Chapter 1
The Concept of Stress

To study medicine without reading is like sailing an uncharted sea.

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Stress, Behavior, and Health

Scientists investigating human health and disease are now reformulating the basic tenets upon which disease theory is based. For generations, the delivery of health care services was built upon the "one-germ, one-disease, one-treatment" formulations that arose from the work of Louis Pasteur. Although clearly one of the great advances in medicine, yielding massive gains against the infectious diseases that plagued humanity, the "germ theory" of disease also represents an intellectual quagmire that threatens to entrap us in a unidimensional quest to improve human health.

The germ theory of disease ignores the fact that by the year 1960, the primary causes of death in the USA were no longer microbial in nature. Rather, other pathogenic factors had emerged. Even four decades ago, it was noted, "New knowledge… has increased the recognition that the etiology of poor health is multifactorial. The virulence of infection interacts with the particular susceptibility of the host" (American Psychological Association, 1976, p. 264). Thus, in addition to mere exposure to a pathogen, one's overall risk of ill health seems also to be greatly influenced by other factors. Recent evidence points toward health-related behavior patterns and overall lifestyle as important health determinants.

The significance of health-related behavior in the overall determination of health status is cogently discussed by Jonas Salk (1973) in his treatise *The Survival of the Wisest.* Salk argues that we are leaving the era in which the greatest threat to human health was microbial disease, only to enter an era in which the greatest threat to human health resides in humanity itself. He emphasizes that we must actively confront health-eroding practices such as pollution, sedentary lifestyles, diets void of nutrients, and practices that disregard the fundamentals of personal and interpersonal hygiene at the same time that we endeavor to treat disease.

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Stress! While this word is relatively new in the English lexicon, few words have had such far-reaching implications. Evidence of the adverse effects of stress is well documented in innumerable sources. Homer's *Iliad* describes the symptoms of post-traumatic stress as suffered by Achilles. In *The New Testament*, Acts, Chap. 5, describes what may be the sudden death syndrome as it befell Ananias and his wife Saphira, after being confronted by Peter the Apostle, for withholding money intended for missionary service.

Excessive stress has emerged as a significant challenge to public health. More than 30 years ago, the Office of the US Surgeon General declared that when stress reaches excessive proportions, psychological changes can be so dramatic as to have serious implications for both mental and physical health (US Public Health Service, 1979). More recently, the Global Burden of Disease Study (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006) revealed that mental illnesses represent a significant contributor to the burden of global disease in high-income and low- and middle-income countries. The disability-adjusted life year (DALY) represents the number of years of life lost to premature death and disability; the disease burdens are listed by selected illnesses for high-income countries:

Ischemic heart disease	12.39 DALY
Cerebrovascular disease	9.35 DALY
Unipolar depressive disorders	8.41 DALY
Alzheimer's disease and other dementias	7.47 DALY
Trachea, bronchus, lung cancers	5.40 DALY
Hearing loss, adult onset	5.39 DALY
Chronic obstructive pulmonary disease	5.28 DALY
Diabetes mellitus	4.19 DALY
Alcohol use disorders	4.17 DALY
Osteoarthritis	3.79 DALY

Ten leading causes of burden of disease (DALYs) by high income group, 2001

It should be noted that mental illnesses not only rank as the third most burdensome disease process but also consistent with the observations of Salk (1973) almost 40 years ago, infectious diseases represent significantly less of a global burden upon health compared to neuropsychiatric disorders and alcohol use. According to the US Surgeon General (U.S. Department of Health and Human Services, 1999), for persons ages 18–54 years, anxiety and stress-related diseases are the major contributors to the mental illness in the USA, with more than twice the prevalence (16.4%) of mood disorders (7.1%). Stress seems to have reached almost epidemic proportions. Table 1.1 underscores the role that stress may play as a public health challenge.

Finally, reviews by McEwen (2008), Marketon and Glaser (2008), Black and Garbutt (2002), Kubzansky and Adler (2010) and Brydon, Magid, & Steptoe (2006) point out the contribution that stress makes to a wide variety of physical diseases.

