Chapter 5
Measurement of the Human Stress Response

In the final analysis, the empirical foundation of epistemology is measurement.

When an unexplained phenomenon, such as a stress-related disease, is first observed, it is common to search for possible etiological factors. This search often culminates in a phenomenological theory; in this case, perhaps a theory of stress arousal and subsequent pathogenesis. On the basis of the formulated theory, for example, of stress arousal, it is then a useful next step to design an experiment in order to test the theory and any proposed relationships critical to the theory. Inherent in the design of the experiment is the designation of key variables and some means of measuring, recording, or otherwise quantifying those relevant variables. Relevant to the present discussion, this would typically involve a means of measuring the stress response and perhaps its pathological effects.

As we review the literature concerning human stress, it is obvious that in addition to the lack of a universal definition of stress, the field has also been plagued by a plethora of inconsistencies and potential phenomenological errors in the measurement of the human stress response. If we cannot reliably and validly measure the human stress response, what degree of credibility do we place upon investigations into its phenomenology? Indeed, meta-analytic research has suggested that the measurement of independent and dependent variables may be the single most important aspect of research design—even more important than the structure of the research design itself (Cohen, 1984; Fiske, 1983; Smith, Glass, & Miller, 1980). With regard to stress research, it may be argued that the confounded or inappropriate measurement process has the greatest ability to limit the generation of useful data regarding this important public health phenomenon (Cattell & Scheier, 1961; Everly & Sobelman, 1987; Stamm, 1996). Thus, the purpose of this chapter is to discuss the measurement of the human stress response.

In Chap. 2, a systems model of the nature of the human stress response was constructed (Fig. 2.6). As a means of integrating the following measurement-based discussions, that basic model is reproduced here, with key measurement technologies having been superimposed (see Fig. 5.1). Let us take this opportunity to examine more closely the measurement of the human stress response.

G.S. Everly and J.M. Lating, *A Clinical Guide to the Treatment of the Human Stress Response*, DOI 10.1007/978-1-4614-5538-7_5,

