STRESS AND COPING

Edited By

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Lawrence Erlbaum Associates, Publishers
1985 Hillsdale, New Jersey London

Lawrence Erlbaum Associates, Inc., Publishers
365 Broadway
Hillsdale, New Jersey 07642

Library of Congress Cataloging in Publication Data

ISSN 0883-0908
ISBN 0-89859-564-9

Printed in the United States of America
10 9 8 7 6 5 4 3 2 1

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Preface

This is the first in a series of volumes based on the annual University of Miami Symposia on Stress and Coping. These symposia are concerned with current research pertaining to developmental, physical health and mental health aspects of stress and coping. The purpose of the University of Miami Symposia on Stress and Coping is to bring together scholars from different disciplines to share their research experiences related to this topic. We hope that in so doing we can bring to our symposia audiences and to our readers some sense of the rapid progress that is taking place in stress and coping research. One facet of this progress is reflected in the increasing use of psychophysiological procedures. Another important aspect is the increasingly sophisticated measurement of psychosocial and biobehavioral responses. Still another feature of this progress is reflected in the integration of biomedical and psychosocial approaches to achieve a common purpose. Thus, it is fitting that our symposia are held at both the Mailman Center of the University of Miami Medical School Campus and the psychology department of the arts and sciences campus.

This first volume was designed to provide a general discussion of the concept of stress, an overview of psychophysiologic processes involved in stress and coping, and evidence relating behavioral stresses to immune response, sleep disorders, depression and cardiovascular disease. This volume also deals with prenatal, neonatal, and childhood stress as well as with psychosocial aspects of stress and coping involving anger, type A behavior, depression, hardiness and self-consciousness.

The volume is divided into three sections: psychophysiological, developmental and psychosocial aspects of stress and coping. The first section on psychophysiological aspects includes chapters on biobehavioral responses to stressors,

arousal/sleep states and stress, neurochemical mechanisms of stress, and the effects of stress on hypertension and on the immune response. Befittingly, the first chapter, "Stress is a noun! No, a verb! No, an adjective!", debates the very semantics of the term stress. Dr. Engel suggests that the term stress is ambiguous since it means different things to different investigators and that there are a number of incorrect premises implicit in most concepts of stress. In his chapter he reviews several of the conceptual ways in which the term has been used and how this usage has led to confusion. In addition he presents a number of research findings from his own laboratory which bear on the concept of stress as it has been used by other investigators and finally suggests some alternative strategies for addressing the experimental and clinical issues on stress. Dr. Engel's modest proposal is that instead of using stress as a label we should be explicit in characterizing the stimulus-response relationships we are actually observing and we should specify the conditions under which we observe those relationships. Second, he suggests that we should stop invoking stress as a catch all term to disguise the fact that we do not understand why a phenomenon is occurring. And third, we should begin to attend more to coping.

In the subsequent chapter on biobehavioral responses to stressors by Drs. Schneiderman and McCabe, the authors concur with Engel's contention that operationally defined the term stress is important, but suggest that stress has been useful as an organizing term because it has permitted scientists to synthesize large numbers of otherwise isolated and disparate observations into relatively few principles. And, of course, it brings together groups of scientists, as in this symposium, who share common concerns. Schneiderman and McCabe go on to define stress as a change or threat of change demanding adaptation by an organism. The stimulus for this change is called the stressor and the adaptation is described in terms of behavioral and physiochemical responses. The authors then review the literature on stress and coping response systems. They include the sympatho-adrenomedullary system (SAM), the hypothalamico-pituitary-adrenocortical system (HPAC), brain catecholamines, endogenous opiates and the immune system. They then present a number of biobehavioral models of organic pathology for disease processes such as hypertension, arteriosclerosis and coronary heart disease, and arrhythmias. Throughout their chapter, the active coping process and its physiological equivalents and passive coping or conservation-withdrawal behaviors and their physiological correlates are contrasted. The authors conclude that "when an individual continually engages in defensive or conservation-withdrawal behaviors almost continuously over a period of many years, this habit may lead to chronic disease, particularly when such adjustments interact with environmental insult such as excess sodium ingestion or the steady consumption of an atherogenic diet". Although the exact manner by which environmental stressors and organism responses interact to produce specific pathologies in humans is presently unknown, the data reviewed in this chapter suggest that stress-related biobehavioral factors may play a role in the patho-

genesis of some cases of essential hypertension, coronary heart disease and other disorders.

The essence of the chapter on arousal, sleep and stress by Ray Winters is summarized in the words of Dement (1974) who said "It is virtually axiomatic that a disturbance of mind will manifest itself in the sleeping state as well as in the waking state." As Dr. Winters suggests, "sleep and arousal patterns are sensitive barometers of one's ability to cope with the vagaries of life. Therefore, understanding the relationships that these patterns bear to various psychological and behavioral control systems is essential to the advancement of our understanding of stress itself." Winters reviews data on sleep deprivation and sensory deprivation as stressors, and various applications to anxiety, depression, schizophrenia, narcolepsy and insomnia. The view espoused in this chapter is that all control systems in the body serve in one way or the other to maintain physical and emotional health, and to serve this purpose the set point in any one of these systems may be altered. In terms of a control system that regulates full brain arousal, stress has been defined as a significant variation from the set point. Arousal is viewed as a system variable for the regulation of full brain arousal but also one that regulates sleep. Winters hypothesizes that chronic forms of stress whether they are due to endogenous disfunction or the results of environmental stressors may result in changes in the set point for full brain arousal. Disease may be a consequence of such a change.

In a chapter entitled "Neurochemical Mechanisms Underlying Stress-Induced Depression" Drs. Weiss and Goodman present data on rats being exposed to strong uncontrollable shocks after which they show depressed behavior. Evidence is presented that such stress-induced depression in rats represents a good animal model for important aspects of clinical depression in humans. Recent work from their laboratory indicates that stress-induced depression is caused by substantial central nervous system depletion of norepinephrine, specifically in the locus coeruleus region of the brain stem resulting in a functional blockade of alpha 2 receptors in this brain region. As a result of changes in this region, the authors suggest that transmitters in other brain regions will also be affected and review recent data from other laboratories using different animal models that implicate serotonin and possibly acetylcholine as being involved in the process by which uncontrollable shock leads to behavioral depression. These results, the authors suggest, are compatible with clinical findings that point to disturbances of norepinephrine, serotonin and possibly acetylcholine in human depression. The neurochemical changes are also consistent with known actions of antidepressant treatments.

