randomized controlled trial. *Nutrients, 10*(10).

Kendzierski, D., & Johnson, W. (1993). Excuses, excuses, excuses: A cognitive behavioral approach to exercise implementation. *Journal of Sport and Exercise Psychology, 15*(2), 207–219.

Kim, S. Y., Jeon, S. W., Shin, D. W., Oh, K. S., Shin, Y. C., & Lim, S. W. (2018). Association between physical activity and depressive symptoms in general adult populations: An analysis of the dose–response relationship. *Psychiatry Research, 269,* 258–263.

Knaepen, K., Goekint, M., Heyman, E. M., & Meeusen, R. (2010). Neuroplasticity-exercise-induced response of peripheral brain-derived neurotrophic factor: A systematic review of experimental studies in human subjects. *Sports Medicine, 40*(9), 765–801.

Konopka, A. R., Trappe, T. A., Jemiolo, B., Trappe, S. W., & Harber, M. P. (2011). Myosin heavy chain plasticity in aging skeletal muscle with aerobic exercise training. *Journals of Gerontololgy, Series A: Biological Sciences and Medical Sciences, 66*(8), 835–841.

Krabbe, K. S., Nielsen, A. R., Krogh-Madsen, R., Plomgaard, P., Rasmussen, P., Erikstrup, C., et al. (2007). Brain-derived neurotrophic factor (BDNF) and type 2 diabetes. *Diabetologia, 50*(2), 431–438.

Kucyi, A., Alsuwaidan, M. T., Liauw, S. S., & McIntyre, R. S. (2010). Aerobic physical exercise as a possible treatment for neurocognitive dysfunction in bipolar disorder. *Postgraduate Medicine, 122*(6), 107–116.

Kudoh, A., Ishihara, H., & Matsuki, A. (1999). Pituitary-adrenal and parasympathetic function in chronic schizophrenic patients with postoperative ileus or hypotension. *Neuropsychobiology, 39*(3), 125–130.

Kvam, S., Kleppe, C. L., Nordhus, I. H., & Hovland, A. (2016). Exercise as a treatment for depression: A meta-analysis. *Journal of Affective Disorders, 202,* 67–86.

Larson, S. L., & Uwagbai, O. N. (2016). An activity-induced rash. *American Family Physician, 94*(9), 735–736.

Lawlor, D. A., & Hopker, S. W. (2001). The effectiveness of exercise as an intervention in the management of depression: Systematic review and meta-regression analysis of randomised controlled trials. *British Medical Journal, 322*(7289), 763–767.

Legakis, I. N., Mantzouridis, T., Saramantis, A., Phenekos, C., Tzioras, C., & Mountokalakis, T. (2000). Human galanin secretion is increased upon normal exercise test in middle-age individuals. *Endocrine Research, 26*(3), 357–364.

Levine, M. D., Marcus, M. D., & Moulton, P. (1996). Exercise in the treatment of binge eating disorder. *International Journal of Eating Disorders, 19*(2), 171–177.

Lewis, G. D., Farrell, L., Wood, M. J., Martinovic, M., Arany, Z., Rowe, G. C., et al. (2010). Metabolic signatures of exercise in human plasma. *Science Translational Medicine, 2*(33), 33ra37.

Li, J. Y., Kuo, T. B., Hsieh, I. T., & Yang, C. C. (2012). Changes in hippocampal theta rhythm and their correlations with speed during different phases of voluntary wheel running in rats. *Neuroscience, 213,* 54–61.

Li, M., Long, C., & Yang, L. (2015). Hippocampal–prefrontal circuit and disrupted functional connectivity in psychiatric and neurodegenerative disorders. *BioMed Research International,* 810548. Retrieved from *www.hindawi.com/journals/bmri/2015/810548*.

Linke, S. E., Ciccolo, J. T., Ussher, M., & Marcus, B. H. (2013). Exercise-based smoking cessation interventions among women. *Womens Health (London), 9*(1), 69–84.

Lloyd-Jones, D., Adams, R., Carnethon, M., De Simone, G., Ferguson, T. B., Flegal, K., et al. (2009). Heart disease and stroke statistics—2009 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 119*(3), e21–e181.

Luger, A., Deuster, P. A., Kyle, S. B., Gallucci, W. T., Montgomery, L. C., & Gold, P. W. (1987). Acute hypothalamic–pituitary–adrenal responses to the stress of treadmill exercise: Physiologic adaptations to physical training. *New England Journal of Medicine, 316*(21), 1309–1315.

Lundberg, T. R., Fernandez-Gonzalo, R., Tesch, P. A., Rullman, E., & Gustafsson, T. (2016). Aerobic exercise augments muscle transcriptome profile of resistance exercise. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology, 310*(11), R1279–R1287.

Lysenko, E. A., Popov, D. V., Vepkhvadze, T. F., Lednev, E. M., & Vinogradova, O. L. (2016). Effect of combined aerobic and strength exercise on regulation of mitochondrial biogenesis, protein synthesis and degradation in human skeletal muscle. *Human Physiology, 42,* 634–644.

Lysenko, E. A., Vepkhvadze, T. F., Lednev, E. M., Vinogradova, O. L., & Popov, D. V. (2018). Branched-chain amino acids administration suppresses endurance exercise-related activation of ubiquitin proteasome signaling in trained human skeletal muscle. *Journal of Physiological Sciences, 68*(1), 43–53.

Mahoney, D. J., & Tarnopolsky, M. A. (2005). Understanding skeletal muscle adaptation to exercise training in humans: Contributions from microarray studies. *Physical Medicine and Rehabilitation Clinics of North America, 16*(4), 859–873.

Makizako, H., Liu-Ambrose, T., Shimada, H., Doi, T., Park, H., & Tsutsumimoto, K. (2015). Moderate-intensity physical activity, hippocampal volume, and memory in older adults with mild cognitive impairment. *Journals of Gerontololgy, Series A: Biological Sciences and Medical Sciences, 70*(4), 480–486.

Masters, K. S. (1992). Hypnotic susceptibility, cognitive dissociation, and runner's high in a sample of marathon runners. *American Journal of Clinical Hypnosis, 34*(3), 193–201.

Mattick, R. P., Breen, C., Kimber, J., & Davoli, M. (2014). Buprenorphine maintenance versus placebo or methadone maintenance for opioid dependence. *Cochrane Database of Systematic Reviews, 2,* CD002207.

McNeil, J. K., LeBlanc, E. M., & Joyner, M. (1991). The effect of exercise on depressive symptoms in the moderately depressed elderly. *Psychology and Aging, 6*(3), 487–488.

Mead, G. E., Morley, W., Campbell, P., Greig, C. A., McMurdo, M., & Lawlor, D. A. (2009). Exercise for depression. *Cochrane Database of Systematic Reviews, 3,* CD004366.

Mehler, P. S., Cleary, B. S., & Gaudiani, J. L. (2011). Osteoporosis in anorexia nervosa. *Eating Disorders, 19*(2), 194–202.

Mehler, P. S., & MacKenzie, T. D. (2009). Treatment of osteopenia and osteoporosis in anorexia nervosa: A systematic review of the literature. *International Journal of Eating Disorders, 42*(3), 195–201.

Melancon, M. O., Lorrain, D., & Dionne, I. J. (2014). Changes in markers of brain serotonin activity in response to chronic exercise in senior men. *Applied Physiology, Nutrition, and Metabolism, 39*(11), 1250–1256.

Melo, M. C., Daher Ede, F., Albuquerque, S. G., & de Bruin, V. M. (2016). Exercise in bipolar patients: A systematic review. *Journal of Affective Disorders, 198,* 32–38.

Meneses-Echavez, J. F., Correa-Bautista, J. E., Gonzalez-Jimenez, E., Schmidt Rio-Valle, J., Elkins, M. R., Lobelo, F., et al. (2016). The effect of exercise training on mediators of inflammation in breast cancer survivors: A systematic review with meta-analysis. *Cancer Epidemiology, Biomarkers and Prevention, 25*(7), 1009–1017.

Merom, D., Phongsavan, P., Wagner, R., Chey, T., Marnane, C., Steel, Z., et al. (2008). Promoting walking as an adjunct intervention to group cognitive behavioral therapy for anxiety disorders: A pilot group randomized trial. *Journal of Anxiety Disorders, 22*(6), 959–968.

Meyer, C., & Taranis, L. (2011). Exercise in the eating disorders: Terms and definitions. *European Eating Disorders Review, 19*(3), 169–173.

Meyer, T., Broocks, A., Bandelow, B., Hillmer-Vogel, U., & Ruther, E. (1998). Endurance training in panic patients: Spiroergometric and clinical effects. *International Journal of Sports Medicine, 19*(7), 496–502.

Moola, F. J., Gairdner, S. E., & Amara, C. E. (2013). Exercise in the care of patients with anorexia nervosa: A systematic review of the literature. *Mental Health and Physical Activity, 6,* 59–68.

Moore, G. F., Moore, L., & Murphy, S. (2011). Facilitating adherence to physical activity: Exercise professionals' experiences of the National Exercise Referral Scheme in Wales: A qualitative study. *BMC Public Health, 11*(1), 935.

Morgan, W. P. (1979). Anxiety reduction following acute physical activity. *Psychiatric Annals, 9*(3), 36–45.

Morres, I. D., Hatzigeorgiadis, A., Stathi, A., Comoutos, N., Arpin-Cribbie, C., Krommidas, C., et al. (2019). Aerobic exercise for adult patients with major depressive disorder in mental health services: A systematic review and meta-analysis. *Depression and Anxiety, 36*(1), 39–53.

Nabkasorn, C., Miyai, N., Sootmongkol, A., Junprasert, S., Yamamoto, H., Arita, M., et al. (2006). Effects of physical exercise on depression, neuroendocrine stress hormones and physiological fitness in adolescent females with depressive symptoms. *European Journal of Public Health, 16*(2), 179–184.

National Institute for Clinical Excellence. (2004). Depression: Management of depression in primary care. Retrieved from *www.nice.org.uk/guidance/cg23*.

Neudecker, C., Mewes, N., Reimers, A. K., & Woll, A. (2019). Exercise interventions in children and adolescents with ADHD: A systematic review. *Journal of Attention Disorders, 23*(4), 307–324.

Ng, F., Dodd, S., & Berk, M. (2007). The effects of physical activity in the acute treatment of bipolar disorder: A pilot study. *Journal of Affective Disorders, 101*(1–3), 259–262.

Ng, L. W., Ng, D. P., & Wong, W. P. (2013). Is supervised exercise training safe in patients with anorexia nervosa?: A meta-analysis. *Physiotherapy, 99*(1), 1–11.

Ng, Q. X., Ho, C. Y. X., Chan, H. W., Yong, B. Z. J., & Yeo, W. S. (2017). Managing childhood and adolescent attention-deficit/hyperactivity disorder (ADHD) with exercise: A systematic review. *Complementary Therapies in Medicine, 34,* 123–128.

Noble, E. E., Mavanji, V., Little, M. R., Billington, C. J., Kotz, C. M., & Wang, C. (2014). Exercise reduces diet-induced cognitive decline and increases hippocampal brain-derived neurotrophic factor in CA3 neurons. *Neurobiology of Learning and Memory, 114,* 40–50.

Ott, C. V., Johnson, C. B., Macoveanu, J., & Miskowiak, K. (2019). Structural changes in the hippocampus as a biomarker for cognitive improvements in neuropsychiatric disorders: A systematic review. *European Neuropsychopharmacology, 29*(3), 319–329.

Paffenbarger, R., Jr., Lee, I., & Leung, R. (1994).

Physical activity and personal characteristics associated with depression and suicide in American college men. *Acta Psychiatrica Scandinavica, 89*(Suppl. 377), 16–22.

Pajonk, F. G., Wobrock, T., Gruber, O., Scherk, H., Berner, D., & Kaizl, I. (2010). Hippocampal plasticity in response to exercise in schizophrenia. *Archives of General Psychiatry, 67*(2), 133–143.

Pendleton, V. R., Goodrick, G. K., Poston, W. S., Reeves, R. S., & Foreyt, J. P. (2002). Exercise augments the effects of cognitive-behavioral therapy in the treatment of binge eating. *International Journal of Eating Disorders, 31*(2), 172–184.

Petruzzello, S. J., Landers, D. M., Hatfield, B. D., Kubitz, K. A., & Salazar, W. (1991). A meta-analysis on the anxiety-reducing effects of acute and chronic exercise: Outcomes and mechanisms. *Sports Medicine, 11*(3), 143–182.

Phillips, C., & Fahimi, A. (2018). Immune and neuroprotective effects of physical activity on the brain in depression. *Frontiers in Neuroscience, 12,* 498.

Pilegaard, H., Saltin, B., & Neufer, P. D. (2003). Exercise induces transient transcriptional activation of the PGC-1alpha gene in human skeletal muscle. *Journal of Physiology, 546*(Pt. 3), 851–858.

Radley, J. J., & Sawchenko, P. E. (2011). A common substrate for prefrontal and hippocampal inhibition of the neuroendocrine stress response. *Journal of Neuroscience, 31*(26), 9683–9695.

Rico, B., Xu, B., & Reichardt, L. F. (2002). TrkB receptor signaling is required for establishment of GABAergic synapses in the cerebellum. *Nature Neuroscience, 5*(3), 225–233.

Rimmele, U., Zellweger, B. C., Marti, B., Seiler, R., Mohiyeddini, C., Ehlert, U., et al. (2007). Trained men show lower cortisol, heart rate and psychological responses to psychosocial stress compared with untrained men. *Psychoneuroendocrinology, 32*(6), 627–635.

Ruas, J. L., White, J. P., Rao, R. R., Kleiner, S., Brannan, K. T., Harrison, B. C., et al. (2012). A PGC-1alpha isoform induced by resistance training regulates skeletal muscle hypertrophy. *Cell, 151*(6), 1319–1331.

Schlittler, M., Goiny, M., Agudelo, L. Z., Venckunas, T., Brazaitis, M., Skurvydas, A., et al. (2016). Endurance exercise increases skeletal muscle kynurenine aminotransferases and plasma kynurenic acid in humans. *American Journal of Physiology: Cell Physiology, 310*(10), C836–C840.

Schmitt, B., Fluck, M., Decombaz, J., Kreis, R., Boesch, C., Wittwer, M., et al. (2003). Transcriptional adaptations of lipid metabolism in tibialis anterior muscle of endurance-trained athletes. *Physiological Genomics, 15*(2), 148–157.

Schocken, D. D., Holloway, J. D., & Powers, P. S. (1989). Weight loss and the heart: Effects of anorexia nervosa and starvation. *Archives of Internal Medicine, 149*(4), 877–881.

Schuch, F. B., Vancampfort, D., Rosenbaum, S., Richards, J., Ward, P. B., Veronese, N., et al. (2016). Exercise for depression in older adults: A meta-analysis of randomized controlled trials adjusting for publication bias. *Brazilian Journal of Psychiatry, 38*(3), 247–254.