Contained within the Surgeon General's report, *Healthy People* (U.S. Public Health Service, 1979), was the most significant indication ever that stress and its potentially pathological effects are considered serious public health factors. The Surgeon General's report on mental health (U.S. Department of Health and Human

Table 1.1 Stress and trauma as public health challenge

- Recent evidence suggests that 82.8% of adults in the USA will be exposed to a traumatic event during their lifetime (Breslau, 2009)
- Suicide rates in the military seem to be increasing (Kang & Bullman, 2009)
- Twelve-month DSM-IV disorders are highly prevalent in the USA, with 14% experiencing moderate to severe cases (Kessler, Chiu, Demler, & Walters, 2005)
- Suicide was the tenth leading cause of death in the USA in 2007 and an estimated 11 attempted suicides occur per every suicide death
- An elevated rate of major depression was equal to the rate of PTSD in New York City residents several months after the attacks on the World Trade Center of September 11, 2001 (Galea et al., 2002)
- Rates of trauma occurrence related to violence, injury/shock trauma, trauma to others, and unexpected death peaked sharply at age 16–20 years (Breslau, 2009)
- The lifetime prevalence of criminal victimization was assessed among female health management organization patients and found to be about 57%
- In 2001, the terrorist attacks against the World Trade Center and the Pentagon focus terrorism against the USA
- Of 2050 American Airlines (AA) flight attendants, 18.2% reported symptoms consistent with probable posttraumatic stress disorder (PTSD) in the aftermath of the September 11 attacks (Lating, Sherman, Everly, Lowry, & Peragine, 2004)
- Clearly, trauma and stress are at epidemic proportions in the USA. It seems clear that such conditions represent a "clear and present danger" to the psychological health of American society
- Perhaps of greatest concern, from a public health perspective is the realization that veterans returning from military service in Iraq and Afghanistan are returning home with a high prevalence of PTSD and PTSD-like syndromes. A recent review of 29 published studies revealed varying estimates of PTSD. "Among previously deployed personnel not seeking treatment, most prevalence estimates range from 5 to 20%. Prevalence estimates are generally higher among those seeking treatment: As many as 50% of veterans seeking treatment screen positive for PTSD…Combat exposure is the only correlate consistently associated with PTSD" (Ramchand et al., 2010, p. 59)
- The Veterans Affairs (VA) estimate that about 26% of veterans seeking treatment at VA facilities meet criteria for PTSD (U.S. Department of Veteran Affairs, Veterans Health Administration, Office of Public Health and Environmental Hazards, 2010)

Services, 1999) extended those observations made 20 years earlier and even sought to quantify the burden that mental illnesses represent as a disease entity. If, indeed, the aforementioned appraisals are credible, then what has emerged is a powerful rationale for the study of the nature and treatment of the human stress response. To that end, this book is written.

Defining Stress

In this book written for clinicians, the focus is on the treatment of pathogenic stress. Yet it may be argued that effective treatment emerges from an understanding of the phenomenology of the pathognomonic entity itself. In this first chapter, the reader

will encounter some of the basic foundations and definitions upon which the treatment of pathognomonic stress is inevitably based.

It seems appropriate to begin a text on stress with a basic definition of the stress response itself. The term *stress* was first introduced into the health sciences in 1926 by Hans Selye. As a second-year medical student at the University of Prague, he noted that individuals suffering from a wide range of physical ailments all seemed to have a common constellation of symptoms, including loss of appetite, decreased muscular strength, elevated blood pressure, and a loss of ambition (Selye, 1974). Wondering why these symptoms seemed to appear commonly, regardless of the nature of the somatic disorder, led Selye to label this condition as "the syndrome of just being sick" (Selye, 1956).

In his early writings, Selye used the term *stress* to describe the "sum of all nonspecific changes (within an organism) caused by function or damage" or, more simply, "the rate of wear and tear in the body." In a more recent definition, the Selyean concept of stress is "the nonspecific response of the body to any demand" (Selye, 1974, p. 14).

Paul Rosch (1986) provides an interesting anecdote. Recognizing that the term stress was originally borrowed from the science of physics, he relates how Selye's usage of the term did not conform to original intent:

> In 1676, Hooke's Law described the effect of external stresses, or loads, that produced various degrees of "strain," or distortion, on different materials. Selye once complained to me that had his knowledge of English been more precise, he might have labeled his hypothesis the "strain concept," and he did encounter all sorts of problems when his research had to be translated. (Rosch, 1986, p. ix)

Indeed, confusion concerning whether stress was a "stimulus," as used in physics, or a "response," as used by Selye, has plagued the stress literature. As Rosch (1986) describes:

> The problem was that some used stress to refer to disturbing emotional or physical stimuli, others to describe the body's biochemical and physiologic response … and still others to depict the pathologic consequences of such interactions. This led one confused British critic to complain, 35 years ago, that stress in addition to being itself was also the cause of itself and the result of itself, (p. ix)

To summarize the discussion so far, the term *stress* used in the Selyean tradition refers to a response, whereas in its original usage, within the science of physics, it referred to a stimulus, and the term *strain* referred to the response.