David Anderson, in a chapter on "Behavior Stress and Experimental Hypertension," reviews a number of his own studies on dogs as well as other studies in the literature that support the hypothesis that biobehavioral factors can contribute significantly to the development of hypertension by virtue of their effects on renal regulation of sodium. Research from Dr. Anderson's laboratory has shown

that either biobehavioral or dietary factors can increase blood pressure in genetically-susceptible animals. In addition, he reports that sustained hypertension can develop in genetically-normotensive animals in response to a combination of chronic exposure to mild stress and a high salt diet, even when neither alone produces progressive daily blood pressure changes. These experiments, he suggests, "showed that the hypertensive response is associated with a significant decrease in urinary sodium concentration, indicating inappropriate sodium retention." In addition, he reviews data suggesting that distress-salt hypertensive responses can be attenuated by increases in potassium intake.

A relatively recent newcomer to the field of stress research is the study of stress effects on the immune response, the subject of the chapter by Priscilla Campbell and John Cohen. As the authors suggest, "with the last decade anecdotal evidence of the effects of stress on immune responses has been supported by an increasing amount of experimental data." In their chapter they first present models of two kinds of immune responses and indicate what kinds of immunity they mediate. They then suggest several ways in which stress could affect immune responses, and finally they describe what they feel are some of the more compelling studies documenting effects of stress on the immune response.

The section on developmental aspects of stress and coping includes chapters on the use of cortisol as a measure of stress, prenatal stress and its effect on fetal activity, stress and coping in the neonate in response to aversive stimulation and coping with separation stress by infants and young children. In the first chapter in this section, entitled "The Use and Abuse of Cortisol as a Measure of Stress," Drs. Levine and Coe use data from their own laboratory on separations of primate mothers and their infants to discuss the uses and abuses of cortisol as a measure of stress. They suggest that, as is the case with most physiological measures, there are two features of the cortisol response to consider if one is going to utilize this measure with any degree of sophistication. "The first has to do with the quantitative aspect, which mainly involves alteration in the amount of hormones secreted under specific experimental or observational circumstances. There are also important qualitative features, such as the dynamics of the pituitary-adrenal system. There is a time course of the release of ACTH and subsequently of adrenocorticoids, which may be ignored by many investigators who arbitrarily choose a time point and take a single sample as a sole measure of adrenal function. The time course of the adrenal response has been shown not only to vary under different circumstances but to vary widely depending on time of day, reproductive state, and genetic variables." Thus, their chapter is predominantly methodological as they attempt to show how cortisol can be used within a specific investigative area to provide useful information which would not have been possible if the activity of the pituitary-adrenal system had not been measured.

In the subsequent chapter the effect of prenatal stress on fetal activity is discussed in the context of the existing literature and preliminary data from a

study on ultrasound feedback by Sandberg, Field, Quetel, Garcia, and Rosario. The conclusions drawn from data on the effects of providing ultrasound feedback to reduce pregnancy anxiety suggest that the ultrasound feedback does seem to attenuate at least third trimester pregnancy stress and fetal activity. However, the relationships are complex, and the differences between the experimental and control groups were more definitively based on evening monitoring of fetal activity by the mothers than morning monitoring by ultrasound. In addition, because of transactional and nonlinear relationships between pregnancy anxiety and fetal activity, it would appear that a pregnancy anxiety/fetal activity relationship assessed during the same trimester or averaged across pregnancy would yield inconsistent relationships, a finding that may help explain some of the inconsistencies in the literature. The authors suggest that a larger data set may yield transactional relationships between pregnancy anxiety and fetal activity, with the fetal activity effects of anxiety only emerging in those with early and persistent pregnancy anxiety as well as a curvilinear relationship with fetal activity extremes (high or low) being related to pregnancy anxiety.

In a chapter on stress and coping in the human neonate by Drs. Gunnar, Malone, and Frisch, the authors' data indicate that measures of adrenocortical activity can serve as useful tools when studying stress and coping during the first days of life. The authors conclude from their data that "not only is the pituitary of the newborn responsive to aversive stimulation, but the magnitude of the response appears to vary with the intensity of the stressor." They also conclude that measures of adrenocortical activity provide a means of analyzing the functional consequences of changes in behavioral state. By measuring cortisol levels they were able to obtain support for the hypothesis that quiet sleep serves as a coping mechanism in the newborn. Of additional interest is the suggestion from their data that the healthy human newborn is extremely resilient in the face of stresses such as the one used here, namely circumcision without anesthesia.

In the subsequent chapter Tiffany Field reviews studies on separations of young primates and reports data on brief separations of infants and young children during their mothers' hospitalization for the birth of another child as well as more permanent separations from peers when transferred to new schools. The stages of separation stress (agitation and depression) are described along with individual styles of coping with separation stress. Basically the data from Field's studies on human infants and children are consistent with early separation data on primates. The infants and young children experienced a similar biphasic response with agitation followed by depression, and similar functions appeared to be affected including play and sleep behavior, heart rate, activity level, eating, elimination, and illness. Field suggests that separation distress occurs primarily because the infant has lost its primary source of stimulation and arousal modulation. "An infant, already highly aroused due to the agitation of losing the mother, may become even more agitated during any attempt made by substitute caregivers to provide stimulation and arousal modulation." In this case, Field

argues, that the most adaptive coping response for the infant may be to withdraw and remain inactive in order to avoid or "ward off" the stimulation of others or the stimulation of its own activity. Depressed activity levels or conservation/withdrawal may then continue at least until physiological equilibrium is restored or until other caregivers have become familiar with the individual's unique stimulation and arousal modulation needs.

The final section of this volume on the psychosocial aspects of stress and coping include chapters on type A behavior, coping and anger, stress and depression, health, stress and coping, and self consciousness and coping. The first chapter in this section by Drs. Dembrowski and MacDougall is entitled "Beyond Global Type A: Relationships of paralinguistic attributes, hostility, and anger-in to coronary heart disease." The authors begin their chapter with a relevant anecdote that summarizes their chapter. This concerns the circumstances surrounding the death of the pioneer cardiovascular pathologist, John Hunter, who had claimed that interpersonal conflict precipitated his own angina attacks and, indeed, he died suddenly in 1793 after an intense verbal altercation with a colleague. Several other anecdotes are given from the 17th–19th century writings by physicians citing characteristics such as pronounced speech mannerisms and exaggerated job involvement characteristics leading to coronary heart disease, characteristics that have come to be associated with the term type A behavior. The authors then review the data from several collaborative studies on coronary prone behavior including the Western collaborative group study, the Multiple Risk Factor Intervention Trial study, the Duke study, and the Framingham study. The authors suggest that because a large number of negative findings have emerged, it is not clear that the association between type A behavior pattern and coronary heart disease is as well established as had been previously thought. They suggest that some of the attributes of the type A behavior pattern are much more strongly associated with the incidence of coronary heart disease than are others, that the type A attribute of hostility is a strongly associated trait and that the attribute of anger-in (suppression of appropriate displays of anger or irritation) which is not presently included in the conceptual definition of type A behavior pattern but which is clearly related to hostility, may be more strongly associated with coronary heart disease. Another attribute, for example, job involvement which is included in the conceptual definition of type A behavior may be benign or even a protective correlate.