Shah, A., Alshaher, M., Dawn, B., Siddiqui, T., Longaker, R. A., Stoddard, M. F., et al. (2007). Exercise tolerance is reduced in bipolar illness. *Journal of Affective Disorders, 104*(1–3), 191–195.

Shroff, H., Reba, L., Thornton, L. M., Tozzi, F., Klump, K. L., Berrettini, W. H., et al. (2006). Features associated with excessive exercise in women with eating disorders. *International Journal of Eating Disorders, 39*(6), 454–461.

Siddarth, P., Rahi, B., Emerson, N. D., Burggren, A. C., Miller, K. J., Bookheimer, S., et al. (2018). Physical activity and hippocampal subregion structure in older adults with memory complaints. *Journal of Alzheimers Disease, 61*(3), 1089–1096.

Sime, W. (1981). Role of exercise and relaxation in coping with acute emotional stress. In S. Fuenning & R. Rose (Ed.), *Physical fitness and mental health: Proceedings of the first research seminar* (pp. 92–95). Lincoln: University of Nebraska.

Sime, W. (1987). Exercise in the prevention and treatment of depression. In W. P. Morgan & S. E. Goldston (Ed.), *Exercise and mental health* (pp. 145–152). Washington, DC: Hemisphere.

Sime, W. (2002). Guidelines for clinical application of exercise therapy for mental health. In J. van Roalte & B. Brewer (Ed.), *Exploring sport and exercise psychology* (pp. 159–187). Washington, DC: American Psychological Association.

Sime, W., & Hellweg, K. (2001). Stress and coping. In M. Herridge, J. Roitman, M. Kelsey, T. LaRontaine, L. Miller, M. Wegner, et al. (Ed.), *ACSM'S resource manual for guidelines for exercise testing and prescription* (pp. 541–548). Philadelphia: Lippincott Williams & Wilkins.

Sinyor, D., Schwartz, S. G., Peronnet, F., Brisson, G., & Seraganian, P. (1983). Aerobic fitness level and reactivity to psychosocial stress: Physiological, biochemical, and subjective measures. *Psychosomatic Medicine, 45*(3), 205–217.

Solmi, M., Veronese, N., Correll, C. U., Favaro, A., Santonastaso, P., Caregaro, L., et al. (2016). Bone mineral density, osteoporosis, and fractures among people with eating disorders: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica, 133*(5), 341–351.

Sothmann, M. S., Hart, B. A., & Horn, T. S. (1991). Plasma catecholamine response to acute psychological stress in humans: Relation to aerobic fitness and exercise training. *Medicine and Science in Sports and Exercise, 23*(7), 860–867.

Spalding, T. W., Lyon, L. A., Steel, D. H., & Hatfield, B. D. (2004). Aerobic exercise training and cardiovascular reactivity to psychological stress in sedentary young normotensive men and women. *Psychophysiology, 41*(4), 552–562.

Steptoe, A., Edwards, S., Moses, J., & Mathews, A. (1989). The effects of exercise training on mood and perceived coping ability in anxious adults from the general population. *Journal of Psychosomatic Research, 33*(5), 537–547.

Stonerock, G. L., Hoffman, B. M., Smith, P. J., & Blumenthal, J. A. (2015). Exercise as treatment for anxiety: Systematic review and analysis. *Annals of Behavoiral Medicine, 49*(4), 542–556.

Strasser, B., Geiger, D., Schauer, M., Gatterer, H., Burtscher, M., & Fuchs, D. (2016). Effects of exhaustive aerobic exercise on tryptophan-kynurenine metabolism in trained athletes. *PLOS ONE, 11*(4), e0153617.

Strohle, A., Hofler, M., Pfister, H., Muller, A. G., Hoyer, J., Wittchen, H. U., et al. (2007). Physical activity and prevalence and incidence of mental disorders in adolescents and young adults. *Psychological Medicine, 37*(11), 1657–1666.

Stubbs, B., Firth, J., Berry, A., Schuch, F. B., Rosenbaum, S., Gaughran, F., et al. (2016). How much physical activity do people with schizophrenia engage in?: A systematic review, comparative meta-analysis and meta-regression. *Schizophrenia Research, 176*(2–3), 431–440.

Stubbs, B., Vancampfort, D., Rosenbaum, S., Firth, J., Cosco, T., Veronese, N., et al. (2017). An examination of the anxiolytic effects of exercise for people with anxiety and stress-related disorders: A meta-analysis. *Psychiatry Research, 249,* 102–108.

Suarez-Manzano, S., Ruiz-Ariza, A., De La Torre-Cruz, M., & Martinez-Lopez, E. J. (2018). Acute and chronic effect of physical activity on cognition and behaviour in young people with ADHD: A systematic review of intervention studies. *Research in Developmental Disabilities, 77,* 12–23.

Sylvia, L. G., Ametrano, R. M., & Nierenberg, A. A. (2010). Exercise treatment for bipolar disorder: Potential mechanisms of action mediated through increased neurogenesis and decreased allostatic load. *Psychotherapy and Psychosomatics, 79*(2), 87–96.

Sylvia, L. G., Nierenberg, A. A., Stange, J. P., Peckham, A. D., & Deckersbach, T. (2011). Development of an integrated psychosocial treatment to address the medical burden associated with bipolar disorder. *Journal of Psychiatric Practice, 17*(3), 224–232.

Szuhany, K. L., Bugatti, M., & Otto, M. W. (2015). A meta-analytic review of the effects of exercise on brain-derived neurotrophic factor. *Journal of Psychiatric Research, 60,* 56–64.

Tartibian, B., FitzGerald, L. Z., Azadpour, N., & Maleki, B. H. (2015). A randomized controlled study examining the effect of exercise on inflammatory cytokine levels in post-menopausal women. *Post Reproductive Health, 21*(1), 9–15.

Tartibian, B., Hajizadeh Maleki, B., Kanaley, J., & Sadeghi, K. (2011). Long-term aerobic exercise and omega-3 supplementation modulate osteoporosis through inflammatory mechanisms in post-menopausal women: A randomized, repeated measures study. *Nutrition and Metabolism (London), 8,* 71.

Traustadottir, T., Bosch, P. R., & Matt, K. S. (2005). The HPA axis response to stress in women: Effects of aging and fitness. *Psychoneuroendocrinology, 30*(4), 392–402.

Tucker, L. A., Cole, G. E., & Friedman, G. M. (1986). Physical fitness: A buffer against stress. *Perceptual and Motor Skills, 63*(2 Pt. 2), 955–961.

Ussher, M. H., Taylor, A., & Faulkner, G. (2008). Exercise interventions for smoking cessation. *Cochrane Database of Systematic Reviews, 4,* CD002295.

van Praag, H. (2008). Neurogenesis and exercise: Past and future directions. *Neuromolecular Medicine, 10*(2), 128–140.

Vancampfort, D., Firth, J., Correll, C. U., Solmi, M., Siskind, D., De Hert, M., et al. (2019).

The impact of pharmacological and non-pharmacological interventions to improve physical health outcomes in people with schizophrenia: A meta-review of meta-analyses of randomized controlled trials. *World Psychiatry, 18*(1), 53–66.

Vancampfort, D., Rosenbaum, S., Schuch, F., Ward, P. B., Richards, J., Mugisha, J., et al. (2017). Cardiorespiratory fitness in severe mental illness: A systematic review and meta-analysis. *Sports Medicine, 47*(2), 343–352.

Vancampfort, D., Rosenbaum, S., Ward, P. B., & Stubbs, B. (2015). Exercise improves cardiorespiratory fitness in people with schizophrenia: A systematic review and meta-analysis. *Schizophrenia Research, 169*(1–3), 453–457.

Vancampfort, D., Sienaert, P., Wyckaert, S., De Hert, M., Stubbs, B., Soundy, A., et al. (2015). Health-related physical fitness in patients with bipolar disorder vs. healthy controls: An exploratory study. *Journal of Affective Disorders, 177,* 22–27.

Vancampfort, D., Vanderlinden, J., Stubbs, B., Soundy, A., Pieters, G., De Hert, M., et al. (2014). Physical activity correlates in persons with binge eating disorder: A systematic review. *European Eating Disorders Review, 22*(1), 1–8.

Vargas-Ortiz, K., Perez-Vazquez, V., Diaz-Cisneros, F. J., Figueroa, A., Jimenez-Flores, L. M., Rodriguez-DelaRosa, G., et al. (2015). Aerobic training increases expression levels of SIRT3 and PGC-1alpha in skeletal muscle of overweight adolescents without change in caloric intake. *Pediatric Exercise Science, 27*(2), 177–184.

Verburgh, L., Konigs, M., Scherder, E. J., & Oosterlaan, J. (2014). Physical exercise and executive functions in preadolescent children, adolescents and young adults: A meta-analysis. *British Journal of Sports Medicine, 48*(12), 973–979.

von Haaren, B., Ottenbacher, J., Muenz, J., Neumann, R., Boes, K., & Ebner-Priemer, U. (2016). Does a 20-week aerobic exercise training programme increase our capabilities to buffer real-life stressors?: A randomized, controlled trial using ambulatory assessment. *European Journal of Applied Physiology, 116*(2), 383–394.

Vukovic, J., Colditz, M. J., Blackmore, D. G., Ruitenberg, M. J., & Bartlett, P. F. (2012). Microglia modulate hippocampal neural precursor activity in response to exercise and aging. *Journal of Neuroscience, 32*(19), 6435–6443.

Vysniauske, R., Verburgh, L., Oosterlaan, J., & Molendijk, M. L. (2016). The effects of physical exercise on functional outcomes in the treatment of ADHD: A meta-analysis. *Journal of Attention Disorders, 24*(5), 644–654.

Walker, E. R., McGee, R. E., & Druss, B. G. (2015). Mortality in mental disorders and global disease burden implications: A systematic review and meta-analysis. *JAMA Psychiatry, 72*(4), 334–341.

Wang, D., Wang, Y., Wang, Y., Li, R., & Zhou, C. (2014). Impact of physical exercise on substance use disorders: A meta-analysis. *PLOS ONE, 9*(10), e110728.

Wang, P. W., Lin, H. C., Su, C. Y., Chen, M. D., Lin, K. C., Ko, C. H., et al. (2018). Effect of aerobic exercise on improving symptoms of individuals with schizophrenia: A single blinded randomized control study. *Frontiers in Psychiatry, 9,* 167.

Wegner, M., Helmich, I., Machado, S., Nardi, A. E., Arias-Carrion, O., & Budde, H. (2014). Effects of exercise on anxiety and depression disorders: Review of meta-analyses and neurobiological mechanisms. *CNS and Neurological Disorders: Drug Targets, 13*(6), 1002–1014.

Weiss, E. R. (2018). Supervised exercise in anorexia nervosa treatment: Directions for research and recovery. *The New School Psychology, 15*(1), 20–38.

Weybrew, B. B. (1963, November 12). *Autonomic resiliency, subjective symptomology, and submarine stress* (Memorandum Report No. 63-12). New London, CT: Naval Medical Research Laboratory.

Wigal, S. B., Emmerson, N., Gehricke, J. G., & Galassetti, P. (2013). Exercise: Applications to childhood ADHD. *Journal of Attention Disorders, 17*(4), 279–290.

Wipfli, B. M., Rethorst, C. D., & Landers, D. M. (2008). The anxiolytic effects of exercise: A meta-analysis of randomized trials and dose–response analysis. *Journal of Sport and Exercise Psychology, 30*(4), 392–410.

Wright, K., Armstrong, T., Taylor, A., & Dean, S. (2012). "It's a double-edged sword": A qualitative analysis of the experiences of exercise amongst people with bipolar disorder. *Journal of Affective Disorders, 136*(3), 634–642.

Wright, K. A., Everson-Hock, E. S., & Taylor, A. H. (2009). The effects of physical activity on physical and mental health among individuals with bipolar disorder: A systematic review. *Mental Health and Physical Activity, 2*(2), 86–94.

CHAPTER 22

Wearing the Clinical Hat

Paul M. Lehrer
Robert L. Woolfolk

As researchers, we most often randomize assignment to treatments in order to attenuate the effects of individual variance and analyze group means looking for methods that are, on average, effective at reducing stress. When confronted with an ideograph, an individual human being and a patient, the task is entirely different. In the clinical arena, we call upon different skills and employ a somewhat different approach to achieve a different goal. We want to find out what it takes to ameliorate the stress, but our focus is often broader (Woolfolk, 2015). Stress may be only one aspect of the complex lifeworld of the patient. It is a lifeworld we must comprehend, unless we are functioning adjunctively, only in the role of a "stress management consultant," concurrent with another clinician being the primary therapist. Most typically, we are the only professional offering the patient psychosocial therapy. It is likely that we are engaged in a complex sea of life problems that may or may not be intertwined with stress (Woolfolk & Allen, 2006). The two of us have a combined century of clinical experience over the past 50 years. This period spans the time when psychoanalysis ruled psychiatry, through the biomedical revolution in psychiatry and the advent of cognitive-behavioral therapy, to the present day in which diagnosis is being rethought and empirically supported treatments have emerged from a third generation of behavior therapy. In this chapter, our goal is to boil down the accumulated scientific findings about helping people manage stress, factor these findings, and from them provide some rules of thumb, some bit of clinical tradecraft that will enable clinicians to make optimal use of the tools provided in this volume.

What follows are some principles that we have developed:

1. One size does not fit all. Each person is unique. Creative modifications of research protocols are to be encouraged if they are effective with a given patient.
2. Some trial and error is part of the therapeutic process. Fitting stress management into the patient's life may require some ingenuity.

3. A person experiencing stress is a complex organism, and a psychobiosocial perspective is likely to be optimal.
4. The therapist must comprehend a patient's lifeworld, his or her patterns of cognition and emotion, and the totality of the life situation, including, but not limited to, interpersonal relationships, work, health, socioeconomic condition, current events, and pastimes.

DIFFERENT STROKES FOR DIFFERENT FOLKS

For various and often idiosyncratic reasons, some people find particular stress management methods appealing and useful, while they instinctively dislike others. Some people may particularly value or devalue meditation or Qigong because of their ethnic or family values or because their own personal spiritual pathways may have led them toward or away from an interest in the Asian religious traditions from which mindfulness and mantra meditation or Qigong originated. Some people think about their body experiences more than others do and may have received training in music, dance performance, or athletics or may have a more general interest in physical fitness. These individuals may find it both easier and more conducive to engage in exercise or muscle relaxation training. People with a musical background are often particularly moved by music therapy. People who are more highly hypnotizable tend to have a more positive attraction to hypnosis or autogenic training. They may tend to gravitate toward methods emphasizing personal experience over more objective methods of controlling particular responses, such as biofeedback, at least in initial sessions (Wickramasekera, 2005). They often do poorly with biofeedback methods that emphasize objective measures more than inner experience. People with an interest in science or biomechanics may have a particular interest in various kinds of biofeedback or neurofeedback and often particularly dislike hypnotic methods, thinking them too ephemeral, and do poorly with them. People who are more psychologically minded often find cognitive therapy the most meaningful. All of these relationships have been described in previous chapters. Methods that have particular personal appeal and are easily learned tend to be practiced more than methods that are difficult, aversive, or simply unappealing.