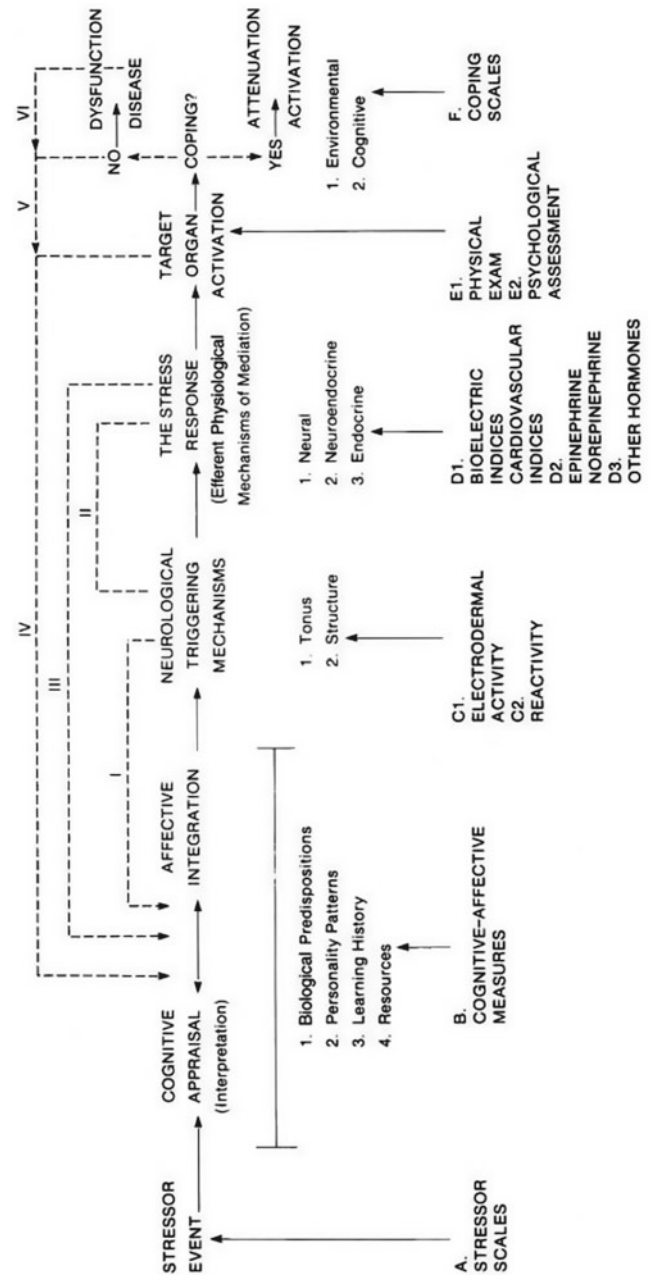

Fig. 5.1 Measurement of the human stress response

Stressor Scales

Historically, one of the most widely used measurement tool for the assessment of human stress, in reality, does not measure stress at all—it measures stressors. The Social Readjustment Rating Scale (SRRS), the "grandfather" of attempts at measuring stress, was developed by Thomas Holmes and Richard Rahe (1967), based upon the theory that "life change" is causally associated with subsequent illness. This notion was by no means a new idea. Adolph Meyer pioneered empirical investigations into the relationship between psychosocial events and illness with the advent of his "life chart" as a means of creating a medical history.

The SRRS contains 43 items consisting of commonly experienced "life events." Each life event is weighted with a life change unit score (LCU). Respondents are simply asked to check each of the items they have experienced within the last 12 months. The arithmetic summation of LCUs represents the total LCU score, which can then be converted to a relative health risk statement, that is, the risk of becoming ill within a stipulated time period. The association between high LCU scores and risk of subsequent illness is assumed to be a function of the fact that organisms must adapt to novel stimuli and otherwise new life events. The physiology of adaptation has long been known to be the same physiology as the stress response. Thus, stress may be seen as the linchpin between life events and illness as conceived of and measured by the SRRS.

The SRRS is not without its critics, however. Two major issues have been raised:

1. Life events scales should be modified so as to assess the perceived desirability of the life event. It has been suggested that negative life events are potentially more pathogenic than positive life events (Sarason, Johnson, & Siegel, 1978).
2. It has also been suggested that "minor hassles" are more important predictors of illness than major life events (Kanner, Coyne, Schaefer, & Lazarus, 1981).

Other noteworthy efforts in the assessment of stressor stimuli should be mentioned. In an attempt to improve the SRRS with regard to the issue of event desirability, Sarason and his colleagues (1978) created the Life Experiences Survey (LES), which not only lists a series of life events but also inquires into the desirability of each of the events. In a far more ingenious approach to the life events issue, Lazarus and his colleagues investigated the daily hassles versus major life events issue as it pertains to the prediction of subsequent illness (Kanner et al., 1981). The Hassles Scale lists a series of minor daily hassles, that is, sources of frustration that commonly recur to many individuals. The scale also includes an "uplifts" assessment that theoretically serves to mitigate the adverse impact of negative life events.

The LES and the Hassles Scale are creative and alternative approaches to the assessment of stressors. Another entry into the genre of stressor scales is the Stressful Life Experiences Screening (SLES; Stamm, 1996). This instrument consists of 20 items that inquire as to (1) the presence of a stressful life experience, and (2) the degree of "stressfulness" of that experience. The long form (SLES-L) takes 5–10 min to complete.

The Life Stressor Checklist—Revised (Wolfe & Kimerling, 1998) consists of 30 "events" that satisfy the DSM-IV definition of traumatic. The self-report scale takes 15–30 min to complete and is designed for use with adults. The scale is not only an indicator of traumatic events, but it also serves as an assessment of the events' current impact upon the individual.

Most recently, the Stress and Adversity Inventory (STRAIN) was created by the University of Los Angeles's Laboratory for Stress Assessment and Research. This is a computerized self-report inventory that consists of 96 questions that cover moderate to severe stressors that are typically experienced by the adult population. The strain is designed for use with adults and takes approximately 30–45 min to complete (UCLA Laboratory for Stress Assessment and Research: http://www.uclastresslab.org/products/strain–stress-and-adversity-inventory).

As for the genre of stressor scales, Monroe (1983) notes, "Although findings of event—illness associations appear to be consistent in that increased life events predict dysfunction in both retrospective and prospective studies, the magnitude of the association reported typically has been low" (p. 190). The recognized consistency in the life events research combined with its low-effect size leads one to believe that life events scales such as the SRRS do indeed tap some domain that has meaning in stress phenomenology; however, there appear to be other mediating variables that need to be better understood. From the view of the present model, life events scales tap the stressor domain and therefore cannot be said to assess either the stress response itself or the causal mechanisms that undergird stress arousal. Nevertheless, scales such as the SRRS can be of value, especially in stress research when the researcher wishes to obtain valid and reliable assessments of the "background noise," that is, intervening or other otherwise confounding variables in psychosocial stressor research (see Everly & Sobelman, 1987).