In the next chapter on coping and anger, Dr. Murray presents a series of studies from his laboratory suggesting that "the mere ventilation of anger is not a sufficient condition for the reduction of the instigation to angry aggression, arguing against a hydraulic model of catharsis in which it is assumed that anger can be drained off by simple expression." Instead, the author suggests that these results are more consonant with the notion that merely expressing anger, or even ruminating about it, may keep the person stirred up and more prone to be more aggressive at times. Combinations of anger reducing techniques that proved to be

effective in these studies were ventilation and cognitive reinterpretation, primarily when the ventilation was aimed at the provoker and the provoker provided the reinterpretation. The author attributes the effectiveness of these strategies to the restoration of equity. The ventilation procedure may be seen as an action designed to restore equity and the reinterpretation by the provoker as restoring equity. Thus, getting some sort of retraction by the provoker seems extremely important in reducing angry aggression, while getting no meaningful response from the provoker might be viewed as increasing inequity. Although these results are complicated by interactions with self-esteem factors, the author suggests that a loss of equity could reduce self-esteem and its gain could restore self-esteem. On the other side, low self-esteem could make the individual susceptible to inequities which the high self-esteem person ignores to a great extent. In any case, both anger and arousal and its reduction appear to be closely connected with the self-esteem of the person.

In a chapter by Paul Blaney, a critical review is provided on the literature on stress and depression in adults. Following on the contradictory and unsuccessful findings in the existing literature the author suggests the necessity of a more complex model which would require a greater theoretical consideration of the "goodness of fit" or "congruence" between stressors and possible moderators. "This line of reasoning would require taxonomies of stressors, personality styles, coping styles and support resources which are coordinated with one another." As an illustration, the author contrasts two stressors, death of a loved one and loss of job. In the case of "personality" as moderator, the author suggests that it may be that dependent persons are more stressed by death, while achievement-oriented persons are more stressed by job loss. Dr. Blaney suggests that problem-focused coping and support may be most useful as buffers in the case of job loss, where decisions must be made and actions taken. On the other hand, emotion-focused coping and support may be most useful in the case of death of a loved one. "When emotional needs are primary, problem focused coping and support may be worse than nothing at all, but when concrete action is needed, emotional oriented coping and support may distract from needed initiative." Blaney further suggests that effective buffers to stress are not only related to the availability of the repertoire of coping skills and support resources but also to the competency of the stressed person in knowing which needs are most important to deal with and being able to seek external support in a way which does not alienate others as well as the competency of persons in the network in perceiving which of the stressed person's needs are the most important to deal with and providing the needed support in a clear but noncontrolling fashion.

In the chapter by Jack Tapp entitled "Multisystems holistic model of health, stress and coping," the literature on hardiness versus helplessness and active coping versus conservation-withdrawal is reviewed. Dr. Tapp then proposes a model that describes the relationship between the organism's pattern of reactivity

distress that is dependent on the individual's hardiness-helplessness coping patterns and the degree of stress. Conservation-withdrawal reactions are reflected in physiological reactive patterns. The model, according to the author, provides for instances wherein the hardy individual who is overwhelmed with stressors fails to use active coping mechanisms and is stressed into conservation withdrawal. Similarly, a helpless individual under high degrees of stress would be expected to be forced into active coping patterns. A hardy individual would use conservation-withdrawal under appropriate circumstances as a means of exerting control in those instances where there is no control over some of the environmental circumstances.

The final chapter by Drs. Carver and Scheier on self consciousness, expectancies and the coping process is suitably an optimistic ending for this volume. The major focus of the chapter is the role that attentional processes play in the self-regulation of behavior. Following a review of the literature on a control-theory approach to behavior, self-regulation and stress in situations such as test anxiety, social anxiety and depression, and coping with life's difficulties, the authors address the question of optimism and successful coping. The most fundamental prediction they make is that "high levels of self-consciousness should facilitate efforts, persistence and striving towards effective coping among persons on the favorable side of the expectancy watershed—in this case among persons with a positive, optimistic outlook on life. But high levels of self-consciousness should have the opposite effect—they should be associated with impaired efforts, with disengagement and withdrawal among persons with a more negative, pessimistic outlook." Carver and Scheier, not unlike Lazarus et al., argue for the view that "positively toned emotions, such as those engendered by situational hope or dispositional optimism, can serve to increase the degree of persistence of efforts that a person demonstrates when dealing with life's stresses."

This, then, is an overview of our first volume on *Stress and Coping*. We hope that our consideration of these areas in terms of psychophysiological, developmental and psychosocial approaches will be as beneficial to the reader as it has been to us.

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ACKNOWLEDGMENTS

Several individuals, groups and organizations helped make this symposium possible. First, we thank the participants, who gave freely of themselves and helped carry out the symposium on a modest budget. Second, we thank our postdoctoral fellows and graduate

students, whose efforts attenuated the stress for all concerned. Third we thank Ellie Schneiderman for her gracious hospitality. Fourth, we thank for their support and encouragement Arthur Brown, Dean of the College of Arts and Sciences, Clyde Hendrick, former Chair of the Department of Psychology, Mary Jane Jesse, Vice Chair of the Department of Pediatrics, Robert S. Stempfel, Jr. Vice Chair of the Department of Pediatrics and Director of the Mailman Center for Child Development and Stephen Weiss, Chief of Behavioral Medicine, National Heart, Lung and Blood Institute. We gratefully acknowledge financial support from the University of Miami Graduate Student Association, the College of Arts and Sciences, the Mailman Center for Child Development, the Department of Psychology, and National Heart, Lung and Blood training grant HL07426.

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STRESS AND COPING
VOLUME 1

PSYCHOPHYSIOLOGICAL ASPECTS

1 Stress is a Noun! No, a Verb! No, an Adjective!

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INTRODUCTION

It has been my experience, both in laboratory experiments and in clinical studies, that the term stress has not been useful. In fact, it often seems to inhibit rather than facilitate communication either among scientists or between investigators and patients. There are a number of reasons for this difficulty: First, the term is ambiguous since it means different things to different investigators; and second, there are a number of incorrect premises implicit in most concepts of stress. In this chapter I review, briefly, several of the conceptual ways in which the term has been used and how this usage has led to confusion; then I consider some of our laboratory research findings which bear on the concept of stress as it has been used by a number of investiagtors; and finally, I suggest some alternative strategies for addressing the experimental and clinical issues to which the concept of stress often is applied.