A useful analogy is aerobic exercise. For many people, running is the most efficient, straightforward, and convenient exercise. But many people hate to run. Prescribing running to people who will not run is folly. Exercise only benefits people who do it. The same is true of other stress management methods. There are many forms of aerobic exercise and many forms of stress management. A key to being effective in both realms is finding methods that are appealing enough to the patient that the patient will perform them and continue to do so over time.

Put another way, methods that are actually used by people are the ones that work. Some people are willing and able to practice any or all of the methods described in this book. Others will gravitate to some but not to others. For this reason alone, it is incumbent on the practitioner to have expertise in a variety of stress management methods and to use methods likely to be most effective and most appealing for each client. Sometimes a variety of methods can be used. Sometimes a hybrid, individualized combination of features from different methods can be effective. I (RLW) have found that the physical focus of autogenic training, combined with a breath meditation, can be useful in quickly achieving a relaxed state, such that patients may either use the combination or may, based on its effect, become open to experimenting with different methods.

Also, although relatively rare, various stress management methods may have some side effects, which tend to differ among them. Most of the side effects are transient and can be managed by adjusting the technique, although sometimes they may be sufficiently aversive to require changing methods. For example, hyperventilation symptoms sometimes occur in some of the breathing methods; exacerbations of pain or negative emotions with autogenic training, meditation, eye movement desensitization and reprocessing (EMDR), or Qigong; pain or fatigue from some types of yoga positions or physical exercise; and feelings of invalidation from cognitive therapy. The fact that the various methods have different side-effect profiles supports our recommendation that a practitioner will be more effective to the extent that he or she is competent in applying several of the methods described in this book.

In our own clinical practices, we use multiple strategies for picking the methods to use with each person. First, we often look at the person's presenting problem. The person may find particular meaning in a method that directly addresses the problem as he or she experiences it. If the person complains of headaches or various muscular aches and pains, we may suggest muscle relaxation or electromyographic biofeedback; for concentration difficulties, neurofeedback; for hyperventilation symptoms, one of the breathing methods; for worry symptoms, cognitive therapy or mantra meditation; for posttraumatic stress disorder (PTSD) symptoms, EMDR; and so forth. These are not definitive choices, however, because some individuals may not gravitate toward or do well with one or another of these methods or may have a particular preference for another. In fact, although each of the methods may have an especially strong effect on some specific symptoms, all of them have widely generalized effects on stress and may affect all of the symptoms and mechanisms described in this book. Thus muscle relaxation, neurotherapy, EMDR, music, mindfulness, and cognitive therapy all have pronounced effects on the autonomic and immune systems, even though these effects are only indirectly targeted by the methods. Similarly, muscle relaxation and biofeedback may reduce worry activity, whereas cognitive therapy and hypnosis can relax the muscles. Because some methods may have more direct effects on some symptoms than others, we often combine techniques and teach more than one to an individual. Personal preferences also are key. Some people are intrigued by hypnosis, mindfulness, or yoga, but some are averse. Some people like the idea of objective physiological feedback from a physiological monitor; others prefer to focus on cognitions or inner mental or physical experience. In our practice, we often begin stress management therapy by describing several methods, relating the discipline, length of time, and type of home practice needed for each, and, following the client's preferences for the first method we choose, often give guidance based on our observations of the client's symptoms and personality.

Despite some rough guidelines, there is an element of trial and error in finding the right fit between person and stress management technique, and, despite demonstrated efficacy, one cannot know in advance whether a particular method will work on the next particular person you see. Often it is useful to ask patients what activities have in the past created relaxation or freedom from stress. Sometimes an activity will emerge from this query that can be employed or adapted into a stress management regimen. Doing crossword puzzles, gardening, arranging a stamp collection, fishing, bird watching, and even routine housework have emerged as activities that work for some people. Distraction and escape from stressors can be achieved through many activities, some of which involve some elements of the tested techniques presented in this book. During 50 years of working with people with stress, we have found that many males who will not "sit still" for the validated approaches find weightlifting not only relaxing but also something they will commit to on a frequent enough basis to provide significant benefits. No controlled studies testing this impression are available, but, as Paul Meehl (1997) once wrote, some phenomena can be credible without the existence of controlled studies.

Some of the observations made here have been quantified in observations from my (PML) students in a stress management class. In each session, one of the methods described here was presented. At the end of the course, students were asked to rank-order the methods in terms of personal usefulness for managing stress. The results are shown in Table 22.1. There was a large disparity among students and among methods, with a substantial standard deviation in ranks among methods. This validates our hypothesis of "different strokes for different folks." Overall, methods did differ from each other. (A repeated-measures analysis of variance [ANOVA] was performed and yielded significant effect across seven interventions, $F(6,96) = 5.96$, $p < .0001$, including Huynh–Feldt correction for sphericity.) Students tended to find progressive muscle relaxation and breathing methods most useful, while finding autogenic training and hypnosis least useful. However, students' self-ratings of improvement in stress after daily practice of these hypnotic techniques were highly correlated with scores on the Stanford Clinical Scale of Hypnotic Susceptibility, $r = .52$, $p < .03$, suggesting that the hypnotic methods were particularly useless to the majority of students with low to moderate hypnotic susceptibility.

Finally, a close reading of this book will show a different pattern of side effects of the various methods. The "autogenic discharges" that occur in autogenic training rarely occur with biofeedback or progressive muscle relaxation. People may incorrectly practice the progressive relaxation method and strain some muscles or tendons during the "tense–relax" training periods, and some medications, such as phenothiazines, may diminish sensations and control of the muscles, rendering this method rather frustrating. Exercising too rigorously could produce physical problems in people with musculoskeletal or cardiovascular problems. Straining too much during yoga can also produce musculoskeletal problems. People may hyperventilate, particularly during initial practice of breathing techniques or some meditative methods involving breathing. EMDR involves exposure to intense feelings and memories, and some people may not be able to process them appropriately. Overtraining of particular brain regions or mistaken emphasis on the wrong regions can be a particular side effect of neurofeedback. Increased body awareness can produce transient body anxiety, an occurrence that is mentioned more frequently in the autogenic training, meditation, and breathing literature. Growing awareness of muscle

TABLE 22.1. Ranked Usefulness of Stress Management Techniques in a Class of 17 Freshmen Exposed to All

Method	Mean	*SD*	Minimum	Maximum
Progressive muscle relaxation	2.4	1.5	1	6
HRV biofeedback/breathing	3.1	1.4	1	7
Mindfulness	3.3	1.8	1	7
Knowledge of stress effects	4.5	2.0	1	7
Cognitive restructuring	4.6	1.8	1	7
Autogenic training	4.7	1.8	1	7
Hypnosis	5.4	1.8	1	7

Note. 1, most useful; 7, least useful; HRV, heart rate variability.

sensations can lead to feelings of muscular discomfort during early progressive relaxation sessions, when some muscle groups have not yet been trained. Personality changes described for mantra meditation, even when generally beneficial, may destabilize some family and personality system patterns and cause adjustment problems. Mindfulness may elicit destabilizing experiences for people with schizophrenia. Reemergence of dormant symptoms, transient increases in pain, and even psychotic experiences have been reported in Qigong. People can experience invalidation and a jolt to self-esteem from disputations in cognitive therapy. People with low hypnotizability may find hypnotic inductions aversive. Although the various side effects can render some of the methods ineffective or, in rare cases, harmful, the problems can mostly be mitigated or eliminated in the hands of skilled and experienced clinicians. Nevertheless, difficulties with one method in a particular person might indicate that a shift to another method would be beneficial.

LIMITATIONS OF RESEARCH METHODOLOGY AND THE IDIOGRAPHIC NATURE OF PROBLEMS

This book has focused on empirically validated treatments and has summarized effects of each method on a variety of problems and disorders. For the clinician, this information is only partially useful. None of the methods has been fully evaluated for the treatment of the full gamut of problems that the clinician will see, and almost none of them have been evaluated for treating the full complexity of problems and personality styles that almost all clients present. The drug industry has a defined methodology for evaluating the effects of a method on the broad array of people having a particular problem. First, the drug is evaluated in an uncontrolled trial for safety and feasibility among a small number of highly screened and motivated people. This is called a "phase I trial." Then the drug is evaluated in a highly controlled trial against a placebo among patients who are particularly motivated to adhere to treatment regimens, screening out people with various comorbid disorders. These are called "phase II trials." Virtually all of the research on the methods described in this book involve either phase I or phase II trials. However, before a drug can be put to market, it must be evaluated in a large population of ordinary people in the usual clinical setting, without particular compensation or motivation to take the drug other than that a physician may recommend taking part in the trial. The patient must grant informed consent (itself an inestimable form of cognitive dissonance bias, favoring the drug). These are phase III trials, and they must be carried out on thousands or tens of thousands of individuals in order to find effects in the general population. Phase III trials inevitably include people who may not take the medication as prescribed or may find unpleasant or even dangerous side effects due to personal physical or emotional idiosyncrasies or comorbidities. Such trials often cost tens of millions of dollars, money that is almost never available for the methods described in this book and for which no corporation could hope to recoup the investment costs.

Additionally, even if phase III trials were available for some of the methods and if effect sizes were relatively large, statistical results never are sufficient to completely determine the best approach with an individual client. Not all individuals in a clinical trial are helped, and a few are hurt. Not all methods will be practiced by everyone, and not all of them can be learned by everyone. For this reason, clinical practice is an art, not a science or a field of engineering. Indeed, for many individuals, just the warm and understanding interest of another individual may be as strong a therapeutic influence as any of the methods described in this book. Although sometimes dismissed as a "placebo," "expectancy," or "suggestion" effect, these effects should not, in fact, be dismissed. They are large, often accounting for a larger proportion of therapeutic effect than the incremental effect of a real treatment method, chemical or psychological, particularly for problems involving emotion or stress. None of the methods has the huge effects of antibiotics on infectious disease, although not even these are 100% effective, and they require art on the part of the physician in prescribing and monitoring effects.

Finally, not all methods for stress management used throughout the world are included in this volume. We excluded some because we knew that the volume of empirical literature to support them was too small. This is not to say that the methods are less effective, but only to say that they have not yet been sufficiently studied in controlled trials of any sort. We also find new methods almost daily that all seem to have a stress management effect, from adjusting posture to adopting a religious belief or learning to play a digeridoo. Future volumes will undoubtedly include new methods.

BIOLOGICAL BARRIERS TO STRESS MANAGEMENT

Treating the whole person requires a contextualist approach. We must understand the roles that people play in their lives and identify what stressors exist. Making certain that a patient has a clean bill of somatic health and functioning is critical. Conditions affecting the nervous system can have a direct effect on stress symptoms, and conditions impairing other body systems can similarly impair allostatic capacity, increase vulnerability to stress, and themselves constitute sources of stress.

Indeed, volumes have been written on biological and genetic contributions to stress vulnerability. As developmental research has found, some infants are born more physiologically reactive than others and more prone to later development of emotional disorder. Also stress symptoms are often transmitted through generations, probably with an epigenetic, as well as experiential, contribution (Bergman, Ludwig, Westrup, & Welch, 2019; Bleker et al., 2018; Jones et al., 2019). Genetic components in certain severe emotional disorders such as schizophrenia and bipolar disorder have been well established (Prata, Costa-Neves, Cosme, & Vassos, 2019). Heredity plays a role in anxiety and mood disorders (Bengtson, Aamodt, Vatn, & Harris, 2015; McGuffin, Katz, & Bebbington, 1987). Over the last five decades, pharmacotherapy has superseded psychosocial interventions as the treatment of choice. Although it is unquestionably the case that many individuals suffering from stress, as well as diagnosed mental disorders, have benefited from drug treatment, the predominance of drug therapy has not brought about the efficacious era that its early adherents had predicted. In a rather stunning development, sparked by European research (Seikkula et al., 2006; Wunderink, Nieboer, Wiersma, Sytema, & Nienhuis, 2013), it is now recognized (Insel, 2013; McGorry, Alvarez-Jimenez, & Killackey, 2013) that even in schizophrenia overreliance on medication maintenance can have adverse iatrogenic effects, with less recovery shown than with treatment approaches having a strong psychosocial emphasis. Disaffection with pharmacotherapy has been expressed in the statements of two recent directors of the National Institute of Mental Health (Insel, 2012; Hyman, 2012).

For other, less severe disorders, it is often less clear whether drug treatment should ever be used prior to or in preference to psychological intervention, although this is, in fact, a common practice. Although drug treatment is less labor-intensive and sometimes cheaper than psychological treatment, it is also sometimes riskier, with more unwanted side effects. For most emotional disorders amenable to drug treatment, there seems to be a consensus that the combination of both types of intervention produces the best and most long-lasting results. However, for many milder anxiety or mood disorders, a psychological monotreatment, including stress management components, is equivalent and perhaps preferable, in view of its fewer side effects and absence of problems analogous to abstinence syndrome associated with many drugs (Cuijpers et al., 2013; DeRubeis, Siegle, & Hollon, 2008).

IMPORTANT TOPICS NOT COVERED IN THIS BOOK

This book has concentrated on psychophysiological and cognitive methods for controlling the stress response. In doing this, we have not discussed the *sources* of stress, about which many volumes have already been written. The most potent sources of allostatic overload and more severe stress reactions are well known, including war and/or natural disasters; stress among caretakers of dementia patients or people with severe mental or physical disabilities; or victimhood from assault, robbery, sexual, racial, or religious discrimination, emotional, physical, or sexual abuse, or poverty. Although severe and often public and easy to see, the emotional damage done by these conditions may be so severe that victims often do not wish to talk or even think about them, a reluctance that often in itself leads to exacerbation of traumatic stress symptomatology. Here the therapist's first task is often just to allow the client to approach the topic and share his or her experiences. When needed, other methods described in this book can be useful at a later stage of treatment. Milder stress reactions, often from the stressors of modern life, may be more amenable to these methods from the start, although these, too, often require hours of empathetic discussion of how the stress occurs and ways of managing it. Such stressors include marital and interpersonal conflict, pressures of work and family, uncertainties about employment, management of important life transitions (e.g., from home to college, college to work, job changes, retirement), managing health changes, relationship changes (e.g., marriage, divorce, bonding, breakups), and similar life events. There are large specialized literatures on all of these topics. These topics often constitute the core of psychotherapeutic treatment. As recent American Psychological Association surveys find, even global and national problems present significant sources of stress, including degradation of the environment and various political uncertainties. As this writing, the coronavirus pandemic is raging throughout the world causing untold amounts of stress from fear of the disease, loss of income, and the isolation caused by necessary lockdowns. Coping with these stressors can also arise as therapeutic issues. Think here of family separation, housing loss or dislocation, common prejudices, and changes in residence, stage of life, or lifestyle. Some clinical wisdom can be very helpful in assisting individuals in identifying and modifying a stress-inducing lifestyle. Prevention, as well as tertiary intervention, is important in stress management.