Using the term *stress* to denote a response left Selye without a term to describe the stimulus that engenders a stress response. Selye chose the term *stressor* to denote any stimulus that gives rise to a stress response.

In summary, drawing upon historical precedent, and consistent with Selye's original notion, the term *stress* is used within this volume to refer to a physiological reaction, or response, regardless of the source of the reaction. The term *stressor* refers to the stimulus that serves to engender the stress response.

With this fundamental introduction to the concept of stress, let us extend the conceptualization a bit further.

Ten Key Concepts in the Study of Stress

1. The stimulus that evokes a stress response is referred to a *stressor.* There are two primary forms of stressors (Girdano, Dusek, & Everly, 2009): (a) psychosocial stressors (including personality-based stressors) and (b) biogenic stressors.
2. *Psychosocial stressors* become stressors by virtue of the cognitive interpretation of the event, that is, the manner in which they are interpreted, the meanings they are assigned (Ellis, 1973; Lazarus, 1966, 1991, 1999; Lazarus & Folkman, 1984; Meichenbaum, 1977). Selye once noted, "It's not what happens to you that matters, but how you take it." Epictetus is credited with saying, "Men are disturbed, not by things, but the views which they take of them." For example, a traffic jam is really a neutral event; it only becomes a stressor by virtue of how the individual interprets the event (i.e., as threatening or otherwise undesirable). If the individual views the traffic jam as neutral or positive, no stress response ensues. Some stressors are inherently more stressful than others and leave less potential variation for cognitive interpretation (e.g., objective external threats to one's safety or well-being, grief, guilt, etc.). But even in these cases, cognitive interpretation will play a role in the adjustment to the stressor and serve to augment or mitigate the resultant stress response.

   Phenomenological research conducted by Smith, Everly, and Johns (1992, 1993) evaluated the credibility of this notion of a mediating role for psychological variables in the relation between stressor stimuli and the signs and symptoms of distress. Using structural mathematical modeling, exploratory and confirmatory factor analyses, they demonstrated that psychosocial environmental stressors exert their pathogenic effect upon the human organism primarily through cognitive processes. More specifically, evidence of cognitive–affective discord predicted signs and symptoms of physical ill health as well as maladaptive coping behaviors. This notion of a mediating role for cognitive–affective processes in the stressor-to-illness paradigm is explored in Chap. 2.
3. *Biogenic stressors,* on the other hand, require no cognitive appraisal in order to assume stressor qualities; rather, biogenic stimuli possess an inherent stimulant quality. This stimulant characteristic, commonly referred to as a sympathomimetic characteristic, is found in substances such as tea, coffee, ginseng, guarana, ginkgo biloba, yohimbine, amphetamines, and cocaine. Extremes of heat and cold and even physical exercise exert sympathomimetic effects. Biogenic stressors directly cause physiological arousal without the necessity of cognitive appraisal (Ganong, 1997; Widmaier, Raff, & Strang, 2004).

   The inclusion of the biogenic sympathomimetic category of stressors in no way contradicts the work of Lazarus and others who have studied the critical role that interpretation plays in the formation of psychosocial stressors. Such an inclusion merely extends the stressor concept to recognize that stimuli that alter the normal anatomical or physiological integrity of the individual are also capable of activating many of the same psychoendocrinological mechanisms

that we refer to as the *stress response.* Thus, even if a patient convincingly reports that he or she really enjoys drinking 15 cups of caffeinated coffee per day, the clinician must be sensitive to the fact that those 15 "enjoyable" cups of coffee can serve as a powerful stressor activating an extraordinary systemic release of stress-response hormones such as epinephrine and norepinephrine, and in doing so can be a contributing factor in cardiac conduction abnormalities, for example. Similarly, individuals who belong to "Polar Bear" clubs and voluntarily immerse themselves in frigid waters during the winter undergo an extraordinary stress response characterized by massive sympathetic nervous system (SNS) arousal. Thus, even though the consumption of caffeine and the immersion of oneself into frigid bodies of water may truly be reinforcing, that person still experiences a form of physiological arousal that is accurately described as a stress response and may pose some risk to health, depending upon the intensity and chronicity of the exposure to the stressors. These issues are reiterated once again in Chap. 2.In general, it is important for the clinician to understand that by far the greater part of the excessive stress in the patient's life is self-initiated and self-propagated, owing to the fact that it is the patient who interprets many otherwise neutral stimuli as possessing stress-evoking characteristics. Kirtz and Moos (1974) suggest that social stimuli do not directly affect the individual. Rather, the individual reacts to the environment in accordance with his or her interpretations of the environmental stimuli. These interpretations are affected by such variables as personality components or status and social role behaviors. These cognitive–affective reactions are also subject to exacerbation through usually self-initiated exposures to sympathomimetic stimuli, such as excessive caffeine consumption and the like. Having the patient realize and accept reasonable responsibility for the cause and reduction of excessive stress can be a major crossroads in the therapeutic intervention. Therefore, we also discuss this issue in greater detail in Chap. 3.