CONCEPTS OF STRESS

In the latter part of the 17th century, Robert Hooke discovered the fundamental law existing between an external force and the resultant distortion of an elastic body. He proposed that the change in form of the elastic body was proportional to the deforming force. However, it was not until about 100 years later that Thomas Young formulated Hooke's law precisely by defining explicit physical concepts to be associated with "change in form" and "deforming force." Young defined stress as the ratio of the force within the elastic body which

balanced an external applied force, to the area over which the force acts. Thus, physically, stress is a response within an object which is inherent in its structure and which is elicited by the external force. Since any stress which is elicited from an object results in a physical change in that object, that change can be measured. For example, when the applied force is normal to the surface of an object, the object will change in volume, shape or size. These changes are called strains and are measured as the ratio of change in dimension to original dimension—that is, strain is unitless. These very precise physical concepts have been adopted by a wide range of biologists. Unfortunately, there is neither precision nor consistency among these usages. For example, the concept of stress is used in its precise, physical sense by physiologists who study isolated muscles in which responses can be readily measured as physical stresses or strains; however, the terms *stress* and *strain* also are used by organ physiologists whose uses of the terms often are obscure or inconsistent with the physical definitions—for example, contractility of the heart. Furthermore, the terms are used by psychologists working with whole animals where the notions of stress or strain bear only a metaphoric relationship to the physical concepts; the same terms are used by social scientists who apply them to nations or even to international institutions such as economies in contexts where they seem more like hyperboles than scientific concepts. Clearly, this spectrum of usages could—in my opinion, does—diminish rational discourse.

During the last century, as physiology, psychology and medicine evolved from intuitive to rational scientific and clinical disciplines, a number of experimental observations or clinical problems arose for which the concepts of stress or strain seemed useful. In most cases the problems were poorly explained by existing scientific principles, and stress or strain were convenient euphemisms for ignorance. In general, the problems for which these terms were invoked were ones in which either the animal itself was confronted with a strong and/or enduring situation which clearly elicited autonomic and hormonal responses—sometimes characterized as "emergency reactions"—or problems in which it was observed that specific organs emitted strong and enduring responses, sometimes to the point of failure. Thus, concepts of stress evolved that used similar words to characterize vastly different processes. Even when referring to the same data, different investigators often characterized aspects of the same process differently. Thus, the distinguished 19th century physiologist, Claude Bernard, defined stress as an adaptive response to an external stimulus; while the equally distinguished 20th century physiologist, Walter Cannon, defined stress as the stimulus (1963).

Even among those scientists who adhered to the response notion of stress, there emerged important or even inconsistent differences which either were not considered explicitly or were not resolved. For Claude Bernard, stress was an attempt of the body to return an affected system to its homeostatic state, and strain was the relative departure of the system from its homeostatic set point. When the magnitude of the adaptive response was inadequate to counter the

noxious stimulus, or when the adaptive response caused more tissue damage than did the original stimulus, the consequence was disease. Bernard's response notion of stress obviously adhered to the physical concept although it has not been easy to measure strain in such systems. Selye, writing many years later (1974), preserved the response notion of stress; however, he also introduced an important conceptual variant. He limited the stress to the "non-specific" reactions of the body to the external stimulus. Obviously, in doing this Selye lost totally Hooke's notion of specificity; namely, that stress was a function of the nature of the deforming force. Furthermore, Selye emphasized an important physiological and psychological aspect of stimuli which is missing in the physical model: the stress can endure long after the stimulus is withdrawn.

As I have already noted, Cannon defined stress as the external force, i.e., as a stimulus. Most investigators who study whole animals in an attempt to identify behavioral/physiological interactions also equate stress with the stimulus (e.g., see for reviews, Galosy, Clark, Vasko, & Crawford, 1981; Turkkan, Brady & Harris, 1982; Engel & Schneiderman, 1984). Furthermore, many scientists who study human behavior also conceive stress as a stimulus (e.g., see for reviews, Glass, 1977; Rose & Levin, 1979). However, there are a number of scientists who define stress as an interaction between the environment and the individual. Harold Wolff defined stress as "the interaction between the external environment and the organism with the past experiences of the organism as a major factor (1953, p. v)." Recently, Williams (1985) has proposed an interactive model of stress that includes innate or acquired characteristics of the organism, which interact with the environment to produce "qualitatively different patterns of response—extending across many response systems, . . . " Neither the stimulus definitions of stress nor the interactive definitions of stress are consistent with the physical concept of stress. Furthermore, the interactive concepts of stress pose very difficult measurement problems. For example, in Wolff's model it is very difficult to assign any quantitative value to "past experiences," and in Williams' model it is virtually impossible to demonstrate that one has measured all of "the qualitatively different patterns of responses." Thus, it is difficult to see how one could ever design a test of these models.

Lazarus (1966) recognized the diversity of ways in which stress had been used. His solution was to use the term in still another way:

> It seems wise to use "stress" as a generic term for the whole area of problems that includes the stimuli producing stress reactions, the reactions themselves and the various intervening processes . . . . As used here it will be nothing more than a general label like motivation or cognition. It defines a large, complex, amorphous, interdisciplinary area of interest and study. (p. 27)

In this brief review I have tried to show how the terms, stress or strain, have evolved from very precise physical concepts to diverse and inconsistent biological concepts which often are neither testable nor quantifiable. I wish to empha-

size that the problems addressed by various theorists cited earlier are significant and worthy of study. It is only the insistence on using the terms, stress and strain, and some other hypothetical notions that need redressing.

SOME ERRONEOUS ASSUMPTIONS ABOUT STRESS

As I indicated at the beginning of this paper, I have not found the concept of stress useful, and on occasion have even found it counterproductive. In this section I want to consider two specific assumptions which often are made about stress: One clearly is wrong, and the other has been stated so vaguely that one cannot judge its validity. Like most assumptions, these are made in order to simplify the conceptual models. While these assumptions surely do not exhaust the premises made by various theorists, nor do all theorists make these assumptions, I believe that they are sufficiently common, and so clearly confusing, that they merit special consideration. The first assumption I will consider is that autonomically mediated responses can be used interchangeably as indices of stress, or stressfulness, depending upon whether one uses a response or a stimulus definition of stress. The second assumption I will consider is that aversive stimuli—namely, stresses or stressors—elicit nonspecific, nonadaptive responses.

THE INDEX ASSUMPTION

The notion that one or another physiological response can be used as an index of all responses is one that has been with us for years (e.g., Lacey, 1956). It is a very attractive idea for several reasons. First, it is very convenient to have a single index to measure a complex system. Second, it is very economical to be able to measure one index rather than a multitude of effects, especially if the effects are dynamic—that is, they change over time. Third, if one response can serve as an index of several processes, one need not be knowledgeable about so many things; one need only be a specialist in indices. There are a number of examples of the index notion in the literature. These include experimental studies in which one or another physiological response is measured, and its fluctuations are defined as measures of the degree of stress. For some investigators this index is heart rate or electrodermal activity; for others it is catecholamine response. There are also clinical examples of the use of indices: Whenever a therapist proposes to treat a patient's stress response through biofeedback or through relaxation, that therapist is implicitly proposing that some measure or another can be used as an index of the patient's problem, since the therapeutic assumption is that when the biofeedback or relaxation "therapy" is being implemented successfully—namely, the index is being modified successfully,—the patient's

"stress" is being alleviated. What is the evidence that any autonomic response can be used to index the autonomic nervous system?