Cognitive–Affective Correlate Scales

Whereas in the preceding section we discussed the assessment of stressor stimuli, the reader will recall from Chap. 2 the agreement among most stress researchers that in order for psychosocial life events to engender a stress response and subsequent illness, they must first be processed via cognitive–affective mechanisms. It seems theoretically viable, therefore, that one might assess the cognitive–affective domain of respondents as an indirect assessment of the human stress response (Everly & Sobelman, 1987). Derogatis (1977) has argued that such a "self-report mode of psychological measurement contains much to recommend it" (p. 2). Furthermore, Everly has argued that assessment of this domain may be the most practical, efficient, and cost-effective way of measuring the human stress response (Everly & Sobelman, 1987; Everly, Davy, Smith, Lating, & Nucifora, 2011; Nucifora, Hall, & Everly, 2011).

The World Assumption Scale (WAS; Janoff-Bulman, 1996) assesses three core assumptions, or beliefs, about life: the benevolence of the world, the inherent meaningfulness of the world, and self-worth. This self-report scale requires 5–10 min to complete and consists of 32 items scored according to a 6-point Likert scale.

Neurological Triggering Mechanisms

The assessment of the sensitivity of neurological triggering mechanisms is by no means an easy task. Aberrant evoked potentials emerging from the subcortical limbic system would be one indication of an existing hypersensitivity phenomenon within the limbic system. The accurate assessment of subcortical activity via electroencephalography (EEG) is very difficult and may be considered a gross assessment at best, however. False-negative findings are a common problem with such assessment and EEGs in general. Electrodermal responsiveness as assessed via galvanic skin response (GSR) would be another way of assessing the reactivity of neurological triggering mechanisms (Peek, 2003).

Finally, the general assessment of psychophysiological reactivity is believed to be a viable process for assessing the efferent-discharge propensity of the limbic system (Everly & Sobelman, 1987). The phenomenology of this process is based upon the theories of Lacey, Malmo, and Sternbach discussed in Chap. 3.

Measuring the Physiology of the Stress Response

It will be recalled from Chap. 2 that the stress response can be divided into three broad categories: (1) the neural axes, (2) the neuroendocrine axis, and (3) the endocrine axes. Let us briefly review several of the more common assessment technologies used to tap these phenomenological domains.

Assessment of the Neural Axes

Assessment of the neural axes of the human stress response is for the most part an attempt to capture a transitory state measurement phenomenon, as opposed to a more consistent trait. Technologies used for such assessment include (1) electrodermal techniques, (2) electromyographic techniques, and (3) cardiovascular measures.

Electrodermal Measures

The physiological basis of the electrodermal assessment of the stress response is the eccrine sweat gland. Located primarily in the soles of the feet and the palms of the hands, these sweat glands respond to psychological stimuli rather than heat and emerge on the terminal efferent ends of sympathetic neurons. Although the neurotransmitter at the sweat gland itself is Ach, as opposed to NE, the assessment of this activity provides useful insight into the activity of the sympathetic nervous system.

Electrodermal activity may be assessed via active GSR techniques or through passive techniques such as skin potentials (SP), according to Edelberg (1972). Andreassi (1980) has stated that electrodermal techniques are useful indices of somatic arousal.

Electromyographic Measurement

The physiological basis of electromyographic measurement of the stress response is the neurological innervation of the striated skeletal muscles. Electromyography, although an indirect measure of muscle "tension," is a direct measure of the action potentials originating from the neurons that innervate the muscles.

Skeletal muscles receive their neural innervation primarily as a result of alpha motoneuron presence, on the efferent limb, and secondarily as a result of gamma motoneuron activity as well. From the afferent perspective, proprioceptive neurons arising from the muscle spindles contribute to the overall electrical activity that originates from the skeletal musculature. In a relaxed state, skeletal muscle tone serves as a very useful general index of arousal (Gellhorn, 1964a; Gellhorn & Loofburrow, 1963; Jacobson, 1929, 1970; Malmo, 1975; Weil, 1974), yet in a contracted state this utility appears to disappear. Thus, when using skeletal muscles as general indices of arousal and stress responsiveness, it becomes of critical importance to teach patients to first relax those muscles (Everly, Welzant, Machado, & Miller, 1989).