4. Stress is a response, or reaction, to some stimulus. The stressor–stress response notion is illustrated in Fig. 1.1.
5. The stress response represents a physiological reaction, as defined in the Selyean tradition (Cannon, 1914; Selye, 1956) has extended this concept somewhat and conceptualizes the stress response as a "physiologic mechanism of mediation," that is, a medium to bring about a result or effect. More specifically, the stress response may be viewed as the physiological link between any given stressor and its target-organ effect. This then will be the working definition of stress used in this volume: *Stress is a physiological response that serves as a mechanism of mediation linking any given stressor to its target-organ effect or arousal.* This notion is captured in Fig. 1.2.

   When communicating with patients or simply conceptualizing the clinical importance of the stress response, however, (Selye's 1974, 1976) notion that stress is the "sum total of wear and tear" on the individual seems useful.
6. The stress response, as a physiological mechanism of mediation, can be characterized by a widely diverse constellation of physiological mechanisms (Cannon, 1914; Godbout & Glaser, 2006; Gruenewald & Kemeny, 2007;

Fig. 1.1 A basic stress response model

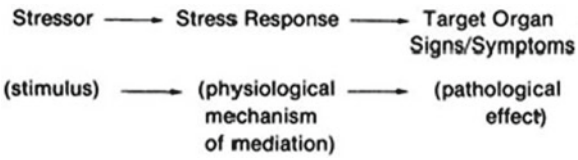

Fig. 1.2 The stress response as a mechanism of mediation

Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Mason, 1972; Selye, 1976; Widmaier, Raff, & Strang, 2004) that may be categorized as (1) neurological response pathways, (2) neuroendocrine response mechanisms, and (3) endocrine response pathways. These potential response mechanisms will be reviewed in detail in Chap. 2.

Although the mechanisms of the stress response are processes of arousal, and the target-organ effects are usually indicative of arousal, the stress response has been noted as entailing such forms of arousal as to cause actual slowing, inhibition, or complete stoppage of target-organ systems (Engel, 1971; Gellhorn 1968, 1969; Gray, 1985; Selye, 1976; Widmaier, Raff, & Strang, 2004). These inhibiting or depressive effects are typically a result of the fact that, upon occasion, stress arousal constitutes the activation of inhibitory neurons, inhibitory hormones, or simply an acute hyperstimulation that results in a nonfunctional state (e.g., cardiac fibrillation). This seeming paradox is often a point of confusion for the clinician; hence, its mention here.

7. Selye (1956, 1976) has argued for the "nonspecificity" of the stress response. Other authors (Harris, 1991; Mason, 1971; Mason et al., 1976; Monroe, 2008) have argued that the psychophysiology of stress may be highly specific with various stressors and various individuals showing different degrees of stimulus or response specificity, respectively. Current evidence strongly supports the existence of highly specific neuroendocrine and endocrine efferent mechanisms. Whether there exists another way of collectively categorizing stress-response mechanisms may be as much a semantic as a physiological issue (Brosschot, Gerin, & Thayer, 2006; Everly, 1985a; Selye, 1980).
8. A vast literature argues that when stress arousal becomes excessively chronic or intense in amplitude, target-organ (the organ affected by the stress response) disease and/or dysfunction will result (Godbout & Glaser, 2006; Gruenwald & Kemeny, 2007; Selye, 1956). When stress results in *organic* biochemical and/or structural changes in the target organ, these results are referred to as a *psychophysiological disease* (American Psychiatric Association, 1968) or a *psychosomatic disease* (Lipowski, 1984). Psychosomatic diseases were first cogently described by Felix Deutsch in 1927. However, it was Helen Dunbar (1935) who published the first major treatise on psychosomatic phenomena. In 1968, in the *Diagnostic and Statistical Manual of Mental Disorders,* second

edition (American Psychiatric Association, 1968), the term *psychophysiological disorder* was used to define a "group of disorders characterized by physical symptoms that are caused by emotional factors" (p. 46). Thus, we see the terms *psychosomatic* and *psychophysiological* used interchangeably to refer to organically based physical conditions resulting from excessive stress.