The most popular theory which incorporates the index notion is so-called activation theory (Duffy, 1972; Lindsley, 1951). Activation theory proposes that there exists a generalized state of physiological arousal ranging from sleep or unconsciousness at one extreme to excitement or agitation at the other extreme. Stress (or stress response) falls toward the excitement end of the continuum. This notion derives from the view, once widely held, that autonomic responses are non specific, all-or-none effects (Cannon, 1929). Phrases such as "massive sympathetic discharge" often are used to characterize a subject's response to stress. If such a general activation occurred, measuring any one manifestation of it would provide a reasonable index of the process. However, it should be clear that the notion of general activation is wrong. As Wurster (1977, p. 239) noted: "The monolithic view of mass sympathetic discharge has fallen."

The alternative model to activation theory is specificity. This concept has its roots in psychosomatic theories which related specific diseases to specific emotions (Alexander, 1943). The most well-known proponent of this view was Dunbar (1938) who argued that specific affects, which were elicited during the course of psychoanalysis could be causally linked to the evolution of specific diseases. The linkage of specific, affective, verbal reports to specific diseases has been refuted. However, the experimental evidence of patterning among peripheral autonomic responses remains firm (Engel, 1972a; Foerster, Schneider, & Walschburger, 1983; Garwood, Engel, & Capriotti, 1982). Autonomic response patterns are specific to the stimulus which elicits them; and they are specific to the subject who emits them—that is, if a group of subjects are exposed to a single stimulus, the stimulus will elicit consistent responses across subjects; and if a single subject is exposed to a set of different stimuli, he will emit a consistent pattern of responses to those stimuli which will be different from the response pattern emitted by another subject. The former has been called stimulus response specificity, and the latter is called individual response specificity (Engel, 1972a; Engel & Moos, 1967). It is individual response specificity, which is especially relevant to the notion that one can use a specific autonomic response as an index of stress. The evidence is clear that different individuals respond to the same set ot stimuli differently, but a single subject responds to different stimuli consistently. Furthermore, the evidence is strong that the consistency of response can be pathognomonic. Thus, persons with complaints related to the head and neck region tend to overreact in muscles spanning those areas, while anxious subjects with cardiac complaints tend to be cardiac reactors (Malmo & Shagass, 1949). A hypertensive patient will respond to a battery of stimuli with response patterns characterized by a high degree of consistency—that is, a given patient is likely to respond with similar patterns to diverse stimuli, and is likely to react maximally in blood pressure no matter what the stimulus may be (Engel & Bickford, 1961). It is clear that the existence of

reliable, individual differences in patterns of physiological reactions to specific stimuli is inconsistent with the notion that one can arbitrarily select any single physiological response and use it as an index of stress. It also should be noted here that psychologists have long been aware that there exist reliable individual differences—e.g., personality traits. Therefore, it should not be surprising that there are reliable, individual differences in physiological performance, just as there are reliable differences in overt or verbal behavior. It is just as unreasonable to characterize someone's physiological reactivity on the basis of a single index as it is to characterize his overt behavior on the basis of a single trait.

THE NONSPECIFICITY ASSUMPTION

The nonspecificity notion is related to the index notion. If there are such things as nonspecific responses to stress (or nonspecific stresses to stressors), then it is likely that these nonspecific responses could be useful indices of stress if they varied systematically. As it is, there are several possible meanings for the concept of nonspecificity: (a) There is a set of responses which occurs reliably, following any superthreshold stimulation; (b) the magnitude of the responses is quantitatively related to the intensity of stimulation but independent of the nature of the stimuli; (c) the responses are elicited in a reliable order which varies with the intensity of the stimuli; (d) the responses not only are elicited in a reliable order, but also vary quantitatively with stimulus intensity once they are elicited; and (e) not only are the responses elicited selectively, but also the responses adapt and/or fatigue selectively. It is clear that these five versions of nonspecificity are progressively more complex; yet each only deals with the stimuli as unique, independent events. However, it is well-known to anyone who studies behaving organisms that stimuli are not merely unique, independent events whose effects are additive. One needs to take into account the laws of behavior when he assesses "stimulus intensity." These laws address such issues as learning history, reinforcement properties, and generalization or interference effects. Furthermore, one needs to understand that multiple stimuli do not merely add, they interact. For example, several years ago Ainslie and I (1974) studied the effect of operant cardiac conditioning on classical cardiac conditioning. We first trained monkeys to associate a 2 min series of clicks with an inescapable electric shock. This procedure is widely used in stress research; for example, when the clicks are superimposed on an appetitive response, it suppresses eating and the process has been called the conditioned emotional response. One effect of the clicks is to produce an increase in heart rate and blood pressure during the 2 min period. After the tachycardia and pressor response had been reliably conditioned to the clicks, we trained some animals to speed their heart rates and other animals to slow their hearts to avoid tail shock. Thus, the animals learned a "coping" behavior, namely that changing heart rate could avoid tail shock. After the

animals had mastered this skill, we repeated the operant cardiac procedure but now superimposed the classical stimulus (clicks) and the unavoidable tail shocks on the schedule. The question, of course, was, "would the classically conditioned stimulus (the stressor) control the cardiovascular responses, or would the operant schedule control these responses?" We found that the heart rate obeyed the operant contingencies: in animals trained to slow heart rate, when the clicks were sounded, heart rate slowed even more; however, in animals trained to speed heart rate there was a tachycardia during the clicks. At the same time, the blood pressure which had never been an operant, continued to rise during the occurrence of the clicks. Thus, before one can begin to characterize responses as nonspecific, he must first define what he means by nonspecificity, and then he must show that the effects he is observing are truly nonspecific—that is, independent of prior experience.

It is not clear whether the nonspecificity notion is wrong, in part because many theorists who refer to nonspecific indices of stress do not make clear which of the five versions of nonspeficity they mean; and in part, because many of the studies in which nonspecific responses are used to gauge stress are not designed with enough behavioral controls to enable one to judge whether the responses—namely, adaptations—observed are general, nonspecific effects, or whether they are learned coping skills which have been generalized. However, it is clear that the discovery that autonomic responses can be operantly conditioned (Engel, 1972b; Miller, 1969) does make the notion of nonspecificity one that needs to be demonstrated very carefully before it can be asserted.