There has been debate on the utility of a particular set of muscles as an index of arousal. That set of muscles is the group known as the *frontalis.* Jacobson (1970) and Shagass and Malmo (1954) first recognized that the frontalis and related facial muscles were prime targets of the stress-arousal process. Budzynski and Stoyva (1969) and Stoyva (1979) explored and refined the clinical utility of these muscles in the treatment of stress-related disorders. Similar work was undertaken by Schwartz et al. (1978), who found the corrugator muscles of similar utility in relation to depression.

It may be argued that the frontalis muscles of the forehead provide a useful site for the assessment of stress arousal. These muscles have been termed "quasi-voluntary" muscles because their autonomic-like properties manifest during emotional states. In support of such a view are the studies indicating that when simple facial expressions are mimicked, an alteration in heart rate and skin temperature can be observed, even when the subjects were simply asked to mimic the expression without any consideration for the cognitive or affective state that might be associated with it. Similarly, Rubin (1977) suggests that the frontalis muscles, in particular, may possess properties of dual innervation: skeletal alpha motoneuron and ANS innervation.

Although, clearly, the frontalis musculature is predominately striated in nature (thus receiving efferent innervation from the alpha motoneuron assemblies), Rubin (1977) has argued that the frontalis also possesses thin nonstriated layers of musculature. These nonstriated muscles apparently receive their innervation (directly or indirectly) from the SNS (Miehlke, 1973). Thus, assessment of the frontalis muscles through electromyographic procedures may well provide insight into alpha

motoneuron activity, sympathetic neural activity, as well as neuroendocrine activity (Everly & Sobelman, 1987). Although there is not total agreement on the utility of the frontalis musculature (Alexander, 1975), Stoyva (1979) provides useful guidelines for the use of that measurement variable.

Clinical biofeedback experience shows the frontalis muscles are useful in the treatment of a wide range of stress-related disorders, including essential hypertension and disorders of the GI system. Most clinicians, over the years, have reported use of the frontalis muscle in electromyographic assessment; however, the trapezius, brachioradialis, and sternocleidomastoid muscle groups have also been utilized.

In summary, most evidence suggests that the electromyographic assessment yields insight into the activity of other major muscle groups (Freedman & Papsdorf, 1976; Glaus & Kotses, 1977, 1978) as well as the generalized activity of the SNS (Arnarson & Sheffield, 1980; Budzynski, 1979; Donaldson, Donaldson, & Snelling, 2003; Field, 2009; Jacobson, 1970; Malmo, 1966; Rubin, 1977; Schwartz & Andrasik, 2003).

Cardiovascular Measurement

Cardiovascular measurement of the stress response entails the assessment of effects of the stress response upon the heart and vascular systems. Common cardiovascular measures include heart rate, peripheral blood flow, and blood pressure.

Heart rate activity as a function of the stress response is a result of direct neural innervation as well as neuroendocrine activity of epinephrine and norepinephrine. During psychosocially induced stress, epinephrine is preferentially released from the adrenal medullae. The ventricles of the heart are maximally responsive to circulating epinephrine and will respond with increased speed and force of ventricular contraction. Of course, direct sympathetic neural activation increases heart rate as well. The measurement of heart rate is most commonly achieved through the use of audio-metric or oscillometric techniques during the normal assessment of blood pressure. Occasionally, heart rate will be measured from ECG techniques via the use of passive electrodes or even through plethysmography.

Plethysmography focuses upon the volume of blood in a selected anatomical site. The most common areas for such assessment of the stress response are the fingers, toes, calves, and forearms. During the stress response, most patients will suffer a reduction of blood flow from these areas. This vasoconstrictive effect is a result of direct sympathetic activity to the arteries and arterioles, as well as of circulating norepinephrine (Hall, 2011). A decline of blood flow to these areas will also result in a reduction of skin temperature. Therefore, skin temperature is also sometimes utilized, although it is not as reliable as plethysmography. So we see that the assessment of peripheral blood flow can be accomplished via the use of plethysmography as well as skin temperature.

Finally, blood pressure is sometimes used as an *acute* index of the stress response. The assessment of blood pressure is generally achieved through the quantification of systolic and diastolic blood pressure, and may be considered highly state dependent.