Sometimes these terms are confused with the development of neurotic-like physical symptoms without any basis in organic pathology. The terms *conversion hysteria* or *somatoform disorders* are usually used to designate such nonorganic physical symptomatology.

The *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition text revision, used the designation "Psychological Factors Affecting Medical Condition" to encompass stress-related physical disorders (American Psychiatric Association, 2000). By virtue of its multiaxial diagnostic schema, this nosological manual allowed clinicians to assess levels of stress and environmental support as they may affect not only physical symptoms but also psychiatric symptoms. Physical symptoms without a basis in or manifestation of organic pathology are subsumed under the somatoform category.

In the context of this volume, it is recognized that stress can be directed toward discrete anatomical or physiological target organs and therefore can lead to physical disorders characterized by organic pathology (i.e., psychophysiological or psychosomatic disorders); yet we must also recognize that the human mind can serve as a target organ. Thus, in addition to somatic stress-related disorders, it seems reasonable to include psychiatric-stress-related disorders as potential target-organ effects as well.

In summary, the terms *psychosomatic* and *psychophysiological* disorders are considered in this book as terms that refer to disorders characterized by physical alterations initiated or exacerbated by psychological processes. If tissue alterations are significant enough, and if the target organ is essential, then psychosomatic disorders could be life threatening. Neurotic-like somatoform disorders, on the other hand, involve only functional impairments of the sensory or motor systems and therefore cannot threaten life. Like the psychosomatic disorder, somatoform disorders are psychogenic; unlike psychosomatic processes, somatoform disorders entail no real tissue pathology. Confusion between the psychosomatic concept, on one hand, and the somatoform concept, on the other, is easily understandable. Yet, such confusion may lead to an underestimation of the potential severity of the disorder, thereby affecting treatment motivation and compliance.

9. Although recent reports emphasize the negative aspects of stress, there do exist positive aspects as well. Previous writers have viewed the stress response as an innate preservation mechanism, which in earlier periods of evolutionary development allowed us to endure the challenges to survival. Numerous researchers (Cannon, 1953; Chavat, Dell, & Folkow, 1964; Henry & Stephens, 1977; Widmaier, Raff, & Strang, 2004) have concluded, and we shall see in later chapters, that the nature of the psychophysiological stress response is that of apparent preparatory arousal–arousal in preparation for physical exertion.

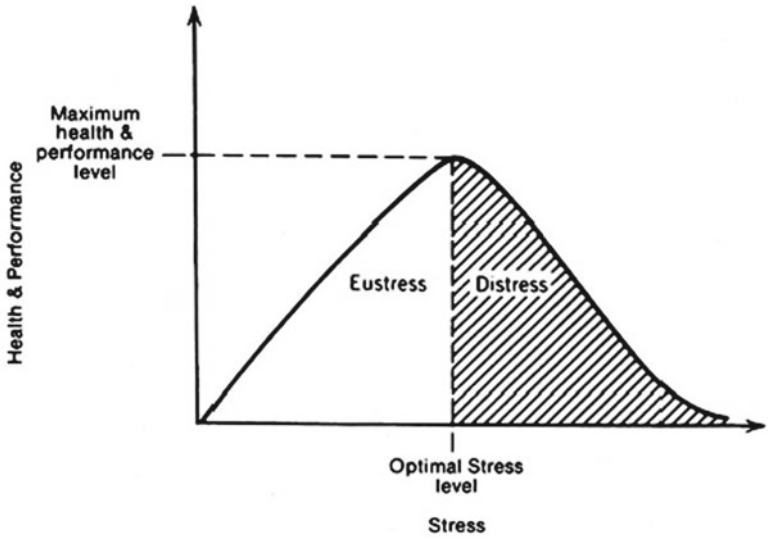

Fig. 1.3 The graphic relationship between stress arousal (*horizontal axis*) and performance (*vertical axis*). As stress increases, so does performance (eustress). At the optimal stress level, performance has reached its maximum level. If stress continues to increase into the "distress" region, performance quickly declines. Should stress levels remain excessive, health will begin to erode as well

When used in such a way, it is easy to see the adaptive utility of the stress response. Yet stress arousal in modern times under circumstances of strictly psychosocial stimulation might be viewed as inappropriate arousal of primitive survival mechanisms, in that the organism is aroused for physical activity but seldom is such activity truly warranted and, therefore, seldom does it follow (Benson, 1975; Widmaier, Raff, & Strang, 2004).