A MODEST PROPOSAL

Scientific concepts are invented because they serve a useful purpose; they permit one to synthesize a number of isolated observations into a general principle. Eventually, as new data are accumulated, old concepts are replaced by new concepts. The problem with the term stress is that too many investigators have tried to append new data to an old idea. The result of this accretion process is that each new theorist has tried to preserve the observations of his predecessor while often ignoring the theoretical frame of reference in which the data were collected. Thus, we are burdened with a term which is scientifically bad because it lacks the fundamental attributes that a valid scientific concept must have: internal consistency and testability: The term stress clearly is used inconsistently, because for some theorists it refers to a stimulus, while for others it refers to a response, and for still others it means an interaction between genetic predispositions and acquired experiences. The term often is used in a way which makes it untestable—for example, when it is equated to an aversive stimulus as merely a synonym for the stimulus with no conceptual status. Furthermore, the term is used as a noun—*the stress*, as a verb—*to stress*, and as an adjective—*the stress*

response. Finally, because of this irrational accretion of meanings, and because of the multiplicity of contexts in which it is purported to operate, investigators have invented factually wrong oversimplifications—for example, by asserting that some responses can be used as indices of the entire autonomic nervous system despite evidence to the contrary. It seems to me that the time has come to wipe the conceptual slate clean and start over.

There are a number of changes that scientists and clinicians could adopt which, in my judgment, would prove beneficial both to science and to patients. First, we could be less otiose in our language. Instead of using stress as a label—for example, as a synonym for aversive stimuli—we could be explicit in characterizing the stimulus-response relationships we are actually observing, and we could specify the conditions under which we observe those relationships. Subsequently, we could design tests to see whether the relationships are unique to the conditions under which they were observed, or whether they do generalize to other contexts. For example, it has been noted that so-called, type-A persons tend to be hyper-reactive when tested in laboratory settings, and some investigators have inferred that this tendency occurs in natural settings as well (Glass, 1977). It would be helpful to test this hypothesis by carrying out the appropriate field studies. Second, we should stop invoking stress as a catchall to disguise the fact that we do not understand why a phenomenon is occurring. If we were to adopt this position, then we might find it possible to design procedures for obtaining the necessary data rather than pretending they are in hand. For example, instead of ascribing behavior to prior learning, it would be better to document the prior learning and then predict future behavior on that basis. It has been my experience that when I can do so, the term stress becomes superfluous both for me and for my patient. Third, we should begin to attend more to coping. There are two, broad lines of evidence to suggest that this avenue will be productive. From a scientific point of view, it seems very likely to me that we will learn more about control mechanisms when our experimental subjects are dealing successfully with our aversive stimuli, than we will when they are failing to do so. From a clinical point of view, the therapeutic challenge is to cope successfully, not to suffer insightfully. While assessment or diagnosis is essential to a successful therapeutic outcome, it is the outcome and not the cause which is the raison d'etre of the therapist.

CONCLUSION

I am sure that most people reading this paper agree that the concept of stress is troublesome. I am equally confident that few of those readers will abandon the concept, despite its counterproductive attributes. For them, stress is neither a noun, nor a verb nor an adjective. It is an escape from reality.

ACKNOWLEDGMENT

I want to express my appreciation to Drs. Kathryn Burgio, Marc Heft, and Robert McRae for their helpful comments to early drafts of this paper.

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2 Biobehavioral Responses To Stressors

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A large number of studies during the past several decades have explored relationships among environmental stimuli, psychological processes, and organismic responses. Those studies that have investigated the various ways in which environmental changes or the perceived threat of such changes lead organisms to make specific adaptive responses are often subsumed under the rubric of stress research. Lazarus (1966), for example, used the term stress as a "generic term for the whole area of problems that includes the stimuli producing stress reactions, the reactions themselves and . . . various intervening processes."

In Chapter 1 of this volume, Dr. Engel points out several ways in which the term stress has been inconsistently applied and has led to confusion. He therefore suggests that the concept should be abandoned by biobehavioral researchers. We agree with Engel's contentions that operational definition is important, that scientific terms should be used consistently, and that concepts such as stress should not be reified. We also believe, however, that stress is useful as a generic term, because it has permitted scientists to synthesize large numbers of otherwise isolated and disparate observations into a relatively few general principles. For our purposes, then, stress is an organizing term rather than an explanatory one.

In the present chapter, stress is defined as a change or threat of change demanding adaptation by an organism. The stimulus for this change is called a stressor, and the adaptation is described in terms of behavioral (e.g., fight or flight) and physiochemical (e.g., increased cardiac output or secretion of norepinephrine) responses. In biobehavioral research, stressors may be physical, psychosocial and/or sociocultural; adaptations may involve the skeletal musculature and/or other bodily systems. Organismic variables interposed between the stressors and adaptations involve personality variables including perceptual and behavioral response styles.

BASIC BIOLOGICAL AND BEHAVIORAL PROCESSES

Hans Selye, who was a major pioneer in stress research, defined stress as a nonspecific response of the body to external demands (Selye, 1936, 1946, 1970). This response included an increased discharge of adrenocorticotrophic hormone (ACTH), glucocorticoids, catecholamines, and the development of thymicolymphatic involution and eosinopenia. Although Selye believed that potent stressors mobilized the entire general adaptive system of the organism, he also contended that specific aspects of a stressor could influence the stereotyped stress response by suppressing or enhancing some of its manifestations.

As pointed out elsewhere (McCabe & Schneiderman, 1984), Selye's view that stressors elicit nonspecific responses was consistent with the neurophysiological (e.g., Magoun, 1958) and psychological (e.g., Schacter, 1966) arousal theories of the 1950s and 1960s. Subsequent work, however, has indicated that the responses to various stressors are more specific than was previously supposed. Mason (1975), for instance, found that heat, cold, exercise, and food deprivation stressors each led to a different profile of hormonal response, and that in the absence of psychological threat, corticosteroid production did not increase.

Although various stressors may elicit different responses, the central nervous system (CNS) appears to be organized to produce integrated responses, rather than changes in single, isolated variables (Hilton, 1975). There is some evidence, for example, that individuals confronted with an aversive situation tend to reveal one pattern of autonomic reactivity if appropriate coping responses are being attempted, but another pattern if coping responses seem unavailable. The former pattern, which is commonly referred to as the defense reaction, has been described extensively (Abrahams, Hilton, & Zbrozyna, 1960; Cannon, 1929; Hess, 1957). It includes increased striate muscle activity, vasodilation in skeletal muscle, heart rate (HR), cardiac output, and blood pressure (BP). A second pattern that occurs during aversive situations in which an active coping response does not seem available to the organism includes increased vigilance, total peripheral resistance, BP, and other manifestations of sympathetic nervous system (SNS) activity accompanied by decreased skeletal movement and a vagally mediated decrease in HR (bradycardia).