Systolic blood pressure is the hemodynamic pressure exerted within the arterial system during systole (the ventricular contraction phase). Diastolic blood pressure is the hemodynamic pressure exerted within the arterial system during diastole (relaxation and filling of the ventricular chambers).

Blood pressure is a function of several variables revealed in the following equation:

$$BP = CO \times TPR$$

where

BP = blood pressure

CO = cardiac output = stroke volume × heart rate

TPR = total hemodynamic peripheral resistance

Blood pressure can be measured noninvasively through auscultation, audiometry, or oscillometry. In noninvasive paradigms, a sampled artery (usually the brachial) is compressed through the use of an inflatable rubber tube or bladder. The bladder is inflated until it totally blocks the passage of blood through the artery. Air pressure, measured in millimeters of mercury (mm Hg) is slowly released from the bladder until a sound is heard or a distension sensed. This sound and distension (called a Korotkoff sound) is indicative of blood being allowed to pass through the once blocked artery. Korotkoff sounds continue until the artery is fully opened and returned back to its natural status. The first Korotkoff sound is indicative of the systolic blood pressure. The passing of the last Korotkoff sound is indicative of the diastolic blood pressure. The technique of audiometry measures blood pressure by the use of a microphone to sense the Korotkoff sounds. Oscillometry detects the Korotkoff phenomenon via a pressure-sensitive device placed on the outside of the artery. Finally, auscultation is the sensing of the Korotkoff sound via stethoscope. Audiometric and oscillometric techniques are far more reliable than is manual auscultation.

In summary, the measurement of cardiovascular phenomena can be seen to tap both neural and neuroendocrine domains; thus, there is an overlap in phenomenology. Also, when using the cardiovascular domain to measure stress arousal, the clinician is interested only in the *acute* fluctuations, as opposed to chronic levels. This is due to the fact that stress exerts its most measurable effect upon the acute status of the cardiovascular system. A multitude of other factors enter into, and otherwise confound, the measurement process when examining cardiovascular indices such as chronic blood pressure and peripheral blood flow, for example.

Assessment of the Neuroendocrine Axis

Assessment of the neuroendocrine axis of the stress response entails measurement of the adrenal medullary catecholamines: epinephrine (adrenaline) and norepinephrine (noradrenaline).

Aggregated medullary catecholamines may be sampled from blood or urine and assayed via fluorometric methods. Reference values range for random sampling up to 18 μg/100 ml urine, for a 24-h urine sample up to 135 μg, and for timed samples, 1.4–7.3 μg/h during daylight hours (Bio-Science, 1982). For aggregated catecholamines sampled from plasma, values range from 140 to 165 pg/ml via radioenzymatic procedures (Bio-Science).

Various fluorometric (Anderson, Hovmoller, Karlsson, & Svensson, 1974; Euler & Lishajko, 1961; Jacobs et al., 1994), chromatographic (Jacobs et al.; Lake, Ziegler, & Kopin, 1976; Mason, 1972), and radioimmunoassay (Jacobs et al.; Mason) methods are available for the assessment of catecholamines. The most useful of all methods may be the high pressure liquid chromatography (HPLC) with electrochemical detection as described in Hegstrand and Eichelman (1981) and McClelland, Ross, and Patel (1985). HPLC allows multiple catecholamines to be derived from sampled plasma, urine, and saliva with superior ease and sensitivity.

Epinephrine can be sampled from urine, plasma, or saliva. When sampled from urine a typical distribution is as follows: (see Jacobs et al., 1994; Katzung, 1992):

Unchanged epinephrine	6%
Metanephrine	40%
Vanillylmandelic acid	41%
4-Hydroxy-3-methoxy-phenylglycol	7%
3, 4-Dihydroxymandelic acid	2%
Other	4%
	100%

Norepinephrine can also be sampled from urine, plasma, and saliva. Table 5.1 provides a range of epinephrine and norepinephrine values when sampled from urine.

Despite the availability of methods such as HPLC, some researchers prefer the assessment of catecholamines by indirect routes, for example, through the assessment of urinary metabolites. Metanephrines and vanillylmandelic acid (VMA) are two popular choices.