Selye (1956, 1974) further distinguishes constructive from destructive stress clearly pointing out that not all stress is deleterious. He argues that stress arousal can be a positive, motivating force that improves the quality of life. He calls such positive stress "eustress" (prefix *eu* from the Greek meaning "good") and debilitating, excessive stress "distress." Figure 1.3 depicts the relation between stress and health/performance. As stress increases, so does health/performance and general well being. However, as stress continues to increase, a point of maximal return is reached. This point may be called the *optimal stress level,* because it becomes deleterious to the organism should stress arousal increase. The point at which an individual's optimal stress level is reached, that is, the apex of one's tolerance for stress as a productive force, seems to be a function of genetic, biological, acquired physiological, and behavioral factors.

10. Last in this series of assumptions about what stress is and is not, is the point that confusion exists regarding the role of the nonmedical clinician in the treatment of the stress response. This is so primarily because the target-organ effects or

pathologies that result from excessive stress are mistakenly thought of as the psychophysiological stress response itself. It is important to remember the distinction that stress is a process of psychophysiological arousal (as detailed in Chap. 2), whereas the effects and pathologies (such as migraine headache, peptic ulcers, etc.) are the manifestations of chronically repeated and/or intense triggerings of the psychophysiological stress response (see Chap. 3). Treating the end-organ pathologies is clearly within the realm of the physician or nonmedical specialist in behavioral medicine. However, the traditional psychologist, counselor, physical therapist, social worker, or health educator can effectively intervene in the treatment of the stress arousal process itself. This includes treating the excessive stress/anxiety that accompanies, and often exacerbates, chronic infectious and degenerative diseases.

It is important to understand that this text addresses the clinical problem of excessive psychophysiological arousal—that is, the excessive stress-response process itself. It is not a detailed guide for psychotherapeutic intervention in the psychological trauma or conflict that may be at the root of the arousal (although such intervention can play a useful role). Nor does this text address the direct treatment of the target organ pathologies that might arise as a result of excessive stress. We shall limit ourselves to a discussion of the clinical treatment of the psychophysiological stress-response process itself.

Based on a review of the literature, we may conclude that treatment of the process of excessive psychophysiological stress arousal may take the form of three discrete interventions (see Girdano, Dusek, & Everly, 2009):

1. Helping the patient develop and implement strategies by which to avoid/minimize/modify exposure to stressors, thus reducing the patient's tendency to experience the stress response (Ellis, 1973; Lazarus, 1991, 2006; Meichenbaum, 2007).
2. Helping the patient develop and implement skills that reduce excessive psychophysiological functioning and reactivity (Girdano et al., 2009; Lazarus, 2006; Lehrer, Woolfolk, & Sime, 2007).
3. Helping the patient develop and implement techniques for the healthful expression, or utilization, of the stress response (see Girdano, Dusek, & Everly, 2009; Lehrer, Woolfolk, & Sime, 2007).

Finally, it has been suggested that the clinicians who are the most successful in treating the stress response have training not only in the psychology of human behavior but also medical physiology (Miller, 1978; Miller & Dworkin, 1977). Our own teaching and clinical observations support this conclusion. If indeed accurate, this conclusion may be due to the fact that stress represents the epitome of mind–body interaction. As Miller (1979) suggests, mere knowledge of therapeutic techniques is not enough. The clinician must understand the nature of the clinical problem as well. Therefore, the reader will find that the treatment section of this text is preceded by a basic discussion of the functional anatomy and physiology of the stress response.

Plan of the Book

The purpose of this text is to provide an up-to-date discourse on the phenomenology and treatment of pathogenic human stress arousal. As noted earlier, once target-organ signs and symptoms have been adequately stabilized, or ameliorated, the logical target for therapeutic intervention becomes the pathogenic process of stress arousal that caused the target-organ signs and symptoms in the first place. To treat the target-organ effects of stress arousal while ignoring their pathogenic, phenomenological origins is palliative at best, and often predicts a subsequent relapse.

