Chapter 24
Summation and Conclusions

With all its sham, drudgery and broken dreams, it is still a beautiful world. Be cheerful. Strive to be happy.

Max Ehrmann

"First study the science. Then practice the art which is born of that science." These words of Leonardo da Vinci have served as the guiding spirit of this volume. Perhaps more than any other pathological process, stress arousal represents the epitome of mind–body interaction. We suggested earlier in this volume that proper clinical understanding and treatment of such conditions that so intimately intertwine psychology and physiology demand that the clinician's attention be directed toward the "science" of physiology (and pathophysiology) as well as the art/science of behavior change. Thus, to be consistent with this stated bias, this volume has first introduced the reader to a rather detailed exploration of the physiological nature and foundations of the human stress response. This, as a preface to the subsequent chapters that directly addressed the treatment of excessive stress arousal and its pathological consequences.

A Treatment Model

In order to assist the reader in seeing the phenomenology of pathogenic stress arousal in its larger context, this volume has introduced an epiphenomenological model of the human stress response, from stressor to target-organ effect. This model

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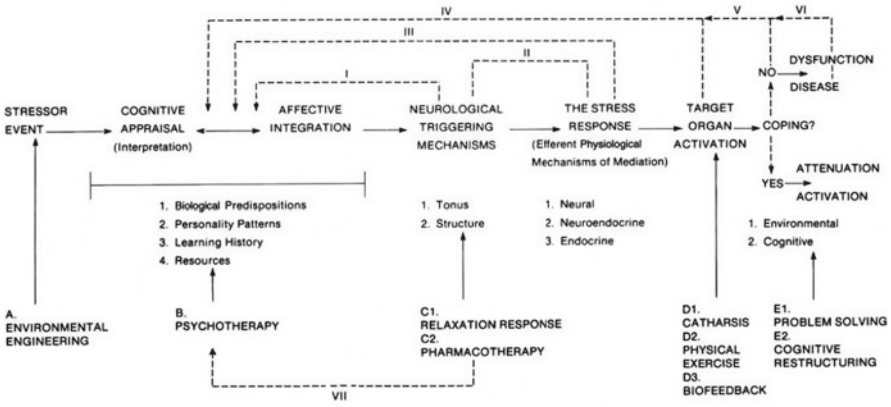

Fig. 24.1 A multidimensional treatment model for the human stress response

was first introduced in Chap. 2 as Fig. 2.6. It represents the larger "overview" of not just stress arousal but its antecedent and consequent constituents. This basic figure was employed again in Chap. 5 (Fig. 5.1) but this time with measurement technologies superimposed. The same basic model was again employed in the introduction to Part II, to demonstrate how treatment interventions might be conceptualized in a coherent and cogent manner via a unifying model. Finally, that same figure is replicated once again to assist in the summary of the text (see Fig. 24.1).

The stress response is predicated upon an event called a *stressor.* The stressor, which can be real or imagined, is typically then perceived and some *cognitive interpretation* is rendered by the individual. The obvious exception would be sympathomimetic and vasoactive stressors, which bypass interpretation. On the basis of the interpretation, the individual will experience some *affect* emerging from the limbic circuitry. Intimately intertwined with the creation of this affect is the activation of a *neurological triggering mechanism* that transduces psychological events into somatic realities, the most important of which is the initiation of the stress response itself: a psychophysiological mechanism of mediation characterized by arousal and possessing three basic efferent limbs: the neural, the neuroendocrine, and the endocrine. These stress arousal mechanisms then exert some *target-organ effect,* that is, signs and symptoms. If *coping* mechanisms employed by the person are not successful, continued arousal and a *psychosomatic disease* is the likely consequence (refer to Fig. 24.1).

Given an understanding of this oversimplified process, we can more appropriately select and implement treatment interventions. Listed in Table 24.1 are the major treatment interventions discussed within this volume and summarized in Fig. 24.1. Having used a treatment model to summarize this volume, let us turn to a clinical protocol to see how it all fits together.