In the case of the metanephrines, one of the major deactivating substances acting upon epinephrine and norepinephrine is the enzyme catecholamine-*O*-methyltransferase (COMT). Metabolites of this deactivation process are metanephrine and normetanephine. Aggregated metanephrines range from 0.3 to 0.9 mg/day in urine. VMA levels range from 0.7 to 6.8 mg/day. VMA is the urinary metabolite of COMT and monoamine oxidase.

Assessment of the Endocrine Axes

According to Hans Selye (1976), the most direct way of measuring the stress response is via ACTH, the corticosteroids, and the catecholamines. The catecholamines have already been discussed. The most commonly used index of ACTH

Table 5.1 Value ranges for urinary epinephrine and norepinephrine

	Epinephrine	Norepinephrine
Basal levels	4–5 μg/day	28–30 μg/day
Aroused	10–15 μg/day	50–70 μg/day
Significant stress	>15 μg/day	>70 μg/day

and corticosteroid activity is the measurement of the hormone cortisol. Cortisol is secreted by the adrenal cortices, activated by ACTH, at a rate of about 25–30 mg/day and accounts for about 90% of glucocorticoid activity.

Cortisol may be sampled from either plasma or urine. Radioimmunoassay plasma levels for a normal adult may range from 5 to 20 μg/100 ml plasma (8 A.M. sample). The normal diurnal decline may result in a level of plasma cortisol at 4 P.M. about one half of the 8 A.M. level. Normal urinary-free cortisol may range from 20 to 90 μg/24 h (see Bio-Science, 1982). It has been suggested that urinary-free cortisol is the most sensitive and reliable indicator of adrenal cortical hyperfunction, followed by plasma cortisol and finally 17-hydroxycorticosteroid (17-OHCS), a cortisol metabolite (Damon, 1981). Normal values for 17-OHCS measured from urine typically range from 2.5 to 10 mg/24 h in the female to 4.5 to 12 mg/24 h in the male adult (Porter–Silber method). Slight increases in 17-OHCS are evidenced in the first trimester of pregnancy and in severe hypertension. Moderate increases can be observed in the third trimester of pregnancy and as a result of infectious disease, burns, surgery, and stress (Bio-Science). In conditions of extreme stress, urinary 17-OHCS may exceed 15 mg/24 h. Plasma assessments of 17-OHCS range from 10 to 14 μg% at 8 A.M. basal levels to 18 to 24 μg% under moderate stress, to an excess of 24 μg% in extremely stressful situations (Mason, 1972).

This section has discussed the assessment of the physiological constituents of the stress response. It should be noted that the assessment of this domain represents a challenging and potentially frustrating exercise. One major factor that confounds the assessment of most physiological variables is the fact that most physiological phenomena used to assess stress arousal are state-dependent variables that wax and wane throughout the course of a day as well as with acute situational demands. Normal diurnal fluctuations as well as acute situational variability can serve to yield false-positive or false-negative findings in the absence of meaningful baseline data. There has even been some question as to the predictive validity of acute physiological indices. Another issue that confounds the overall utility of many physiological measures is that such measures usually require special training, special equipment, or both. The difficulties associated with physiological assessment of the human stress response have been summarized by Everly and Sobelman (1987). Other issues, such as response specificity and organ reactivity, are also reviewed.

Assessment of Target-Organ Effects

Once the stress response has been activated to pathogenic proportions, there emerges another possible assessment strategy for measuring human stress—the assessment of the target-organ effects of the stress response. The assessment of target-organ effects can consist of measuring physical as well as psychological variables.

Physical Diagnosis

The assessment of the physical effects of stress would involve the use of standard diagnostic techniques common to the practice of physical medicine. The goal of such assessments is to measure the integrity of the target organ's structural and functional status. An in-depth discussion of such procedures is far beyond the scope of this volume, however.

It should be mentioned that such assessments are never clearly assessments of stress. One never really knows to what degree pathogenic stress arousal has contributed to the manifestation of target-organ pathology. For this reason, the diagnosis of stress-related target-organ disease is typically a diagnosis by exclusion; that is, one systematically excludes non-stress-related etiological factors while at the same time looking for evidence of pathogenic stress arousal through the assessment of other measurement domains as well. The stress-related diagnosis then emerges from a convergence of these data sets. There are also self-report scales that have shown to be valid and reliable indices of experienced physical illness. The Seriousness of Illness Rating Scale (SIRS; Wyler, Masuda, & Holmes, 1968; see also Rosenberg, Hayes, & Peterson, 1987) is one useful self-report tool for measuring illness and weighting its impact. The Stress Audit Questionnaire (Miller & Smith, 1982) is another. It is important to keep in mind that there is still no certainty as to the extent of the role of stress arousal in the formation of the emergent illnesses/reactions.