The unique interaction of psychological and physiological phenomena that embodies the stress response requires a unique therapeutic understanding, as Miller has noted. Therefore, this volume is divided into three sections: Part I addresses the anatomical and physiological nature of stress arousal. Also discussed are measurement and other phenomenological considerations. Part II offers a practical clinical guide for the actual treatment of the human stress response and addresses a multitude of various technologies. Finally, Part III discusses special topics in the treatment of the human stress response. Also included in this volume are appendices that provide a series of brief discussions on considerations and innovations relevant to clinical practice.

References

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. Text revision). Washington, DC: Author

American Psychological Association. (1976). Contributions of psychology to health research. *The American Psychologist, 31*, 263–273.

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

Black, P. H., & Garbutt, L. D. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research, 52*(1), 1–23.

Breslau, N. (2009). The epidemiology of Trauma, PTSD, and other posttrauma disorders. *Trauma, Violence and Abuse: A Review Journal, 10*(3), 198–210.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The preservative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*(2), 113–124.

Brydon, L., Magid, K., & Steptoe, A. (2006). Platelets, coronary heart disease, and stress. *Brain, Behavior, and Immunity, 20*(2), 113–119.

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

Cannon, W. B. (1953). *Bodily changes in pain, hunger, fear, and rage*. Boston, MA: Branford.

Chavat, J., Dell, P., & Folkow, B. (1964). Mental factors and cardiovascular disorders. *Cardiologia, 44*, 124–141.

Dunbar, H. F. (1935). *Emotions and bodily changes*. New York, NY: Columbia University Press.

Ellis, A. (1973). *Humanistic psychology: The rational-emotive approach*. New York, NY: Julian.

Engel, G. L. (1971). Sudden and rapid death during psychological stress. *Annals of Internal Medicine, 74*, 771–782.

Everly, G. S., Jr. (1985). Occupational stress. In G. S. Everly & R. Feldman (Eds.), *Occupational health promotion* (pp. 49–73). New York, NY: Wiley.
Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., & Vlahov, D. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *New England Journal of Medicine, 346,* 982–987.
Ganong, W. F. (1997). *Review of medical physiology* (18th ed.). Stamford, CT: Appleton & Lange.
Gellhorn, E. (1968). Central nervous system tuning and its implications for neuropsychiatry. *Journal of Nervous and Mental Disease, 147*, 148–162.
Gellhorn, E. (1969). Further studies on the physiology and pathophysiology of the tuning of the central nervous system. *Psychosomatics, 10*, 94–104.
Girdano, D., Dusek, D., & Everly, G. S., Jr. (2009). *Controlling stress and tension*. San Francisco, CA: Pearson Benjamin Cummings.
Godbout, J. P., & Glaser, R. (2006). Stress-induced immune dysregulation: Implications for wound healing, infection disease and cancer. *Neuroimmune Pharmacology, 1*(4), 421–427.
Gray, J. (1985). Issues in the neuropsychology of anxiety. In A. Tuma & J. Maser (Eds.), *Anxiety and anxiety disorders* (pp. 5–26). Hillsdale, NJ: Erlbaum.
Gruenewald, T. L., & Kemeny, M. E. (2007). Aging and health: Psychoneuroimmunological processes. In C. M. Aldwin, C. L. Park, & A. Spiro III (Eds.), *Handbook of health psychology and aging* (pp. 97–118). New York, NY: Guilford Press.
Harris, T. (1991). Life stress and illness: The question of specificity. *Annals of Behavioral Medicine, 13*, 211–219.
Henry, J. P., & Stephens, P. (1977). *Stress, health, and the social environment*. New York, NY: Springer-Verlag.
Kang, H. K., & Bullman, T. A. (2009). Is there an spidemic of suicides among current and former military personnel? *Annals of Epidemiology, 19*(10), 757–760.
Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 617–627.
Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: new perspectives from psychoneuroimmunology. *Annual Review of Psychology, 53*, 83–107.
Kirtz, S., & Moos, R. H. (1974). Physiological effects of social environments. *Psychosomatic Medicine, 36*, 96–114.
Kubzansky, L. D., & Adler, G. K. (2010). Aldosterone: A forgotten mediator of the relationship between psychological stress and heart disease. *Neuroscience & Biobehavioral Reviews, 34*(1), 80–86.
Lating, J. M., Sherman, M. F., Everly, G. S., Lowry, J. L., & Peragine, T. F. (2004). PTSD reactions and functioning of American airlines flight attendants in the wake of September 11. *Journal of Nervous and Mental Disease, 192*(6), 435–441.
Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.
Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.
Lazarus, R. S. (2006). *Stress and Emotion: A new synthesis*. New York, NY: Springer.
Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
Lehrer, P. M., Woolfolk, R. L., & Sime, W. E. (2007). *Principles and Practice of Stress Management* (3rd ed.). New York, NY: The Guildford Press.
Lipowski, Z. J. (1984). What does the word "psychosomatic" really mean? *Psychosomatic Medicine, 46*, 153–171.
Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., & Murray, C. J. (2006). Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data. *The Lancet, 367*(9524), 1747–1757.