Helping people find and utilize natural stress buffers must also often be a source of therapeutic concern. How does one deal with social isolation, sometimes resulting from relocation to a new community, sometimes from lack of social skills or emotional barriers, sometimes from lack of community resources or ways of finding them? How do people find beauty, meaning, relationships, companionship, means of legal, technical, or financial advice and support? Where do we find solace when friends or family members are far away, dysfunctional, or deceased? How does one find social skill and emotional fortitude when deprived of care in childhood without adequate models, instruction, protection, or support?

Nevertheless, as literature reviewed in this volume shows, adding treatment components specifically directed at stress management can provide significant therapeutic gains. Integrating stress management methods into treatment for coping with these stress sources can and should be the topic of another book.

REFERENCES

Bengtson, M. B., Aamodt, G., Vatn, M. H., & Harris, J. R. (2015). Co-occurrence of IBS and symptoms of anxiety or depression, among Norwegian twins, is influenced by both heredity and intrauterine growth. *BMC Gastroenterology, 15,* 9.

Bergman, N. J., Ludwig, R. J., Westrup, B., & Welch, M. G. (2019). Nurturescience versus neuroscience: A case for rethinking perinatal mother–infant behaviors and relationship. *Birth Defects Research, 111*(15), 1110–1127.

Bleker, L. S., van Dammen, L., Leeflang, M. M. G., Limpens, J., Roseboom, T. J., & de Rooij, S. R. (2018). Hypothalamic–pituitary–adrenal axis and autonomic nervous system reactivity in children prenatally exposed to maternal depression: A systematic review of prospective studies. *Neuroscience and Biobehavioral Reviews, 23,* 23.

Cuijpers, P., Sijbrandij, M., Koole, S. L., Andersson, G., Beekman, A. T., & Reynolds, C. F., III. (2013). The efficacy of psychotherapy and pharmacotherapy in treating depressive and anxiety disorders: A meta-analysis of direct comparisons. *World Psychiatry, 12*(2), 137–148.

DeRubeis, R. J., Siegle, G. J., & Hollon, S. D. (2008). Cognitive therapy vs. medications for depression: Treatment outcomes and neural mechanisms. *Nature Reviews Neuroscience, 9*(10), 788–796.

Hyman, S. E. (2012). Revolution stalled. *Science Translational Medicine, 4.* Retrieved from *http://stm.sciencemag.org/content/4/155/155cm11.full.*

Insel, T. R. (2012). Next-generation treatments for mental disorders. *Science Translational Medicine, 4,* 1–9.

Insel, T. R. (2013, April 29). Director's blog: Transforming diagnosis. Retrieved October 15, 2013, from *www.nimh.nih.gov/about/director/2013/transforming-diagnosis.shtml.*

Jones, C. W., Esteves, K. C., Gray, S. A. O., Clarke, T. N., Callerame, K., Theall, K. P., et al. (2019). The transgenerational transmission of maternal adverse childhood experiences (ACEs): Insights from placental aging and infant autonomic nervous system reactivity. *Psychoneuroendocrinology, 106,* 20–27.

McGorry, P., Alvarez-Jimenez, M., & Killackey, E. (2013). Antipsychotic medication during the critical period following remission from first-episode psychosis: Less is more. *Journal of the American Medical Association Psychiatry, 70,* 898–900.

McGuffin, P., Katz, R., & Bebbington, P. (1987). Hazard, heredity and depression: A family study. *Journal of Psychiatric Research, 21*(4), 365–375.

Meehl, P. M. (1997). Credentialed persons, credentialed knowledge. *Clinical Psychology: Science and Practice, 4,* 91–98.

Prata, D. P., Costa-Neves, B., Cosme, G., & Vassos, E. (2019). Unravelling the genetic basis of schizophrenia and bipolar disorder with GWAS: A systematic review. *Journal of Psychiatric Research, 114,* 178–207.

Seikkula, J., Aaltonen, J., Alakare, B., Haarakangas, K., Keränen, J., & Lehtinen, K. (2006). Five-year experience of first-episode nonaffective psychosis in open-dialogue approach: Treatment principles, follow-up outcomes, and two case studies. *Psychotherapy Research, 16,* 214–228.

Wickramasekera, I. E., II. (2005). Best of both worlds: How to integrate hypnosis and biofeedback with empathy and hypnotic assessment procedures. *Biofeedback, 33*(1), 31–34.

Woolfolk, R. L. (2015). *The value of psychotherapy: The talking cure in an age of clinical science.* New York: Guilford Press.

Woolfolk, R. L., & Allen, L. A. (2006). *Treating somatization: A cognitive-behavioral approach.* New York: Guilford Press.

Wunderink, L., Nieboer, R. M., Wiersma, D., Sytema, S., & Nienhuis, F. J. (2013). Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation or maintenance treatment strategy: Long-term follow-up of a 2-year randomized clinical trial. *Journal of the American Medical Association Psychiatry, 70,* 913–920.

Index

Note. *f, n,* or *t* following a page number indicates a figure, note, or a table.

Abdominal breathing, 338–339. *See also* Breathing exercises
Abdominal pain, 141–143. *See also* Gastrointestinal problems; Irritable bowel syndrome (IBS); Pain
Absorption, 45, 389
Acceptance
  cognitive therapy model and, 520
  helpful strategies for coping with stress and, 512–513
  mantra meditation and, 387
  persistence and, 494
  relaxation/meditation/mindfulness (RMM) construct and, 46
  self-acceptance, 419, 425*t*, 430–431
Acceptance and commitment therapy (ACT), 140, 220, 414*t*, 419
Acetylcholine (ACh), 64, 80–81, 619. *See also* Neurotransmitters
ACTH. *See* Adrenocorticotropic hormone
Acute stressors, 96*t*, 97. *See also* Stressors
Addiction. *See also* Substance use/misuse
  EMDR therapy and, 196*t*
  exercise and the walk–talk approach and, 625*t*, 627
  mantra meditation and, 387
  mindfulness meditation and, 424*t*, 429–430
  Qigong therapy and, 459*t*
  respiratory sinus arrhythmia and, 267
Adherence, 178–179, 398–401
Adolescents. *See* Youth stress
Adrenocorticotropic hormone (ACTH), 67–69, 79, 135–136, 610*t*
Aerobic exercise, 25, 144, 266, 457, 557, 577, 606–630, 634, 610*t*–617*t*, 618*f*, 650. *See also* Exercise; Walk–talk approach
Affect, 384*t*, 388
Aftereffect, 52–53, 619
Agoraphobia, 427
AIP model, 195
Air passage, 337–338. *See also* Breathing exercises
Alcohol use/abuse. *See* Addiction; Substance use/misuse
Alertness, 244
Alimentary problems, 169*t*
All-or-nothing thinking. *See* Dichotomous thinking
Allostasis, 4, 24–28, 268
Allostatic load/overload, 24–28, 29, 62, 221–223
Alpha brain-wave frequencies, 214–215, 218–219, 227, 232–236, 239, 243–247, 257–258, 380–382, 386, 426, 479, 531, 547
Alzheimer's dementia, 64, 81
Amino acid transmitters, 63–64. *See also* Neurotransmitters
Amygdala
  mindfulness and, 421
  neurofeedback (NFB) and, 221–224, 222*f*, 223*f*, 225*f*, 227, 231
  overview, 40, 66–71, 79, 95, 221, 231, 277, 609
  posttraumatic stress disorder (PTSD) and, 226–227

Analytically oriented music therapy, *598*. *See also* Music in relation to stress management
Anger. *See also* Hostility
automatic thoughts and, 498, *507f*
exercise and, 608
eye movement desensitization and, 201, 204
mantra meditation and, *384t*
mindfulness meditation and, 420, 421, *422t*, *424t*, 428, 435
music and, *595*
overview, *17t*, 41, 250, 350, 388, 520
progressive relaxation and, 106, 167, 181
Antidepressants, 234–235
Antigen presenting cell (APC), 73
Anti-inflammatory mechanisms, 60, 74–76, *617t*. *See also* Inflammation
Anxiety. *See also* Anxiety disorders
automatic thoughts and, 498
baroreflex (BR) activity and, 272
cytokine immunotherapy and, 59
exercise and the walk–talk approach and, *624t*, 626–627
heart rate variability biofeedback (HRVB) and, 280
mantra meditation and, *384t*, 385–386, 390
mindfulness meditation and, 421, *424t*, 427, 428, 434
music in relation to stress management and, *596*, *597t*
neurofeedback (NFB) and, 215, 244
progressive relaxation therapy and, 169–170, *169t*
Qigong therapy and, *459t*
relaxation-induced, 350
respiratory sinus arrhythmia and, 267
sighing and, 28
whole-body breathing and, 345–346
yoga and, 475–476
Anxiety disorders. *See also* Anxiety; Generalized anxiety disorder; Panic disorder; Phobias and phobic disorders; Posttraumatic stress disorder (PTSD) and symptoms
breathing retraining and, 103
mindfulness meditation and, 427
progressive muscle relaxation (PMR) and, 106
treatments and, 19–20
whole-body breathing and, 345–346
Appraisals
helpful strategies for coping with stress and, *510f*, 512–513
integrating mindfulness into cognitive therapy and, 513–518
problematic strategies for coping with stress and, 510–512, *510f*
schemas and, 505–510, *506f*, *507f*, *509f*
Approach-based responses to stress, 94–95
Arthritis, *459t*
Ashtanga yoga, 474–475. *See also* Yoga
Assertiveness, 117–118. *See also* Communication skills
Assessment
autogenic training (AT) method and, 532–534
EMDR therapy and, 197–199, 200–201, 204–205
heart rate variability biofeedback (HRVB) and, 279, *280t*
hypnosis and, *558–569*
mantra meditation and, 384–390, *384t*
mindfulness meditation and, 432–433
neurofeedback (NFB) and, 217–218, *218f*, *219f*, 231–238, *239f*
progressive relaxation therapy and, 165–170, *169t*
relaxation/meditation/mindfulness (RMM) construct and, 53, 54–55
single-channel EEG assessment and, 217–218, *218f*, *219f*
walk–talk approach and, 633–634
whole-body breathing and, 342–346, *343f*, *347t*, 351–356, *352t*–*353t*, *354f*, *355f*
Asthma
capnometry-assisted respiratory training (CART) and, 312–31
case example of, 315–322, *320f*, *321f*
caveats and contraindications for, 314–315
empirical support for, 307–314, *308f*, *310t*–*311t*
heart rate variability biofeedback (HRVB) and, 276–277, 280
history of, 303–304
hypocapnia and, 305–307
overview, 303–304
Qigong therapy and, *459t*
rationale and empirical foundations of, 304–307
Attention, 45, *424t*
Attentional/cognitive domain in the multidomain model, 421–432, *422f*
Attention-deficit/hyperactivity disorder (ADHD)
EEG assessment and, 233, 234–235
exercise and the walk–talk approach and, *624t*–*625t*, 629
mindfulness meditation and, *424t*, 426
neurofeedback (NFB) and, 216–217, 218, *219f*
posttraumatic stress disorder (PTSD) and, 226
Attribution style, 493–494
Autism spectrum disorders, 61, 81–82
Autogenic training (AT) method
assessment and, *532–534*
autogenic discharges *531–534*, *652*
case example of, *544–545*
clinical applications of, *543–547*, *547t*
history of, *527–528*
method, *535–543*
overview, *527*, *548–549*
research regarding, *545–547*, *547t*
theoretical underpinnings of, *528–532*
Autoimmune processes, 26–27
Automatic thoughts. *See also* Negative thoughts; Thought processes; *individual categories of*
examining and modifying, 503–505, *505t*
integrating mindfulness into cognitive therapy and, 513–518
list of, *499t*
overview, 498–501, *499t*, *501t*–*502t*
schemas and, 505–510, *506f*, *507f*, *509f*
youth stress and, 110–111
Autonomic activity, 22
Autonomic balance, 266–268
Autonomic dysregulation, 26. *See also* Dysregulation

Autonomic nervous system (ANS). *See also* Parasympathetic nervous system (PNS); Sympathetic nervous system (SNS)
  assessment and, 237–238, 239*f*
  cytokines and, 60–61, 70
  exercise and the walk–talk approach and, 608
  mindfulness meditation and, 424*t*
  neurofeedback (NFB) and, 231
  polyvagal theory and, 228, 228*f*
  posttraumatic stress disorder (PTSD) and, 227
  youth stress and, 94–95
Autonomic regulation, 268–270
Avoidance, 94–95, 511
Awareness
  breathing and, 334–335
  integrating mindfulness into cognitive therapy and, 513–515
  mindfulness meditation and, 415–418, 417*t*
  neurofeedback (NFB) and, 244–245
  relaxation/meditation/mindfulness (RMM) construct and, 46
  whole-body breathing and, 348–349

Baroreflex (BR) activity
  complexity of, 268–270
  heart rate variability biofeedback (HRVB) and, 264, 309 272–275, 274*f*
  overview, 23–24, 159, 265–266, 271–272, 372, 619
Basic Quiet Focus level of RMM, 45–46
Basic Relaxation level of RMM, 43–45
Behavior, 27, 65, 424*t*, 428–430. *See also* Regulation
Behavioral activation, 512–513
Behavioral domain in the multidomain model, 421–432, 422*f*
Behavioral interventions, 19, 21
Beliefs, conditional. *See* Conditional beliefs
Beliefs, core. *See* Schemas
Benenzon music therapy, 598. *See also* Music in relation to stress management
Beta brain-wave frequencies, 214, 215–220, 229–230, 233–236, 239, 241, 244–254, 257, 477*t*, 531
Bhakti yoga, 474. *See also* Yoga
Bilateral stimulation, 201–202
Biofeedback. *See also* Biofeedback-based stress management model; Heart rate variability biofeedback (HRVB); Neurofeedback (NFB)
  assessment and, 237–238
  case example of, 254–258, 254*f*, 256*f*, 258*f*
  compared to autogenic training and, 529
  overview, 258–259
  side effects and contraindications, 241
Biofeedback-based stress management model. *See also* Biofeedback
  IBS and abdominal pain and, 142
  overview, 136–141, 138*f*, 145
  side effects of, 145
Biological factors, 61–71, 94–95, 654–655. *See also* Brain functioning and structures; Immune system; Inflammation
Bipolar depression, 623, 625*t*, 626. *See also* Depression
Black-or-white thinking. *See* Dichotomous thinking
Blaming, 499*t*, 500, 502*t*. *See also* Automatic thoughts
Blank-out effect, 381–382
Blood pressure. 4, 11, 21–24, 134, 139, 159, 166, 172*t*, 179, 228, 265, 269, 271–273, 275–276, 278, 281–283, 287, 314, 350, 386, 465, 466*t*, 477, 479, 531, 544–545, 592–593, 597*n*, 611*t*–612*t*, 626. *See also* Hypertension
Body image, 196*t*
Body scan, 203, 418
Body-focused practices, 418
Bohr effect, 330
Bonny method of guided imagery and music, 597–598. *See also* Music in relation to stress management
Brain functioning and structures. *See also* Biological factors; Immune system
  exercise and the walk–talk approach and, 608–622, 610*t*–617*t*, 618*f*
  hypnosis and, 560–561, 581*n*
  hypothalamic–pituitary–adrenal (HPA) axis, 66–69, 78–81
  inflammation and, 61, 83–84
  mantra meditation and, 382–383
  microglial cells and, 81–83, 82*f*
  mindfulness meditation and, 420–421
  music in relation to stress management and, 592, 594–595
  neurofeedback (NFB) and, 221–224, 222*f*, 223*f*, 225*f*
  neurotransmitters and, 63–66
  posttraumatic stress disorder (PTSD) and, 225–228
  relevance of cytokines and, 61–71
  single-channel NFB and, 230–231
  walk–talk approach and, 607
  yoga and, 475
  youth stress and, 94–95
Brain-derived neurotropic factor (BDNF)
  exercise and the walk–talk approach and, 613*t*–614*t*, 619
  walk–talk approach and, 607
  youth stress and, 115–116
Brain-wave training, 214–215. *See also* Neurofeedback (NFB)
Breathing. *See also* Breathing exercises
  autogenic training (AT) method and, 539
  breath awareness, 417*t*
  definitions of, 334–335
  respiratory dysregulation and, 27–28
  sighing and, 28
  strategies for breathing regulation and, 335–340
  systems perspective and, 331–334, 332*f*
Breathing exercises. *See also* Breathing; Breathing retraining; Capnometry-assisted respiratory training (CART); Emotion-focused stress management strategies; Hypocapnia; Mindfulness meditation; Respiratory retraining; Whole-body breathing
  asthma and, 305–307
  heart rate variability biofeedback (HRVB) and, 277, 282–286