The defense reaction or active coping response can be seen during flight or fight (e.g., Adams, Bacelli, Mancia, & Zanchetti, 1968) or during active avoidance conditioning (e.g., Langer, Obrist, & McCubbin, 1979). It can also be seen in attenuated form during the performance of less obviously aversive tasks such as mental arithmetic (e.g., Brod, 1963) or the playing of video-arcade type games (e.g., Glass, Krakoff, Contrada, Hilton, Kehoe, Mannucci, Collins, Snow, & Elting, 1980).

The pattern of responses, which occurs in aversive situations in which an active coping (effortful, defensive) response is not apparent to the organism, has

been reported to occur in the time period immediately preceding instrumental avoidance conditioning sessions in dogs (e.g., Anderson & Tosheff, 1973). It has also been seen during preparation for fighting in cats, both in the short periods preceding contact, and in longer trials during which the animals have been prevented from fighting by a barrier (Adams et al., 1968). Although this pattern of responses, which includes decreased skeletal motor activity, HR, and cardiac output; is commonly seen in aversive situations in which a coping response is not evident, it is also seen—albeit with smaller magnitude responses—during less threatening situations (e.g., reaction time tasks) in which attentive observation of the environment is required (e.g., Williams, Bittker, Buschbaum, & Wayne, 1975).

In our laboratory, we have been tracing CNS pathways that are involved in different patterns of cardiovascular response (e.g., Gellman, Schneiderman, Wallach, & Le Blanc, 1981; Gimpl, Brickman, Kaufman, & Schneiderman, 1976; Kaufman, Hamilton, Wallach, Petrik, & Schneiderman, 1979). Gellman et al. (1981), for example, identified two pathways through the hypothalamus of rabbits, an observation which may provide insight into the CNS substates underlying the active coping (defense reaction) and vigilance (attentional) patterns of response to aversive situations. Thus, electrical microstimulation of the medial hypothalamus, particularly the ventromedial hypothalamic nucleus, elicited circling movements, hind-limb thumping, and increases in HR and BP. In contrast, stimulation of electrode sites only slightly more lateral in the anterior and posterior hypothalamus resulted in tonic immobility, except for a raising of the ears and orienting-like slow movement of the head. Stimulation of these latter hypothalamic sites resulted also in an increase in BP and a decrease in HR mediated by the vagus nerves. In preliminary work, we have also observed that stimulation of the ventromedial hypothalamus in the rabbit leads to an increase in plasma epinephrine and damage to the endothelium of the aorta, observable under the elctron microscope. These findings are consistent with results reported by Sudakov and Yumatov (1978) and by Ulyaninsky, Stephanyan, and Krymsky (1978). Interestingly, both the defense reaction and the vigilance patterns of response were elicited by stimulating the same regions of the hypothalamus that induced rabbits to avoid or escape train electrical stimulation (Sideroff, Elster, & Schneiderman, 1972).

Although alternative patterns of cardiovascular response clearly seem to be associated with active coping and its absence, many important questions related to this dichotomy remain. The extent to which variations in effortfulness, aversiveness, and individual differences may modify the basic responses is unknown. So, too, is the full extent of the manner in which a host of concommitants including glucocorticoids, endogenous opioids, and a variety of hormones may vary as function of behavioral contingencies. Distinctions among active avoidance, passive avoidance and helplessness also have to be investigated and related to physiological changes associated with the basic patterns.

An example of the manner in which the hormonal concomitants of the active coping pattern can vary is seen in the work of Frankenhaeuser (1983). She has provided evidence that effort associated with distress is accompanied by an increase in both catecholamine and cortisol secretion; whereas, effort without distress is accompanied by an increase in catecholamines and cortisol suppression.

Moderate effort without distress (e.g., walking stairs) may lead to the preferential release of plasma norepinephrine (NE); whereas, effort associated with psychological stress (e.g., self-consciousness, harassment) may lead to the preferential release of epinephrine (E) (Dimsdale & Moss, 1980; Glass et al., 1980). Although the release of either E or NE can activate beta-1 adrenoceptors at the heart leading to increased cardiac output, increases in plasma E relative to NE during the maximal effort of competitive sport or during life-threatening emergencies are associated with larger magnitude cardiovascular responses.

Patterns of response can also be seen to change over time as a function of autoregulation, neuronal and hormonal processes. At the outset of highly aversive, ambiguous, overwhelming situations in which the availability of a coping mechanism may not be apparent to the organism, responses may include pronounced SNS arousal, increases in BP, HR, cardiac output, vasodilation in skeletal muscles, secretion of E and NE from the adrenal medulla and activation of the adrenal cortex (e.g., Mason, Mangan, Brady, Conrad, & Rioch, 1961). As the initial, ambiguous, overwhelming situation becomes more predictable and subject to control, both E and cortisol tend to diminish, while motor performance is related to NE release (Frankenhaeuser, 1980; Mason, 1975).

In summary, it appears that in the presence of a novel, intense, highly aversive stressor, in which a coping response is not readily apparent, mammals, including humans, respond by mobilizing both the sympatho-adrenomedullary (SAM) and hypothalamico-pituitary-adrenocortical (HPAC) systems. In less overwhelming situations, the nature of the stimulus, the availability or non-availability of a coping response, and individual difference variables can influence the pattern of physiological responses. In the next sections we look at the manner in which various, potentially interacting response systems are influenced by stressors, and examine the manner in which they may mediate relationships between stress and disease.

RESPONSE SYSTEMS

The psychophysiological responses to stressors can be manifested through several biological response systems. The primary bodily response in active coping is through the SAM system. This system plays a preeminent role by increasing metabolic activity in response to situations perceived as being stressful. However, other systems, such as the HPAC may play some role in the psycho-

physiology of active coping by acting synergistically with the SAM system. For instance, in emergency situations, the glucocorticoids cause the rapid mobilization of amino acids and fats from cellular stores. These then become available directly as an energy source and as a source for the synthesis of energy-rich glucose. The endogenous opiate system may also play a role in active coping by reducing fear, inhibiting pain-related withdrawal behaviors, and providing analgesia during fighting and other coping reactions.

During highly stressful situations in which a coping response is not evident, physiological responses include increased sympathetic activity associated with decreased cardiac output and HR (e.g., von Holst, 1972), increased ACTH and adrenocortical secretions (e.g., Selye, 1936, 1946), decreased levels of brain norepinephrine (NE) (e.g., Weiss, Glazer, & Pohorecky, 1976), and suppression of the immune system (e.g., Riley, 1981). The endogenous opiate system may also be involved. Given the relationship of various physiological responses to stressors, it would be useful to examine each response system in detail.