Finally, the Family Disruption from Illness Scale (Ide, 1996) extends the assessment of physical symptoms somewhat by assessing the degree of disruption that 53 health-related symptoms impose upon daily functioning. Most items represent physical illnesses. This scale, while more recent than the SIRS, is not as comprehensive, nor have its psychometric properties been adequately assessed.

Psychological Diagnosis

The psychological diagnosis of the stress response refers to the measurement of the "psychological" effects of the stress response. There currently exist numerous and diverse methods for the measurement of psychological states and traits. To cover this topic fully would require a volume of its own. Therefore, what we shall do in this

section is merely highlight the paper-and-pencil questionnaires that a clinician might find most useful in measuring the psychological effects of the stress response.

Minnesota Multiphasic Personality Inventory—2

The Minnesota Multiphasic Personality Inventory—2 (MMPI-2) (Butcher, Dahlstrom, Graham, Tellegren & Kraemmer, 1989) is a revision of perhaps one of the most valid and reliable inventories for the assessment of long-term stress on the personality structure of the patient. The numerous clinical and content scales of the MMPI-2 yield a wealth of valuable information. These scales sample a wide range of "abnormal" or maladjusted personality traits (a personality trait is a rather chronic and consistent pattern of thinking and behavior).

The MMPI-2 consists of ten basic clinical scales developed on the basis of actuarial data:

1. Hs: Hypochondriasis
2. D: Depression
3. Hy: Conversion Hysteria
4. Pd: Psychopathic Deviate
5. Mf: Masculinity–Femininity
6. Pa: Paranoia
7. Pt: Psychasthenia (trait anxiety)
8. Sc: Schizophrenia
9. Ma: Hypomania (manifest energy)
10. Si: Social Introversion (preference for being alone)

In addition to the highly researched clinical scales, the MMPI-2 has validity scales that give the clinician a general idea of how valid any given set of test scores is for the patient. This unique feature of the MMPI-2 increases its desirability to many clinicians.

The MMPI-2 offers a virtual wealth of information to the trained clinician; its only major drawback appears to be its length of over 560 items.

The Sixteen Personality Factor Questionnaire (16-PF)

The 16-PF (Cattell, 1972), much the same as the MMPI, assesses a wide range of personality traits. It measures 16 "functionally independent and psychologically meaningful dimensions isolated and replicated in more than 30 years of factor-analytic research on normal and clinical groups" (p. 5).

The 16-PF consists of 187 items distributed across the following scales:

- Reserved–Outgoing

- Less Intelligent–More Intelligent
- Affected by Feelings–Emotionally Stable
- Humble–Assertive
- Sober–Happy-Go-Lucky
- Conservative–Experimenting
- Group-Dependent–Self-Sufficient
- Expedient-Conscientious
- Shy-Venturesome
- Tough-minded-Tender-minded
- Trusting-Suspicious
- Practical-Imaginative
- Forthright-Astute
- Self-Assured-Apprehensive
- Undisciplined Self-Conflict-Controlled
- Relaxed-Tense

Millon Clinical Multiaxial Inventory—II

The Millon Clinical Multiaxial Inventory (MCMI) (Millon, 1983) is a 175-item self-report, true–false questionnaire. The MCMI-II, although not as widely utilized as the MMPI in the diagnosis of major psychiatric disorders, is clearly the instrument of choice when the clinician is primarily interested in personologic variables and their relationship to excessive stress. Furthermore, the MCMI-II offers valuable insight into treatment planning. Another major advantage of the MCMI-II over the MMPI and 16-PF is that it consists of only 175 items. The MCMI-II includes 22 clinical scales broken down into three broad categories; ten basic personality scales reflective of the personality theory of Theodore Millon (1981); three pathological personality syndromes; and nine major clinical psychiatric syndromes (Millon, 1983). From a psychometric perspective, the MCMI-II offers the best of both worlds: an inventory founded in a practical, clinically useful theory as well as rigorous empirical development. The clinical scales of the MCMI-II are listed below:

Schizoid; Avoidant; Antisocial; Narcissism; Passive–aggressive; Compulsive; Dependent; Histrionic; Schizotypal; Borderline; Sadistic; Paranoid; Anxiety; Somatoform; Hypomania; Dysthymia; Alcohol abuse; Drug abuse; Psychotic thinking; Psychotic depression; Psychotic delusions.