Breathing exercises *(cont.)*
instructions for, 352*t*–353*t*, 356–364
neurofeedback (NFB) and, 243
progressive muscle relaxation (PMR) and, 108, 109
relaxation/meditation/mindfulness (RMM) construct and, 50
strategies for breathing regulation and, 335–340
youth stress and, 103–106, 108, 109
Breathing retraining. *See also* Breathing exercises; Capnometry-assisted respiratory training (CART); Respiratory retraining; Whole-body breathing
asthma and, 305–307
progressive muscle relaxation (PMR) and, 108, 109
youth stress and, 103–106, 108, 109
"Broaden-and-build" hypothesis, 49
Brodmann areas (Bas), 224
Buddhism
mindfulness meditation and, 413, 415, 435
Qigong therapy and, 451–452, 453
Buffering factors, 29
Burnout, 4, 345–346, 347*t*
Buteyko method, 306

Calmness, 245
Cancer care, 459*t*, 596, 597*t*. *See also* Pain
Capnometry-assisted respiratory training (CART). *See also* Breathing retraining; Hypocapnia
asthma and, 312–314
case example of, 315–322, 320*f*, 321*f*
caveats and contraindications for, 314–315
overview, 305, 307
panic disorder and, 308–312, 310*t*–311*t*
protocol for, 307–308, 308*f*
Cardiovascular system. *See also* Heart rate variability (HRV)
baroreflex (BR) activity and, 272
cardiac arrhythmia, 351
cardiac vagal tone and, 267
exercise and the walk–talk approach and, 611*t*
mantra meditation and, 384*t*
mindfulness meditation and, 424*t*
music in relation to stress management and, 596, 597*t*
overview, 264–265
Qigong therapy and, 459*t*
resonance and, 272–273
Catastrophizing. *See also* Automatic thoughts
examining and modifying, 503
example of, 500
overview, 499*t*
techniques for reversing, 501*t*
youth stress and, 111
Catecholamines, 4
Cathartic responses, 350
Central nervous system (CNS)
cytokines and, 60–61, 75, 78–79
hypnosis and, 556–557
inflammation and, 59
neurofeedback (NFB) and, 224
neuroinflammation and, 60
posttraumatic stress disorder (PTSD) and, 227
relevance of cytokines and, 61–71
Childhood exposure to stress, 18, 227. *See also* Youth stress
Cholinergic influences, 80–81
Chronic stressors, 96*t*, 97. *See also* Stressors
Clinical expertise
different stress management methods and, 650–653, 652*t*
need for, 8–9
overview, 649–650
philosophical and sociocultural aspects of stress management and, 10–11
Clinically standardized meditation (CSM), 20, 378–379, 393–401, 394*t*, 395*t*, 404–405. *See also* Mantra meditation; Meditation
Closed system, 23, 24. *See also* Feedback
Cochrane system, 596–598, 597*t*. *See also* Music in relation to stress management
Cognitive activation theory of stress (CATS), 591–592
Cognitive and Affective Mindfulness Scale–Revised (CAMS-R), 414*t*, 432
Cognitive functioning. *See also* Thought processes
exercise and the walk–talk approach and, 621–622
heart rate variability biofeedback (HRVB) and, 280
mantra meditation and, 387
mindfulness meditation and, 424*t*, 425–426
overview, 163–164
Cognitive interventions. *See also* Cognitive therapy model
cognitive reappraisal strategies and, 109–112, 117
neurofeedback (NFB) and, 220
overview, 19
Cognitive reappraisal strategies, 109–112, 117. *See also* Emotion-focused stress management strategies
Cognitive restructuring, 512–513, 607
Cognitive therapy model. *See also* Cognitive interventions; Cognitive-behavioral therapy (CBT)
automatic thoughts and, 498–501, 499*t*, 501*t*–502*t*
defining stress and stressor within, 489–492
emotional schemas and, 508–510, 509*f*
expectations of coping and, 492–494
helpful strategies for coping with stress and, 510*f*, 512–513
integrating mindfulness into cognitive therapy, 513–518
limitations and contraindications for, 519–520
overview, 489, 520–521
personal schemas and, 505–508, 506*f*, 507*f*
problematic strategies for coping with stress and, 510–512, 510*f*
research regarding, 518–519
techniques to help a client put things in perspective, 495–497
Cognitive-behavioral music therapy, 598. *See also* Music in relation to stress management
Cognitive-behavioral therapy (CBT). *See also* Cognitive therapy model
biofeedback-based stress management model and, 140
EMDR therapy and, 194
hypnosis and, 559, 560–561, 579, 582*n*

interventions from, 142
mindfulness meditation and, 421
neurofeedback (NFB) and, 220
overview, 21, 520–521
research regarding, 518–519
sleep and, 116
Cognitive-behavioral therapy for insomnia (CBT-I), 116
Communication skills, 117–118. *See also* Interpersonal effectiveness
"Compare yourself to a zero point" technique, 496–497
Compassion, 425*t*, 430–431
Compliance, 540–543
Conditional beliefs, 504–510, 505*t*, 506*f*, 507*f*, 509*f*. *See also* Automatic thoughts
Confucianism, 453
Consciousness, 214–215, 334–335
Context theory of meaning, 164–165
Continuum technique, 495
Control sensations, 152, 174, 176*t*
Control system, 23–25, 264–265
Controllable stressors, 96*t*, 97. *See also* Stressors
COPD, 459*t*
Coping strategies
expectations of, 492–494
helpful strategies for coping with stress and, 510*f*, 512–513
mindfulness meditation and, 428
neurofeedback (NFB) and, 219
pain and, 566
problematic strategies for coping with stress and, 510–512, 510*f*
techniques to help a client put things in perspective, 495–497
youth stress and, 98–99, 99*t*
Core beliefs. *See* Automatic thoughts; Schemas
Corticotropin, 67–68
Corticotropin-releasing hormone (CRH/CRF), 67–68, 221–223, 620
Cortisol
exercise and the walk–talk approach and, 116, 611*t*–612*t*, 620
overview, 66–68
youth stress and, 116
Counting breathing procedure, 335–336. *See also* Breathing exercises
Coupling to movement method, 336–337. *See also* Breathing exercises
COVID-19 pandemic, 17–18, 655
Creative music therapy, 598. *See also* Music in relation to stress management
Cybernetics, 23–24
Cycle of stress, 505–510, 506*f*, 507*f*, 509*f*
Cytokine immunotherapy, 59
Cytokines. *See also* Inflammation
central nervous system and, 60–61
effect of on the HPA axis, 78–81
immune system and, 73, 74–78
neurotransmitters and, 63–66
overview, 83–84
relevance of, 61–71
as systemic stressors, 69–71
Daoism, 453
Data collection, 37
Daydreaming, 44–45
Decentering, 420
Decoupling, 420
Deep Mystery, 47
Deepening level of RMM, 46–47
Default mode network (DMN), 226–227
Depression
autogenic training and, 534, 547*t*
baroreflex (BR) activity and, 272
cognitive therapy and, 490, 492–496, 499*t*, 506*f*, 519–521
cytokine immunotherapy and, 59, 78
exercise and the walk–talk approach and, 116, 607, 613*t*, 620, 622–623, 624*t*, 625*t*, 627–632, 639, 640*n*
heart rate variability biofeedback and, 258, 265, 267, 270, 272, 278, 280
hypnosis and, 573, 575–577, 579, 582*n*
inflammation and, 59–61
mantra meditation and, 384*t*, 387, 391
microglial cells and, 82-83
mindfulness meditation and, 414*t*, 419–421, 422*t*, 424t, 428-432, 437
music in relation to stress management and, 592, 595–601-603, 596, 597*t*
neurofeedback and, 216, 226, 234, 241–242, 244, 250–251, 258
overview, 17*t*, 21, 52, 63, 68, 116, 131, 163, 563, 565, 566
posttraumatic stress disorder (PTSD) and, 226
progressive relaxation therapy and, 169*t*
Qigong therapy and, 458, 459*t*, 460, 465
respiratory training and, 310*t*, 315, 345, 347*t*
respiratory sinus arrhythmia and, 267
yoga and, 475–476
Desensitization, 380–381
Detachment, 51–52
Diabetes control, 169*t*, 459*t*
Diagnostic and Statistical Manual of Mental Disorders (DSM), 20
Dialectical behavior therapy (DBT)
IBS and abdominal pain and, 142
mindfulness meditation and, 413, 426
overview, 414*t*, 419
Diaphragmatic breathing, 243, 338–339. *See also* Breathing exercises; Breathing retraining
Dichotomous thinking. *See also* Automatic thoughts
examining and modifying, 503
example of, 500
overview, 499*t*
techniques for reversing, 501*t*
youth stress and, 111
Differential relaxation, 160–161
Digestion problems, 169*t*. *See also* Gastrointestinal problems
Direction, 47
Discounting positives. *See also* Automatic thoughts
examining and modifying, 503
example of, 500
overview, 498, 499*t*
techniques for reversing, 501*t*

Diseases, 10–11, 265–266, 424*t*. *See also* Cancer care; Health concerns; Medical conditions
Disengagement, 51–52, 99*t*. *See also* Coping strategies
Dissociation, 435–436
Distraction techniques, 112–113. *See also* Emotion-focused stress management strategies
Distress, 51–52
Dopamine (DA), 63–64, 619. *See also* Neurotransmitters
"Double Present Moment Check" strategy, 54
Drug use/abuse. *See* Addiction; Substance use/misuse
Dysfunctional breathing, 28. *See also* Hyperventilation; Respiratory dysregulation
Dyspnea, 169*t*, 349
Dysregulation. *See also* Self-regulation/dysregulation
  allostasis and allostatic overload and, 26
  cybernetics of, 23–24
  immune dysregulation, 26–27
  nature of stress and, 16–17
  overview, 21–23, 29
  respiratory dysregulation and, 27–28
  social systems and, 27

Early exposure to stress, 18, 227. *See also* Youth stress
Eating disorders
  EMDR therapy and, 196*t*
  exercise and the walk–talk approach and, 625*t*, 627–628
  hypnosis and, 582*n*
  mindfulness meditation and, 424*t*, 429
EEG biofeedback. *See* Neurofeedback (NFB)
Efficacy research. *See* Research literature
Electroencephalogram (EEG). *See also* Alpha brain-wave frequencies; Beta brain-wave frequencies; Theta brain-wave frequencies
  assessment and, 231–238, 239*f*
  case example of, 251–252, 252*f*
  effects of stress and, 219–220
  hypnosis and, 556–557
  interventions and, 241–250, 249*f*
  neurofeedback (NFB) and, 214–217, 216*f*, 229–230, 230*f*
  normalization and, 248
  posttraumatic stress disorder (PTSD) and, 225
  single-channel EEG assessment and, 217–218, 218*f*, 219*f*
Emotional control, 28, 166–167, 424*t*
Emotional domain in the multidomain model, 421–432, 422*f*
Emotional reasoning, 111, 499*t*, 500, 502*t*. *See also* Automatic thoughts
Emotion-focused stress management strategies, 103–115, 119. *See also* Breathing exercises; Cognitive reappraisal strategies; Distraction techniques; Mindfulness; Progressive muscle relaxation (PMR); Relaxation
Emotions, 244, 424*t*, 427–428, 512
Empty mind, 463
Endocrine system, 58, 79. *See also* Biological factors
End-tidal carbon dioxide ($ETCO_2$), 135
Engagement, 433–434
Environmental factors and considerations
  autogenic training (AT) method and, 529, 535–536
  biofeedback-based stress management model and, 137
  mantra meditation and, 396–397
  neurofeedback (NFB) and, 244–245
  walk–talk approach and, 634
Epidemiological approach, 94
Epigenetic factors, 18, 654
Epinephrine (adrenaline), 224
Evolutionary perspectives, 4–5, 228
Executive functioning, 424*t*
Exercise. *See also* Lifestyle-focused stress management strategies; Walk–talk approach
  clinical applications of, 622–633, 624*t*–625*t*
  dose–response effectiveness, 630
  effects of, 608–622, 610*t*–617*t*, 618*f*
  limitations and contraindications for, 631–633
  mental health and, 630–633
  overview, 607
  presentation of exercise therapy, 634
  progressive relaxation therapy and, 162
  research regarding, 610*t*–617*t*
  youth stress and, 115–116
Expectations, 492–494, 535
Expertise, clinical. *See* Clinical expertise
Exposure-based techniques, 106, 312, 579–580
External Qigong therapy (EQT), 455. *See also* Qigong therapy
External stressors, 6, 95–97, 96*t*. *See also* Stressors
Eye movement desensitization and reprocessing (EMDR) therapy
  case examples of, 197–206
  evidence that supports, 207
  historical context of, 194–195
  mechanisms of action, 206–207
  overview, 193–194, 197–206, 208
  side effects and contraindications, 207–208
  stress management and, 197
  theoretical underpinnings of, 195–197, 196*t*