Table 24.1 Treatment interventions

Treatment options	Chapter discussion	Purpose
Environmental engineering	Chapters 7, 8	To allow the patient to avoid or minimize exposure to stressors
Psychotherapy	Chapter 8	To avoid stressors; to reinterpret stressors; to increase perception of self-efficacy
Relaxation response	Chapters 9–15, Appendix B	To reduce pathogenic arousal; to increase perception of self-efficacy
Psychopharmacotherapy	Chapter 16	To reduce arousal
Reduction of target-organ arousal	Chapters 10–15	To ventilate the sress response in a health-promoting manner; to reduce target-organ arousal or dysfunction
Problem solving and cognitive restructuring	Chapters 8, 17, 20, 22	To attenuate excessive arousal

A Treatment Protocol

In Chap. 1, the reader may recall that on the basis of a review by Girdano, Dusek, and Everly (2009), we suggested that the treatment of excessive stress arousal may be categorized into three therapeutic genres, or "dimensions":

1. Strategies to avoid/minimize/modify stressors
2. Strategies to reduce excessive arousal and target-organ reactivity/dysfunction
3. Strategies to ventilate, or express, the stress response

The use of such a summative schema facilitates the creation of a generic, multidimensional treatment protocol. This protocol, compared with Fig. 24.1 and Table 24.1, more readily translates into a step-by-step guide for clinical practice and is summarized in Table 24.2.

Having the patient undergo a physical examination is desirable, especially if target-organ disease or dysfunction is manifest. The stress response is the epitome of mind-body interaction. For this reason, it is sometimes difficult to distinguish psychologically induced problems from those problems that possess little or no psychogenic etiology. In some cases, what appear to be stress-related signs and symptoms may in reality be indicative of some neurological pathology or neoplastic phenomenon.

As previously indicated, psychological assessment, especially personologic assessment, can play an important role in treatment planning. The concept of personologic diathesis, when operationalized, provides insight into not only diagnosis but also treatment (see Chap. 7). Broad-spectrum psychological assessments such as the Minnesota Multiphasic Personality Inventory (MMPI) or the Millon Clinical Multiaxial Inventory (MCMI) are useful and efficient assessment tools, especially the latter, which integrates personality assessment (refer also to Chap. 5).

Table 24.2 A general treatment protocol

Physical examination
Psychological assessment (especially personality assessment, see Chap. 5)
Intervention (three dimensions):
1. Helping the patient develop and implement strategies for the avoidance/minimization/modification of stressors
(a) Patient education (see Chaps. 1, 2 for rationale)
(b) Environmental engineering (see Chaps. 18, 19)
(c) Psychotherapy (Chap. 8)
2. Helping the patient develop and implement skills that reduce excessive stress arousal and target-organ reactivity/dysfunction
(a) Meditation (Chap. 10)
(b) Neuromuscular relaxation (Chap. 12; Appendix B)
(c) Respiratory control (Chap. 11)
(d) Hypnosis (Chap. 13)
(e) Biofeedback (Chap. 14)
(f) Psychopharmacotherapy (Chap. 16)
3. Helping the patient develop and implement techniques for the healthful ventilation/expression of the stress response
(a) Catharsis (Chap. 8)
(b) Physical exercise (Chap. 15)
(c) Religious belief (Chap. 17)

The general protocol described in Table 24.2 is not designed to be "blindly" adhered to by the practicing clinician; rather it is provided as a general guide to allow the clinician to formulate a multidimensional, individualized treatment protocol which is far superior to a unidimensional one. Furthermore, aided by the psychological assessment, the treatment plan can be tailored to the specific needs of the patient. Bhalla (1980) found that multidimensional, individualized treatment protocols were generally superior to unidimensional or otherwise "boilerplate" protocols (see also Millon, Crossman, Meagher, Millon, & Everly, 1999).

To reiterate the approach taken in this book, effective treatment emerges from accurate and specific diagnosis. To assess the notion of a personologic diathesis, to actively involve the patient in his or her own therapy, and to recognize the therapeutic value of patient education (see Chap. 8) seems a useful and humanistic approach to the treatment of the human stress response and its target-organ consequences.

A Word About Treatment Adherence

It would be naive to believe or expect that if all of the guidelines in this text are followed, treatment adherence would approach 100%. This is simply not the case. Patient adherence to lifestyle management programs designed to reduce health-related

risk factors (e.g., stress management programs) reportedly ranges from a high of 80% to a low of 20%. It has been suggested that adherence to daily home relaxation sessions is as low as 40%. Even adherence to antihypertensive drug regimens may be as low as 60%. See Meichenbaum and Turk (1987) and Blackwell (1997) for a review of these and other treatment adherence issues.

Nevertheless, we hope that our review of the phenomenology and measurement of stress arousal will render clinical success superior to those treatment conditions where such has not been the case. We also highly recommended that the reader review a manual on facilitating treatment adherence before designing any treatment protocols (e.g., Blackwell, 1997; Meichenbaum & Turk, 1987; Millon et al., 1999).

Summary

In conclusion, within this chapter, we have seen two forms of summary: the first, a treatment model, provided a more conceptual summary; the second, a treatment protocol, provided a more clinically practical summary. We hope that, by providing both, we convey not only a sense of clinical practicality but also a conceptual understanding that will allow the reader to move beyond the limits of this volume.

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