Marketon, J. W., & Glaser, R. (2008). Stress hormones and immune function. *Cellular Immunology, 252*(1–2), 16–26.

Mason, J. W. (1971). A re-evaluation of the concept of "non-specificity" in stress theory. *Journal of Psychiatric Research, 8*(3–4), 323–333.

Mason, J. W. (1972). Organization of psychoendocrine mechanisms: A review and reconsideration of research. In N. Greenfield & R. Sternbach (Eds.), *Handbook of psychophysiology* (pp. 3–76). New York: Holt, Rinehart & Winston.

Mason, J. W., Maher, J., Hartley, L., Mougey, E., Perlow, M., & Jones, L. (1976). Selectivity of corticosteroid and catecholamine responses to various natural stimuli. In G. Servan (Ed.), *Psychopathology of human adaptation* (pp. 147–171). New York: Plenum Press.

McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology, 583*(2–3), 174–185.

Meichenbaum, D. (1977). *Cognitive-behavior modification*. New York: Plenum Press.

Meichenbaum, D. (2007). Stress inoculation training. A preventative and treatment approach. In P. M. Lehrer, R. L. Woolfolk, & W. E. Sime (Eds.) *Principles and practice of stress mamangement* (3rd ed., pp. 497–516. New York: Guilford

Miller, N. E. (1978). Biofeedback and visceral learning. *Annual Review of Psychology, 29*, 373–404.

Miller, N. E. (1979). General discussion and a review of recent results with paralyzed patients. In R. Gatchel & K. Price (Eds.), *Clinical applications of biofeedback* (pp. 215–225). Oxford, UK: Pergamon Press.

Miller, N. E., & Dworkin, B. (1977). Critical issues in therapeutic applications of biofeedback. In G. Schwartz &J. Beatty (Eds.), *Biofeedback: Theory and research* (pp. 129–162). Chicago: Aldine.

Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annual Review of Clinical Psychology, 4*, 33–52.

Ramchand, R., Schell, T. L., Karney, B. R., Osilla, K. C., Burns, R. M., & Caldarone, L. B. (2010). Disparate prevalence estimates of PTSD among service members who served in Iraq and Afghanistan: possible explanations. *Journal of Trauma and Stress, 23*(1), 59–68.

Rosch, P. (1986). Foreword. In J. Humphrey (Ed.), *Human stress* (pp. ix-xi). New York: American Management Systems Press.

Salk, J. (1973). *The survival of the wisest*. New York: Harper & Row.

Selye, H. (1956). *The stress of life*. New York: McGraw-Hill.

Selye, H. (1974). *Stress without distress*. Philadelphia: Lippincott.

Selye, H. (1976). *Stress in health and disease*. Boston: Butterworth.

Selye, H. (1980). *Selye's Guide to Stress Research*. New York: Van Nostrand Reinhold Co.

Smith, K. J., Everly, G. S., & Johns, T. (1992, December). A structural modeling analysis of the mediating role of cognitive-affective arousal in the relationship between job stressors and illness among accountants. Paper presented to the Second APA/NIOSH Conference on Occupational Stress, Washington, DC.

Smith, K. J., Everly, G. S., & Johns, T. (1993). The role of stress arousal in the dynamics of the stressor-to-illness process among accountants. *Contemporary Accounting Research, 9*, 432–449.

U. S. Department of Health and Human Services. (1999). *Mental health: Report of the surgeon general*. Rockville, MD: Author.

U. S. Public Health Service. (1979). *Healthy people*. Washington, DC: U.S. Government Printing Office.

U.S. Department of Veterans Affairs, Veterans Health Administration, Office of Public Health and Environmental Hazards. (2010). Analysis of VA health care utilization among U.S. Global War of Terrorism (GWOT) veterans. Unpublished quarterly report (cumulative through 4th quarter FY 2009). Washington, DC: Author.

Widmaier, E. P., Raff, H., & Strang, K. T. (2004). *Vander, Sherman, Luciano's Human Physiology: The Mechanisms of Body Function*. New York, NY: McGraw-Hill.