Sympatho-Adrenomedullary (SAM) System

During active coping situations, particularly in those perceived as threatening, the SNS releases NE from sympathetic nerve terminals and NE and E from the adrenal medulla. Although Cannon (1929) suggested that activation of the SNS was accomplished as a unitary mass-discharge, this is actually not the case. Plasma NE is preferentially released during moderate exercise (e.g., Dimsdale & Moss, 1980); whereas, plasma E is preferentially released during SNS activity associated with emotional behavior (e.g., Dimsdale & Moss, 1980; Glass et al., 1980). Differential SNS activity is also seen during active coping versus aroused helplessness.

During behavioral stress that elicits active coping (e.g., the defense reaction), the heart increases its rate and force of contraction. The venous side of the circulation decreases its volume, thereby increasing the return of venous blood to the heart. Cardiac output is increased. The arterioles in the skin and gut constrict, increasing resistance to blood flow. This is at least partially offset, however, by increased flow of blood to skeletal muscle. Activation of the SNS during the defense reaction also stimulates (a) adipose tissue to mobilize free fatty acids, (b) the liver to release glucose, and (c) the kidney to release renin and to decrease the excretion of sodium and water. The increases in free fatty acids and glucose provide increased energy resources, and the effects upon the kidney increase arterial blood pressure via the renin-angiotensin-aldosterone system.

In contrast to the defense reaction, which is elicited in situations involving active coping, another pattern of SNS activation occurs in aversive situations in which active coping is not perceived as being possible. In this situation, increases in BP are largely due to an increase in peripheral resistance associated

with vasoconstriction in the skin, gut, and skeletal muscles. Heart rate and cardiac output are actually decreased.

It is important to understand that sympathetic activation can be highly adaptive in challenging situations. Emergency or athletic situations are aided by activation of the SNS. However, if the challenge is too severe, too prolonged, or perhaps too often repeated, or if the individual is in poor physical condition, activation of the SNS can aggravate existing disorders or initiate new pathology. Thus, while Selye's (1936) general adaptation syndrome was originally conceptualized in terms of HPAC activity, it has also become a useful concept in terms of SNS function.

Several studies have suggested that there seems to be a difference between SNS activity occurring during emotional stress (e.g., the defense reaction) not necessarily involving pronounced exertion and SNS activity occurring during aerobic exercise. When catecholamines are released during either exertion or emotional stress, they mobilize lipid stores from adipose tissues (Heindel, Orci, & Jeanrenaud, 1975), which are hydrolized to free fatty acids for energy production in muscular activity (Zieler, Maseri, Klassen, Rabinowitz, & Burgess, 1968). In this way, SNS activation related to exercise leads to the effective utilization and rapid clearance of free fatty acids from the circulation. In contrast, when lipid mobilization induced by emotional stress is not accompanied by vigorous physical activities, the free fatty acids are not cleared as rapidly, and some of them become converted to triglycerides by the liver. These are then circulated as a component of *very low density lipoproteins* (VLDLs) in the blood (Schonfeld & Pfleger, 1971). Remants of these VLDLs become converted into *low density liproproteins* (LDLs) by the liver and are then returned to circulation (Sigurdsson, Nicoll, & Lewis, 1975). It is the LDLs that are the source of most lipid in atherosclerotic plaques (Miller, 1980).

Another important physiological difference between moderate aerobic exertion and sedentary emotional stress is the preferential release of E and NE. Most experimental evidence is consistent with the view that NE is preferentially released during moderate exercise (e.g., Dimsdale & Moss, 1980) and that E is preferentially released during emotional stress (e.g., Dimsdale & Moss, 1980; Glass et al., 1980). The release of NE at the heart during exercise occurs via nerve terminals primarily at the sinoatrial (SA) and atrioventricular (AV) nodes with some limited innervation also occurring at the ventricles. Interestingly, most beta-adrenergic receptors in the heart are not found in the vicinity of the SA and AV node, but are found in the left ventricle (Baker & Potter, 1980). Moreover, most of these receptors in the ventricle cannot be activated by the release of NE by sympathetic nerves. Instead, these receptors appear to be activated by circulating E released by the adrenal medulla in life-threatening situations (e.g., hemorrhage, severe acidosis) or during emotional stress. In hearts compromised by myocardial ischemia, circulating E could sensitize the myocardium and lead to potentially lethal arrhythmias.

Hypothalamico-Pituitary-Adrenocortical (HPAC) System

A wide variety of physical, neurogenic and behavioral stressors can activate the HPAC system. Such stressors include intense heat or cold, infection, surgery, physical restraint or administration of sympathomimetic drugs. Emotional stress associated with novelty, uncertainty, and unpredictability are potent stimuli for both catecholamine and corticosteroid release (Mason, 1975). As previously mentioned, the HPAC system is also activated in response to emotionally stressful situations in which an active coping mechanism is not available (e.g., Henry & Meehan, 1981; Schneiderman, 1983a,b). The resulting behavior is characterized by extreme vigilance, inhibition of movement, SNS activation, but also bradycardia mediated by the parasympathetic nervous system. This "conservation-withdrawal pattern" (Selye, 1946, 1956, 1976) has been associated with suppression of the immune system, peptic ulceration, clinical depression and various cardiovascular disorders (e.g., Henry & Stephens, 1977).

The mechanism by which the HPAC system becomes activated has been traced to parvocellular neurons in the medial basal hypothalamus. Thus, the system is actually under the control of the CNS. In response to stimulation, the neurons in the medial basal hypothalamus secrete *corticotropin releasing factor* (CRF). The chemical structure of CRF is presently unknown, although most hypothalamic releasing and inhibiting hormones appear to be small peptides. In any event, the CRF is transported to the anterior pituitary (i.e., adenohypophysis) via the hypothalamico-hypophysial portal system, which consists of a network of small blood vessels. In the adenohypophysis, CRF stimulates secretion of ACTH into the systemic circulation. The target organ, which is the adrenal cortex, is then activated by ACTH to release adrenocorticosteroids, such as cortisol and corticosterone. Humans and other primates primarily release cortisol (hydrocortisone); whereas, rodents primarily release corticosterone.

The HPAC system appears to be under negative feedback control mechanisms. A stressor, for example, can activate the HPAC system, causing the release of corticosteroids within minutes. The release of corticosteroids, in turn, initiates metabolic effects directed at relieving damage caused by the stressor (e.g., reduction of fever or inflammation). In addition, corticosteroids have direct negative feedback effects (a) on the hypothalamus to decrease formation of CRF, and (b) on the adenohypophysis to decrease the formation of ACTH. This feedback serves to stabilize the concentration of corticosteroids in the circulation when the body is not experiencing stress. However, the stabilizing feedback is disrupted when a stressor does appear.

Several studies have contrasted SAM and HPAC activity during behavioral experiments in humans. In one of these studies, Lundberg (1980) found that a vigilance task, which was interpreted to be stressful, produced excretion of cortisol. In contrast, a self-paced reaction time task elicited the preferential excretion of catecholamines. Both Lundberg (1980) and Frankenhaeuser (1980)