Factor analyses, 42, 50–53
Fear Survey Schedule, 389
Feedback, 23–28, 340
Fibromyalgia, 131, 133, 140, 141, 143–144, 221, 347*t*, 384*t*, 424*t*, 427, 459*t*, 565
Fight-or-flight response, 3–4, 40, 267–268
Five-Facet Mindfulness Questionnaire (FFMQ), 414*t*, 432–433
Fixed mind-set, 492–493
Flexibility, 244–245, 341–342
Focus of attention, 45, 339–340, 460
Focused attention (FA) meditation, 39. *See also* Meditation; Relaxation/meditation/mindfulness (RMM) construct
Focused awareness, 415–416, 417–418
Focusing meditative skill, 390
Fortune telling. *See also* Automatic thoughts
  examining and modifying, 503
  example of, 500

overview, 498, 499*t*
techniques for reversing, 501*t*
youth stress and, 111
Freiburg Mindfulness Inventory (FMI), 414*t*, 432
Full-cap (19-channel) EEG, 235. *See also* Electroencephalogram (EEG)
Functional magnetic resonance images (fMRI), 216–217

Gamma-aminobutyric acid (GABA), 63–64, 475, 619. *See also* Neurotransmitters
Gastrointestinal problems. *See also* Abdominal pain; Irritable bowel syndrome (IBS)
digestion problems, 169*t*
EMDR therapy and, 196*t*
heart rate variability biofeedback (HRVB) and, 280
hypnosis and, 559, 582*n*
mindfulness meditation and, 424*t*
walk–talk therapy and, 622, 626
Generalized anxiety disorder. *See also* Anxiety disorders
EMDR therapy and, 196*t*
hypnosis and, 558
mindfulness meditation and, 419, 421, 427
overview, 19–20, 136, 139
Genetic factors, 18, 654
Glia, 81–83, 82*f*
Global desensitization, 380–381
Global Physical Activity Questionnaire, 633
Glucocorticoids, 4, 66–68, 79–80. *See also* Cortisol
Glutamate, 63–64, 619. *See also* Neurotransmitters
Groundedness, 45
Growth, 425*t*
Growth mind-set, 492–493
Guided awareness, 417–418
Guided imagery. *See also* Imagery
music in relation to stress management and, 597–598
progressive muscle relaxation (PMR) and, 108, 109
youth stress and, 108, 109
Guided meditation, 418
Gut microbiome, 58. *See also* Biological factors

Hatha yoga. *See* Yoga
Headaches, 17*t*, 98, 103, 137, 154, 166, 168, 169*t*, 171, 180, 184. 196*t*, 199, 239, 241, 347*t*, 348*t*, 401–403, 456, 457, 465, 466*t*, 483, 542, 546, 547*t*, 565, 573, 629, 651
Health concerns. *See also* Diseases; Medical conditions; Mental health
heart rate variability and, 265–266, 267
hypnosis and, 558–569
inflammation and, 60
mantra meditation and, 386
mindfulness meditation and, 424*t*, 426–427
music therapy and, 599
philosophical and sociocultural aspects of stress management and, 10–11
Qigong therapy and, 453–454, 458, 459*t*
whole-body breathing and, 347*t*
yoga and, 477, 478*t*–479*t*
Heart failure, 265, 275, 278, 280*t*, 338, 458, 459*t*, 465
Heart rate variability biofeedback (HRVB). *See also* Biofeedback; Heart rate variability (HRV); Heart rate variability (HRV) training
assessment and, 238, 239*f* , 279, 280*t*
case example of, 286–287
history of, 278–279
IBS and abdominal pain and, 142
method, 279–281, 282–286
neurofeedback (NFB) and, 229, 230*f*, 259
overview, 264, 287
polyvagal theory and, 228
side effects and contraindications, 281
theoretical underpinnings of, 264–278, 274*f*, 275*f*
Heart rate variability (HRV). *See also* Cardiovascular system; Heart rate variability biofeedback (HRVB); Heart rate variability (HRV) training; Respiratory sinus arrhythmia (RSA)
autogenic training (AT) method and, 530–531, 538–539
biofeedback-based stress management model and, 137–140
mantra meditation and, 379
music and, 592–593
negative feedback loops and, 23–24
neurotransmitters and, 64
overview, 23, 25, 264–268
Qigong therapy and, 457
resonance and, 272–273
whole-body breathing and, 343–344
Helplessness, 591–592
Hippocampus
exercise and the walk–talk approach and, 518–520, 607–609, 612*t*–613*t*, 618*f*, 619–622
microglial cells and, 81
neurogenesis and, 619–620
overview, 66–71, 82*n*, 221, 226–227, 231, 245, 380, 382
Home practice. *See* Out-of-session practices
Homeostasis, 24–25, 62, 265
Hopelessness, 591–592
Hormones
exercise and the walk–talk approach and, 116, 611*t*–612*t*, 620
music in relation to stress management and, 592, 594–595
Hostility, 280, 388, 576,
Humanistic approaches to therapy, 419
Hyperactivity, 169*t*
Hypertension. *See also* Blood pressure
autogenic training and, 533–534, 544, 546
baroreflex (BR) activity and, 272
exercise and, 623
heart rate variability biofeedback (HRVB) and, 265, 267, 277, 280*t*, 281–282
hypnosis and, 560
overview, 6, 10, 17, 21, 26, 140
progressive relaxation therapy and, 165, 166, 169*t*
Qigong therapy and, 456–458, 459*t*, 465

Hyperventilation. *See also* Breathing; Hypocapnia; Respiratory dysregulation
capnometry-assisted respiratory training (CART) and, 312
heart rate variability biofeedback (HRVB) and, 284
overview, 27–28, 330
stress and, 103, 135–136
whole-body breathing and, 346, 347–348, 348*t*, 349
Hyperventilation theory, 304–305
Hypnosis. *See also* Autogenic training (AT) method
adjunct to CBT, *560–561*
case example of, *575–577*
efficacy of, *558–569*
history of, *553–554*
method, *569–575*
overview, 40, *529–530*, *553*, *577–580*, *580n–583n*
pain and, *559–569*
trance, *557–558*
theoretical underpinnings of, *555–558*
Hypnotic induction, *570–573*
Hypnotizability, 389, *554–559*, *561–569*, *578–579*, *580n*, *581n*, *582n*, 653. *See also* Hypnosis
Hypocapnia. *See also* Breathing exercises; Capnometry-assisted respiratory training (CART); Hyperventilation
asthma and, 305–307
overview, 303–304, 315, 330
panic disorder and, 304–305
whole-body breathing and, 347–348, 348*t*
Hypothalamic–pituitary–adrenal (HPA) axis
cytokines and, 60–61, 66–71, 75–76, 78–81
exercise and the walk–talk approach and, 608
music in relation to stress management and, 592
neurofeedback (NFB) and, 221–223, 222*f*, 223*f*, 225*f*
overview, 66–69
stress-related symptoms and, 132, 135–136
youth stress and, 95
Hypothalamus, 22, 66, 67–68, 70–71, 94, 95, 135, 160, 221, 224, 225*n*, 231, 620. *See also* Hypothalamic–pituitary–adrenal axis.
Hypoxemia, 330

I CAN DO mnemonic, 100–101
Identity, 388, 430–431
If–then statements, 498
Imagery, 108, 109, 436
Immune hypothesis of depression, 61. *See also* Depression; Immune system; Inflammation
Immune system. *See also* Biological factors; Dysregulation; Physical health
cytokines and, 73, 74–76
effects of stressors on, 76–78
heart rate variability and, 269
hypnosis and, 560–561
immune response, 73
inflammation and, 59, 60
mantra meditation and, 384*t*
mindfulness meditation and, 424*t*
overview, 26–27, 71–74, 83–84
progressive relaxation therapy and, 169*t*
Qigong therapy and, 460
walk–talk approach and, 607
Inability to disconfirm, 499*t*, 501, 502*t*. *See also* Automatic thoughts
Inflammation. *See also* Biological factors
cardiac vagal tone and, 267
capnometry-assisted respiratory training (CART) and, 386
cytokines and, 74–76, 80
effects of stressors on, 76–78
exercise and the walk–talk approach and, 617*t*, 619, 630
glia and, 81–83, 82*f*
glucocorticoids and, 67–71, 80
heart rate variability biofeedback and, 276–277
HPA axis and, 78–80
interleukins and, 78–80, 622
mindfulness meditation and, 424*t*
neurofeedback (NFB) and, 223–224
overview, 16, 17*t*, 18, 26, 59–62, 77, 83–84, 621
psychoneuroimmunology and, 58–59
relevance of cytokines and, 61–71
vagus and, 80–81
Inflammation hypothesis of depression, 61. *See also* Depression; Immune system; Inflammation
Insomnia
autogenic training and, 544, 546
cognitive therapy and, 520
heart rate variability biofeedback (HRVB) and, 280
hypnosis and, 560
meditation and, 386
music in relation to stress management and, 592, *596*, *597t*, 601
neurofeedback and, 242, 280*t*
overview, 106, 116
progressive relaxation therapy and, 154–156, 165, 168, 169*t*
Qigong therapy and, 456–458, 459*t*
walk–talk therapy and, 629
whole body breathing and, 333
youth stress and, 116
Insula, 225–227, 277–278
Interferons 59, 75
Interleukins (IL-1 to IL-10), 69, 75–81, 617*t*, 621. *See also* Inflammation
Internal stressors, 95–97, 96*t*. *See also* Stressors
Interoceptive exposure, 312. *See also* Exposure-based techniques
Interpersonal effectiveness. *See also* Communication skills; Lifestyle-focused stress management strategies
mindfulness meditation and, 430–431
respiratory sinus arrhythmia and, 267
youth stress and, 117–118
Interventions, 29, 141–144. *See also* Emotion-focused stress management strategies; Lifestyle-focused stress management strategies; Problem-focused stress management strategies; *individual interventions*
Irritability, 388, 428

Irritable bowel syndrome (IBS). *See also* Abdominal pain; Gastrointestinal problems
hypnosis and, *558*, *562–564*, *582n*
interventions and, 141–143, 166, 184–185, 386, 558
mindfulness meditation and, 424*t*
overview, 17*t*, 26, 131, 133

Jacobson, Edmund, 155–156
Jnana yoga, 474. *See also* Yoga
Judgment focus. *See also* Automatic thoughts
example of, 501
integrating mindfulness into cognitive therapy and, 515–517
overview, 499*t*
techniques for reversing, 502*t*
"Jumping to conclusions" thinking trap, 111

Karmayoga, 474. *See also* Yoga
Kynurenine aminotransferase (KAT) enzymes, 620–621
Kynurenine metabolism, 614*t*–615*t*, 620–621

Labeling. *See also* Automatic thoughts
examining and modifying, 503–504
example of, 500
overview, 499*t*
techniques for reversing, 501*t*
Learning
exercise and the walk–talk approach and, 608–609
neurofeedback (NFB) and, 245
neurotransmitters and, 64
progressive relaxation therapy and, 178–179
Letting be meditative skill, 390
Lifestyle-focused stress management strategies, 115–118, 119, 390. *See also* Exercise; Interpersonal effectiveness; Sleep
Linguistic cognition. *See* Cognitive functioning
LORETA *z*-score NFB (LNFB). *See also* Low-resolution electromagnetic tomography (LORETA)
case example of, 250–258, 252*f*, 253*f*, 254*f*, 256*f*, 258*f*
method, 241–250, 249*f*
neurofeedback (NFB) and, 229–230, 230*f*
overview, 224
posttraumatic stress disorder (PTSD) and, 225, 227–228
Low-resolution electromagnetic tomography (LORETA). *See also* Electroencephalogram (EEG); LORETA *z*-score NFB (LNFB)
assessment and, 233, 236
neurofeedback (NFB) and, 222, 224, 229–230, 230*f*
Lymphocytes, 72, 73. *See also* Immune system
Lymphoid cell lineage, 72–73. *See also* Immune system

Maintenance of change, 178–179, 398–401
Mantra meditation. *See also* Meditation
assessment and, 384–390, 384*t*
case example of, 401–403
contributions of to clinical practice, 403–404
history of, 377–380
method, 393–401, 394*t*, 395*t*
overview, 377–378, 404–405
side effects and contraindications, 391–393
theoretical underpinnings of, 380–383
Manual Assessment of Respiratory Motion (MARM), 346
Medical conditions. *See also* Health concerns; Pain; Physical health
EMDR therapy and, 208
heart rate variability and, 265–266, 267
hypnosis and, 558–569
mantra meditation and, 386
mindfulness meditation and, 424*t*, 426–427
music in relation to stress management and, 592, 599–600
progressive relaxation therapy and, 169*t*
Qigong therapy and, 453–454, 458, 459*t*
whole-body breathing and, 347*t*
yoga and, 475–476
"Medically unexplained physical symptoms" (MUPS). *See also* Symptoms of stress-related disorders
biofeedback-based stress management model and, 136–141, 138*f*
interventions and, 141–144
overview, 131–132, 145
Medications, 234–235, 393, 654
Meditation. *See also* Mantra meditation; Mindfulness; Mindfulness meditation; Qigong therapy; Relaxation/meditation/mindfulness (RMM) construct
IBS and abdominal pain and, 142
overview, 39–40
side effects and contraindications, 391–393
youth stress and, 113–115
Meditative skills, 390
Memory
autogenic training (AT) method and, 532
EMDR therapy and, 197–206
exercise and the walk–talk approach and, 608–609
hypnosis and, 579, 581*n*
mindfulness meditation and, 435–436
neurofeedback (NFB) and, 223–224
neurotransmitters and, 64
sleep and, 116
whole-body breathing and, 350
"Mental filtering" thinking trap, 111
Mental health. *See also* Health concerns
cytokines and, 60–61
exercise and the walk–talk approach and, 630–633
hypnosis and, 558–559
nature of stress and, 17, 18*t*
respiratory sinus arrhythmia and, 267
whole-body breathing and, 347*t*
Metacognition, 248, 426
Microglial, 81–83, 82*f*
Mild cognitive impairment (MCI), 64
Mind reading. *See also* Automatic thoughts
examining and modifying, 504
example of, 500
overview, 111, 498, 499*t*
techniques for reversing, 501*t*

Mind wandering, 44, 48–49
Mind–body benefits, 426–427, 474–475
Mindful Attention Awareness Scale (MAAS), 414*t*, 432
Mindful awareness, 513
Mindful Basic Relaxation level of RMM, 43–45
Mindful Deepening level of RMM, 46–47
Mindful nonjudgment, 515–517
Mindful Quiet Focus level of RMM, 45–46
Mindful self-compassion training (MSC), 414*t*
Mindful Transformation/Transcendence level of RMM, 47–48
Mindfulness. *See also* Emotion-focused stress management strategies; Meditation; Mindfulness meditation; Relaxation/meditation/mindfulness (RMM) construct
  children and, 113–115, 118
  cognitive therapy model and, 510, 512–521
  helpful strategies for coping with stress and, 512–513
  hypnosis and, 339, 582
  IBS and abdominal pain and, 142
  mantra meditation and, 377–379
  neurofeedback and, 220
  overview, 39–55, 547, 606
  psychophysiology and, 142
  Qi-gong therapy and, 462, 466, 650
  research regarding, 50–51
  youth stress and, 113–115
Mindfulness meditation. *See also* Breathing exercises; Meditation; Mindfulness; Mindfulness-based stress reduction (MBSR)
  assessment and, 432–433
  case example of, 437–438
  challenges to, 434–435
  clinical applications of, 421–432, 422*f*, 424*t*–425*t*, 433–436
  compared to Qigong therapy, 455–456
  history of, 413–415, 414*t*
  instructions for, 417*t*
  meditational object, 377, 379, 392
  method, 415–420, 417*t*
  multidomain model of, 421–432, 422*f*, 424*t*–425*t*
  overview, 412–459, 651, 652*t*, 653
  resource material for, 436–437
  theoretical underpinnings of, 420–421
Mindfulness-based cancer recovery (MBCR), 414*t*
Mindfulness-based cognitive therapy (MBCT), 414*t*, 419–420
Mindfulness-based eating awareness treatment (MB-EAT), 414*t*, 419–420, 437–438
Mindfulness-based flourishing program (MBFP), 414*t*
Mindfulness-based interventions (MBIs), 414*t*
Mindfulness-based relapse prevention (MBRP), 414*t*
Mindfulness-based stress reduction (MBSR). *See also* Mindfulness meditation
  guided meditations and, 417–418
  history of, 413–415, 414*t*
  method, 418–420
  nonjudgment and, 516
  overview, 413, 414*t*
Mindfulness-oriented recovery enhancement (MORE), 414*t*
Minnesota Multiphasic Personality Inventory (MMPI), 534
Modifying negative thoughts, 503
Monoamine neurotransmitters, 63–64. *See also* Neurotransmitters
Mood, 59, 384*t*, 387, 424*t*
Motivation, 540–543
Multicomponent treatment, 545
Muscle relaxation techniques. *See* Progressive muscle relaxation (PMR)
Muscle tension assessment, 237–238, 239*f*
Muscular events. *See also* Skeletal muscles
  autogenic training (AT) method and, 536–537
  cognition and, 163
  exercise and the walk–talk approach and, 616*t*
  speech musculature, 175, 176*t*
  whole-body breathing and, 347*t*
Music in relation to stress management. *See also* Rhythm
  amplitude, 594
  arousal and relaxation and, 592–593
  case example of, 601–602
  chords, 594
  chronic stress and, 595
  clinical applications of, 600–601
  meditation and, 382, 389, 433
  music therapy training and, 598–600
  negative effects of music and, 602–603
  neurofeedback and, 246
  overview, 39, 53, 97, 105, 591–605
  pitch, 594
  psychophysiology of, 595
  research regarding, 596–598, 597*t*
  rhythm, 594
  types of music therapy, 597–598
Myeloid cells, 72, 73. *See also* Immune system
Mystical experiences, 48

Narrow focus, 245–246
Nature of stress, 16–18, 17*t*, 18*t*
Negative emotions, 49
Negative feedback loop, 23–24, 25, 28. *See also* Feedback
Negative filtering, 499*t*, 500, 501*t*. *See also* Automatic thoughts
Negative mood and affect, 384*t*
Negative thoughts, 498–501, 499*t*, 501*t*–502*t*. *See also* Automatic thoughts
Nerve stimulators, 272
Neurofeedback (NFB). *See also* Biofeedback-based stress management model
  assessment and, 231–238, 239*f*
  case example of, 250–258, 252*f*, 253*f*, 254*f*, 256*f*, 258*f*
  history of, 214–217, 216*f*
  method, 241–250, 249*f*
  overview, 214–217, 216*f*, 258–259
  posttraumatic stress disorder (PTSD) and, 224–228
  side effects and contraindications, 239–241
  single-channel EEG assessment and, 217–218, 218*f*, 219*f*
  theoretical underpinnings of, 219–231, 222*f*, 223*f*, 225*f*, 228*f*, 230*f*

Neurogenesis, 619–620
NeuroGuide program, 224, 229–230, 242, 249*f*
Neuroinflammation, 60, 81–83, 82*f*. *See also* Inflammation
Neuromuscular circuits, 158–159, 164
Neurophysiological perspectives
  neurofeedback (NFB) and, 221–224, 222*f*, 223*f*, 225*f*
  progressive relaxation therapy and, 159–160
  relaxation/meditation/mindfulness (RMM) construct and, 40, 49–50
Neuroregulation, 424*t*
Neurotransmitters
  exercise and the walk–talk approach and, 616*t*–617*t*, 618–619
  stressors and, 63–66
Nijmegen Scale, 136, 345
Nonjudgment, 515–517
Norepinephrine (NE), 63–64, 224, 619. *See also* Neurotransmitters
Nucleus tractus solitarius (NTS), 70–71, 229, 269, 277

Obsessive–compulsive disorder (OCD), 196*t*, 558, 582*n*
Open awareness (open monitoring), 39, 415–416. *See also* Mindfulness; Relaxation/meditation/mindfulness (RMM) construct
Open system, 23, 24. *See also* Feedback
Operant conditioning techniques, 214–215, 221
Oscillation, 25, 265, 266, 268, 273
Out-of-session practices, 105–106, 540–543
Overgeneralizing. *See also* Automatic thoughts
  examining and modifying, 503
  example of, 500
  overview, 499*t*
  techniques for reversing, 501*t*
Overmeditation, 392. *See also* Meditation
Oxytocin, 5, 67, 593–595, 600

Paced breathing, 28, 53, 227, 271, 277, 279, 283, 308, 310, 314, 332, 340, 429. *See also* Breathing
Pain 17*t*, 21, 44, 108, 131, 139, 144, 154, 166, 169*t*, 180, 196*t*, 206, 220, 222, 267, 278, 280*t*, 303, 315, 345, 346, 347*t*, 348, 364–365, 383, 384*t*, 413, 414*t*, 416, 417*t*, 422*t*, 424*t*, 426–427, 428, 434. 454, 455, 456, 458, 460, 465, 529, 532, 543, 544, 592, 597*t*, 599, 601, 631–632, 634*t*, 635, 637, 651, 653
  abdominal pain, 135–136, 141–143
  back pain, 41
  EMDR therapy and, 196*t*
  heart rate variability biofeedback (HRVB) and, 280
  hypnosis and, 555–583, 581*n*, 582*n*
  mantra meditation and, 384*t*
  mindfulness meditation and, 420–421, 424*t*
  music and, 592
  procedural pain, 113, 567–568
  progressive relaxation therapy and, 169*t*
  Qigong therapy and, 459*t*
  yoga and, 477
Panic attacks, 103, 427, 434
Panic disorder. *See also* Anxiety disorders
  breathing retraining and, 103, 305, 347*t*
  capnometry-assisted respiratory training (CART) and, 308–312, 310*t*–311*t*, 315–319
  EMDR therapy and, 196*t*
  hypocapnia and, 304–305
  mindfulness meditation and, 114. 387
  neurofeedback and, 257
  overview, 19–20, 251, 534
  respiration and, 304
  walk–talk therapy and, 626
Parasympathetic nervous system (PNS). *See also* Heart rate variability (HRV); Respiratory sinus arrhythmia (RSA); Vagus nerve
  autogenic training and, 530, 532
  biofeedback-based stress management model and, 137–140
  brainstem and, 231
  breathing and, 247, 416
  exercise and the walk–talk approach and, 608
  eye movement desensitization and, 207
  heart rate variability and, 264, 266
  heart rate variability biofeedback and, 224
  music and, 594, 595
  music in relation to stress management and, 594
  negative feedback loops and, 25
  neurofeedback (NFB) and, 231
  neurotransmitters and, 64
  overview, 21–22, 25, 64, 80, 267
  regulation and dysregulation and, 23
  stress-related symptoms and, 132, 134–135
  youth stress and, 94–95
Parasympathetic rebound, 21–22, 138, 166, 268
Parkinson's disease, 81
Passive attention, 339–340, 529
Pathogen recognition receptors (PRRS), 73
Pathogen-associated membrane patterns (PAMPS), 73
Perception, 17, 430, 495–497
Perfectionism, emotional, 512
Performance, 21, 39, 133, 219, 338, 434, 476–477, 478, 492–493, 506, 515
  cognitive performance, 98, 116, 216, 222, 224, 612–613, 650
  performance enhancement, 216, 217, 231, 236, 239, 242–243, 251, 255–256, 258–259, 278, 280, 282, 287, 386–387, 400, 547, 595
Persistence, 494
Personality disorders, 505–506
Personalizing. *See also* Automatic thoughts
  examining and modifying, 503
  example of, 500
  overview, 498, 499*t*
  techniques for reversing, 502*t*
Pfaffenberg Physical Activity Questionnaire, 633
Pgc-1α enzyme, 615*t*, 620–621
Pharmacotherapy, 20–21, 654. *See also* Medications
Phobias and phobic disorders, 19–21, 137, 166, 180–184, 196*t*, 226, 267, 306, 308, 310*t*–311*t*, 314, 347*t*, 430, 547, 560, 577, 579, 582*n*, 626. *See also* Anxiety disorders
Physical exercise. *See* Exercise

Physical health. *See also* Aerobic exercise; Diseases; Health concerns; Medical conditions
  dysregulation and. See Dysregulation
  hypnosis and, 558–569
  immune dysregulation and, 26–27
  inflammation and, 60
  mindfulness meditation and, 424*t*
  nature of stress and, 17, 18*t*
Physiological domain in the multidomain model, 421–432, 422*f*
Physiological mediators
  exercise and the walk–talk approach, 608–622, 610*t*–617*t*, 618*f*
  mantra meditation and, 379–380
  mindfulness meditation and, 424*t*
  progressive relaxation therapy and, 157–163
  stress-related symptoms and, 132–136
Physiological regulation, 65. *See also* Regulation
Pie chart technique, 496
Polyvagal theory, 134–135, 228, 228*f*
Positive emotion, 48–49
Positive feedback loops, 24. *See also* Feedback
Postconcussion syndrome, 248, 250
Posttraumatic stress, 98
Posttraumatic stress disorder (PTSD) and symptoms
  children and adolescents and, 98
  EMDR therapy and, 207, 208
  heart rate variability biofeedback (HRVB) and, 225, 227, 228, 280
  LORETA *z*-score NFB (LNFB), 250
  mantra meditation and, 384*t*, 403
  mindfulness meditation and, 427
  neurofeedback (NFB) and, 224–228
  neurotransmitters and, 64
  polyvagal theory and, 228, 228*f*
  Qigong therapy and, 459*t*
  respiratory sinus arrhythmia and, 267
  treatments and, 20
  yoga and, 476
Prayerfulness, 47
Prefrontal cortex, 40, 63, 66–70, 79, 94, 222, 225–226, 278, 420, 425, 432, 609
Prerecorded relaxation instructions. *See* Recorded relaxation instructions
Problematic strategies for coping with stress, 510–512
Problem-focused stress management strategies, 100–102, 118–119
Problem-solving, 246, 512–513
Procedural pain, 567–568. *See also* Pain
Processive stressors, 62, 69–71
Process-oriented approach, 328
Procrastination, 511
Productivity, 386–387
Progress monitoring, 540–543
Progressive muscle relaxation (PMR). *See also* Emotion-focused stress management strategies; Relaxation
  compared to autogenic training and, 529–530, 534
  feedback loops and, 24
  overview, 40
  relaxation/meditation/mindfulness (RMM) construct and, 50, 52–53
  youth stress and, 106–109
Progressive relaxation therapy. *See also* Progressive muscle relaxation (PMR); Relaxation; Tension
  assessment and, 165–170, 169*t*
  case example of, 179–185
  clinical applications of, 168, 169*t*
  cognition and, 163–164
  history of, 155–156
  introducing to the client, 170–171
  method, 170–179, 172*t*, 173*t*, 176*t*, 177*t*
  overview, 151–155, 185
  physical environment and equipment for, 171–172, 172*t*
  physiology and, 157–163
  tensions and, 164–165
  theoretical underpinnings of, 156–165
Proinflammatory mechanisms, 60. *See also* Inflammation
Psychoeducation, 140–141, 518
Psychological factors, 18, 94, 137, 347*t*. *See also* Mental health
Psychoneuroimmunology, 58–59. *See also* Biological factors; Brain functioning and structures; Immune system; Inflammation; Stress
Psychopharmacology, 20–21, 654. *See also* Medications
Psychophysiological model of mind, 163–164
Psychophysiological stress profile (PSP), 137–140, 138*f*
Psychophysiological stress treatments. *See also* *individual interventions*
  biofeedback-based stress management model and, 136–141, 138*f*
  music in relation to stress management and, 595
  overview, 20–21, 131–132
Psychosis, 579
Psychotic-type thinking, 426, 461–462

Qigong therapy
  case examples of, 464–466, 466*t*
  effects on stress and stress-related problems, 456–460, 459*t*
  history of, 450–454, 452*f*
  method, 454–456, 462–464
  overview, 450, 466–467
  side effects and contraindications, 461–462

Raja yoga, 474–475. *See also* Yoga
Reactivity, 22, 610*t*–611*t*
Readiness demands, 511–512
Reappraisals, 512–513
Receptivity, 339–340, 390
Recorded relaxation instructions, 153–154, 162, 528
Regenerative system, 592
Regret orientation, 499*t*, 500, 502*t*. *See also* Automatic thoughts
Regulation. *See also* Self-regulation/dysregulation
  allostasis and allostatic overload and, 26
  cybernetics of, 23–24
  exercise and the walk–talk approach and, 608–609
  nature of stress and, 16–17
  neurotransmitters and, 65
  overview, 21–23, 29

Regulatory neurotransmitters, 63–64. *See also* Neurotransmitters
Relation to self and others domain in the multidomain model, 421–432, 422*f*
Relaxation. *See also* Breathing exercises; Emotion-focused stress management strategies; Mindfulness; Progressive muscle relaxation (PMR); Progressive relaxation therapy; Relaxation/meditation/mindfulness (RMM) construct
  autogenic training (AT) method and, 533–534, 536–537
  capnometry-assisted respiratory training (CART) and, 312
  feedback loops and, 24
  heart rate variability and, 25
  helpful strategies for coping with stress and, 512–513
  hypnosis and, 572, 578–579
  mantra meditation and, 378
  music in relation to stress management and, 592–593
  neurofeedback (NFB) and, 243
  overview, 39–40
  progressive relaxation therapy and, 157–158
  Qigong therapy and, 457, 460, 463–464
  techniques for, 142
  treatments and, 19
  whole-body breathing and, 349
Relaxation-induced anxiety, 145, 170, 532–534
Relaxation overdose, 350
"Relaxation patter," 153
Relaxation response, 238–239, 239*f*
Relaxation therapy, 20
Relaxation/meditation/mindfulness (RMM) construct. *See also* Meditation; Mindfulness; Relaxation; RMM theory
  egalitarian training techniques for, 43
  factor analyses and, 42
  "5 + 1" model" and, 43–50
  implications for training and assessment, 53–55
  lexicon of, 42–43
  neurophysiological perspectives, 40
  overview, 39–40, 55
  research regarding, 50–53
Religion, 435, 451–452
Research literature, 8–9, 50–53, 653–654. *See also* Assessment
Resilience
  autonomic balance and, 267–268
  heart rate variability and, 265–266, 279
  overview, 29
  persistence and, 494
  walk–talk approach and, 607
  youth stress and, 98–99
Resistance
  adherence and, 391–393
  autogenic training (AT) method and, 542
  cognitive therapy model and, 519–520
  mantra meditation and, 398
  progressive relaxation therapy and, 178–179
Resonance, 140, 272–273, 275–276, 275*f*, 283–286
Resonance-frequency cardiovascular stimulation, 275–276, 275*f*, 283–286
Respiratory drive, 27, 271, 331
Respiratory dysregulation. *See also* Breathing; Hyperventilation; Respiratory system
  overview, 27–28
  progressive relaxation therapy and, 169*t*
  sighing and, 28
  whole-body breathing and, 347–348, 348*t*
Respiratory one method (ROM), 335, 378–379. *See also* Mantra meditation; Meditation
Respiratory retraining. *See also* Breathing retraining; Respiratory system; Whole-body breathing
  case example of, 364–368, 365*f*, 366*f*
  instructions for, 352*t*–353*t*, 356–364
  method, 351–364, 352*t*–353*t*, 354*f*, 355*f*
  overview, 327
  side effects and contraindications, 347–351, 348*t*
  strategies for breathing regulation and, 335–340
Respiratory sinus arrhythmia (RSA). *See also* Heart rate variability (HRV)
  heart rate variability biofeedback (HRVB) and, 271, 275–276, 282–286
  overview, 266–268, 282–283
  stress-related symptoms and, 134–135
  vagal tone and, 269
  youth stress and, 94–95
Respiratory system. *See also* Breathing; Respiratory dysregulation; Respiratory retraining
  biofeedback-based stress management model and, 139
  mantra meditation and, 384*t*
  overview, 329–331, 329*f*
  stress-related symptoms and, 135
  whole-body breathing and, 343–344
Retraining of breathing. *See* Breathing exercises; Breathing retraining; Respiratory retraining
Reverse coupling, 336–337
Rhythm, 382, 594. *See also* Music in relation to stress management
RMM theory, 40–43, 53–55. *See also* Relaxation/meditation/mindfulness (RMM) construct
Root mean square of successive differences (RMSSD), 266
Ruminative thinking patterns, 425–426

Sadness, 498
Salience network (SN), 226–227
Schemas
  cognitive therapy model and, 505–510, 506*f*, 507*f*, 509*f*
  emotional, 509
  integrating mindfulness into cognitive therapy and, 513–518
Schizophrenia
  exercise and the walk–talk approach and, 624*t*, 628–629
  immune or inflammatory processes and, 61
  microglial cells and, 81–82
  mindfulness meditation and, 426
Schultz, Johannes Heinrich, 527–528, 548–549
Seizures, 169*t*, 196*t*, 215–216

Selective serotonin reuptake inhibitors (SSRIs), 63–64, 234–235
Self, 430–431
Self-acceptance, 419, 425*t*, 430–431. *See also* Acceptance
Self-awareness
mindfulness meditation and, 419, 425–426
neurofeedback (NFB) and, 214–215
posttraumatic stress disorder (PTSD) and, 226–227
Self-blame, 387
Self-compassion, 425*t*, 430–431
Self-discipline, 390
Self-esteem, 573
Self-hypnosis, 573–575. *See also* Hypnosis
Self-operations control, 158–159
Self-perception, 334–335
Self-referential thinking, 48–49
Self-regulation/dysregulation. *See also* Dysregulation; Regulation
autogenic training (AT) method and, 529
cytokines and, 80
nature of stress and, 16–17
neurofeedback (NFB) and, 227–228
neurotransmitters and, 65
progressive relaxation therapy and, 158–159
whole-body breathing and, 350
Sensorimotor rhythm (SMR). *See also* Neurofeedback (NFB)
neurofeedback (NFB) and, 242, 244
overview, 215
sleep and, 220
Serotonin (5-HT), 63–64, 619, 620. *See also* Neurotransmitters
"Should" statements, 498. *See also* Automatic thoughts
Shoulds, 499*t*, 500, 501*t*. *See also* Automatic thoughts
Sickle cell disease, 562
Single-channel NFB, 230–231. *See also* Neurofeedback (NFB)
Sixteen Personality Factor Questionnaire (16-PF), 51, 389
Skeletal muscles. *See also* Muscular events
autogenic training (AT) method and, 536–537
exercise and the walk–talk approach and, 616*t*
progressive relaxation therapy and, 158–160, 166, 173–174, 173*t*
whole-body breathing and, 340–341
Sleep. *See also* Lifestyle-focused stress management strategies
heart rate variability biofeedback (HRVB) and, 280
mantra meditation and, 384*t*
music in relation to stress management and, 596, 597*t*
neurofeedback (NFB) and, 220
pain and, 566
progressive relaxation therapy and, 167–168, 169*t*
Qigong therapy and, 459*t*
walk–talk approach and, 633
whole-body breathing and, 347*t*
youth stress and, 116–117
Smith Relaxation States Inventory 3 (SRSI3), 50
Social factors. *See also* Interpersonal effectiveness
as a buffering factor, 29
overview, 27
regulation and dysregulation and, 24
status and, 17
technology and, 12
tend-and-befriend stress response and, 5–6
Slow breathing, 28, 103–105, 108, 115, 135–136, 221, 227, 238, 269, 275, 279. 281, 285, 305, 313–314, 318–322, 335–343, 347, 351, 355*t*, 360, 365, 367, 416, 426, 453, 462–463, 594. *See also* Breathing; Hyperventilation;
Respiratory dysregulation; Respiratory system
Social stressors, 16–17
Social support, 6
Sociocultural aspects, 10–13
Socioeconomic status, 17, 29
Socratic methods, 111–112
Somatoform symptoms, 196*t*, 347*t*. *See also* "Medically unexplained physical symptoms" (MUPS)
Sources of stress, 17–18, 18*f*
Speech musculature, 175, 176*t*
Spiritual domain in the multidomain model, 421–432, 422*f*
Spirituality, 48, 425*t*, 431–432
Stanford Hypnotic Clinical Scale for Children (SHCS:C), 568, 580*n*
Stanford Scales (SSHS), 555–556
Sterile inflammation, 59, 75. *See also* Inflammation
Stress. *See also* Stress management in general; Stress response; Stressors
cognitive therapy model and, 489–492
contemporary conceptualizations of, 6–7
EMDR therapy and, 197
heart rate variability biofeedback (HRVB) and, 280
history of the concept of, 3–4
overview, 591–592, 606
progressive relaxation therapy and, 169*t*
techniques to help a client put things in perspective, 495–497
Stress management in general. *See also individual methods*
biological barriers to, 654–655
idiographic nature of, 9–10
need for clinical expertise and, 8–9
overview, 7–8, 29, 655–656
philosophical and sociocultural aspects of, 10–13
selecting methods for, 650–653, 652*t*
Stress response. *See also* Stress; Stress management in general; Stressors
assessment and, 238–239, 239*f*
cytokines and, 61–71
EMDR therapy and, 197
mantra meditation and, 384*t*, 386
mindfulness meditation and, 424*t*
nature of stress and, 16–18, 17*t*, 18*t*
neurofeedback (NFB) and, 221–224, 222*f*, 223*f*
overview, 606, 655–656
types of, 4–6
youth stress and, 94–95

Stress-appraisal model, 508
Stressors. *See also* Stress; Stress management in general; Stress response
cognitive therapy model and, 489–492
effects of on immune functioning and inflammation, 76–78, 83–84
EMDR therapy and, 197
interpretations of, 491–492
overview, 62
progressive relaxation therapy and, 157
youth stress and, 95–97, 96*t*
Substance use/misuse. *See also* Addiction
exercise and the walk–talk approach and, 625*t*, 627
heart rate variability biofeedback (HRVB) and, 280
mantra meditation and, 384*t*, 387
mindfulness meditation and, 424*t*, 429–430
posttraumatic stress disorder (PTSD) and, 226
Qigong therapy and, 459*t*
Suffocation false-alarm theory, 304–305
Surface electromyographic biofeedback (sEMG), 153, 154, 156, 171–172
Sympathetic adrenal medullary system, 132
Sympathetic nervous system (SNS). *See also* Autonomic nervous system (ANS)
biofeedback-based stress management model and, 137–140
exercise and the walk–talk approach and, 608
heart rate variability biofeedback (HRVB) and, 276
music in relation to stress management and, 594
negative feedback loops and, 25
neurofeedback (NFB) and, 231
overview, 5, 21, 23–25, 28, 40, 65, 80–81, 94, 132–134, 138, 141, 142, 144, 151, 157, 159, 160, 221–222, 224, 225*n*, 228, 231, 255, 267–271, 276, 350–351, 379, 416, 451, 530–531, 608–609, 610*t*, 619–620
regulation and dysregulation and, 23
stress-related symptoms and, 132–133, 135–136
youth stress and, 94–95
Sympathetic–adrenomedullary system (SAM), 608
Symptom Checklist–90—Revised (SCL-90-R), 51, 389, 398
Symptoms of stress-related disorders, 131–136. *See also* "Medically unexplained physical symptoms" (MUPS)
Systematic desensitization, 380–381
Systems and systems theory
cytokines and, 62, 69–71
neurofeedback (NFB) and, 220–221
overview, 23
whole-body breathing and, 331–334, 332*f*

Tape-recorded relaxation instructions. *See* Recorded relaxation instructions
Technology, 12, 119, 162
Tend-and-befriend, 5
Tellegen Absorption Scale, 389
Tension. *See also* Progressive relaxation therapy
assessment and, 165–170, 169*t*, 237–238, 239*f*
control of in progressive relaxation therapy, 167–168
mantra meditation and, 385–386
meaning and purpose of, 164–165
method of diminishing tensions, 161
progressive relaxation therapy and, 157–158, 167–168, 175
respiratory sinus arrhythmia and, 275–276
skeletal muscles and, 173–174, 173*t*
whole-body breathing and, 347*t*
Therapist–client relationship, 172, 397
Theta brain-wave frequencies, 214, 217–219, 233–235, 239, 245–248, 380
Thinking traps. *See* Automatic thoughts
Third-Generation Mindfulness Theory. *See* RMM theory
Thoracic breathing, 28, 133–135, 339, 348, 351, 367, 426, 479*t*, 531, 609, 618
Thought processes. *See also* Automatic thoughts; Cognitive functioning; Negative thoughts
automatic thoughts and, 498–501, 499*t*, 501*t*–502*t*
cognitive reappraisal strategies and, 109
expectations of coping and, 492–494
integrating mindfulness into cognitive therapy and, 513–518
interpretations of stressors and, 491–492
mindfulness meditation and, 425–426
thought disorders, 424*t*
TOPS questionnaire, 246
Toronto Mindfulness Scale (TMS), 414*t*, 433
Traditional Chinese medicine (TCM), 450, 453, 454. *See also* Qigong therapy
Training
capnometry-assisted respiratory training (CART) and, 307–308, 308*f*
cognitive therapy model and, 519–520
heart rate variability biofeedback (HRVB) and, 282–286
mindfulness meditation and, 436
music therapy and, 598–600
Qigong therapy and, 464
relaxation/meditation/mindfulness (RMM) construct and, 43, 53–54, 55
whole-body breathing and, 328
yoga and, 480
Trait anxiety, 530–531
Trance experiences, 435–436, 554, 557–558. *See also* Hypnosis
Transcendence, 47–49
Transcendental meditation (TM). *See also* Mantra meditation; Meditation
adherence and, 399
contributions of to clinical practice, 403–404
overview, 378–379
yoga and, 475
Transpersonal approaches to therapy, 419
Trauma, 194–195, 207–208, 224–228, 435–436. *See also* Posttraumatic stress disorder (PTSD) and symptoms
Trauma-focused cognitive-behavioral therapies (TF-CBT), 206
Traumatic brain injury (TBI), 226, 248, 250
Trophotropoic activity, 22
Tryptophan metabolism, 620

Uncontrollable stressors, 96*t*, 97. *See also* Stressors
Unfair comparisons, 499*t*, 500, 502*t*, 504. *See also* Automatic thoughts
Unpleasant awareness, 348–349. *See also* Awareness

Vagal activity, 276
Vagal social engagement system, 134–135
Vagus nerve. *See also* Parasympathetic nervous system (PNS)
  biofeedback-based stress management model and, 139
  complexity of, 268–270
  music in relation to stress management and, 594
  overview, 266–268
  stress-related symptoms and, 134–135
Vascular configuration, 139. *See also* Heart rate variability (HRV)
Vascular dilation, 537–538
Ventilation. *See* Capnometry-assisted respiratory training (CART); Hyperventilation; Hypocapnia; Respiratory system
Vipassana meditation, 419
Visceral organs, 539
Visual Analogue Scale (VAS), 346
Visual cognition, 164. *See also* Cognitive functioning
Vulnerability, 18, 29

Walking meditation, 418. *See also* Meditation; Walk–talk approach
"Walking with blinders" thinking trap, 111
Walk–talk approach. *See also* Exercise
  case example of, 639–640
  clinical applications of, 622–633, 624*t*–625*t*
  history of, 607–608
  limitations and contraindications for, 631–633
  mental health and, 630–633
  method, 633–639, 634*t*
  overview, 606–607
  physiological mediators, 608–622, 610*t*–617*t*, 618*f*
  research regarding, 610*t*–617*t*
Wandering mind, 434–435
Well-being, 384*t*, 431–432, 599
"What I can still do" technique, 496
"What if?" thoughts. *See also* Automatic thoughts
  example of, 500
  overview, 499*t*
  techniques for reversing, 502*t*
Whole-body breathing. *See also* Breathing exercises; Respiratory retraining
  assessment and, 342–346, 343*f*, 347*t*
  case example of, 364–368, 365*f*, 366*f*
  history of, 327–328
  instructions for, 352*t*–353*t*, 356–364
  method, 352*t*–353*t*, 354*f*, 355*f*
  overview, 327
  as a process-oriented approach, 328
  side effects and contraindications, 347–351, 348*t*
  theoretical underpinnings of, 329–342, 329*f*, 332*f*
Width breathing, 341. *See also* Breathing exercises
Worry, 512

Yale Physical Activity Survey, 633
Yoga
  case example of, 480
  caveats and contraindications for, 477
  history of, 472–474
  method, 474–477, 478*t*–479*t*, 480, 481*t*
  mindfulness meditation and, 418
  relaxation/meditation/mindfulness (RMM) construct and, 50–51
Youth stress
  biological factors in, 94–95
  cognitive reappraisal strategies, 110
  controllable stressors, 97
  defining, 94
  disengagement coping, 99
  EMDR therapy and, 194
  emotion-focused strategies for, 103–115
  lifestyle-focused strategies for, 115–118
  mindfulness meditation and, 425*t*
  music in relation to stress management and, 600
  overview, 93, 118–119
  primary control coping, 99
  problem-focused strategies for, 100–102
  secondary control coping, 99
  signs and symptoms of, 97–98
  sources of, 95–97, 96*t*
  stress management techniques for, 98–100, 99*t*, 118
  uncontrollable stressors, 97

Zen traditions, 413, 415