Millon Clinical Multiaxial Inventory—III

While the MCMI-II is still in use, Millon has published an even more current version of the MCMI, the Millon Clinical Multiaxial Inventory—III (MCMI-III) (Millon, Millon, Davis, & Grossman, 2009). The MCMI-III still contains 175 items

but 95 were changed or reworded from the MCMI-II. The MCMI-III has been updated into a 4th edition. The updated MCMI-III scales are as follows:

There exist *14 Personality Disorder Scales* that are coordinated with the *DSM-IV* Axis II disorders:

1—Schizoid
2A—Avoidant
2B—Depressive
3—Dependent
4—Histrionic
5—Narcissistic
6A—Antisocial
6B—Sadistic (Aggressive)
7—Compulsive
8A—Negativistic (Passive-Aggressive)
8B—Masochistic (Self-Defeating)
S—Schizotypal
C—Borderline
P—Paranoid

In addition there are ten Clinical Syndrome Scales:

A—Anxiety
H—Somatoform
N—Bipolar: Manic
D—Dysthymia
B—Alcohol Dependence
T—Drug Dependence
R—Posttraumatic Stress Disorder
SS—Thought Disorder
CC—Major Depression
PP—Delusional Disorder

There are also five scales that serve to detect response patterns that might call into question the test results:

Modifying Indices

X—Disclosure
Y—Desirability
Z—Debasement

Random Response Indicators

V—Invalidity
W—Inconsistency

Lastly, there are 42 subscales that serve to give the clinician insight into the psychological processes that undergird clinically significant elevations. These are the Grossman Facet Scales.

Common Grief Response Questionnaire

The Common Grief Response Questionnaire (CGQ) (McNeil, 1996) consists of 86 self-report items using a 7-point Likert response scale. The purpose of the CGQ is to assess the frequency of occurrence of numerous grief reactions to the death of a loved one.

Impact of Events Scale—Revised

The Impact of Events Scale—Revised (IES-R) (Weiss & Marmar, 1993) is a revision of the original IES. The IES-R consists of 22 self-report items purported to assess posttraumatic stress. Three response dimensions are tapped: intrusive ideation, avoidance and numbing, as well as hyper-arousal. The IES-R takes about 10 min to complete and is a widely used research tool.

Penn Inventory for Posttraumatic Stress Disorder (PENN)

Similar to the IES-R, the PENN (Hammarberg, 1992) is a measure of posttraumatic symptoms. It is, however, is a more global measure, consisting of 26 self-report items.

Stanford Acute Stress Reaction Questionnaire

The Stanford Acute Stress Reaction Questionnaire (SASRQ) (Cardena & Spiegel, 1993; Shalev, Peri, Canetti, & Schreiber, 1996) consists of 30 self-report items that assess acute stress disorder. The scale takes 5–10 min to complete and appears to be useful in predicting PTSD.

Taylor Manifest Anxiety Scale

The Taylor Manifest Anxiety Scale (TAS) (Taylor, 1953), unlike the inventories previously described, measures only one trait—anxiety. Its 50 items are derived from the MMPI. The TAS measures how generally anxious the patient is and has little ability to reflect situational fluctuations in anxiety.

State–Trait Anxiety Inventory

The State–Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch, & Luchene, 1970) is a highly unique inventory in that it is two scales in one. The first 20 items measure state anxiety (a psychological state is an acute, usually situationally dependent condition of psychological functioning). The second 20 items measure trait anxiety. This is the same basic phenomenon as that measured by the TAS. The STAI can be administered in full form (40 items) or be used to measure only state or trait anxiety.

Affect Adjective Checklist

Another unusual measuring device is the Affect Adjective Checklist (AACL) (Zuckerman, 1960). Like the STAI, the AACL can be used to measure a psychological state or trait by using the same items (21 adjectives) and merely changing the instructions. The client may use the checklist of adjectives to describe how he or she feels in general or under a specific set of conditions—"now" for instance. Zuckerman and Lubin (1965) later expanded the AACL by adding specific items to assess hostility and depression. The more recent (1985) scale is called the Multiple Affect Adjective Checklist (MAACL).

Subjective Stress Scale

The Subjective Stress Scale (SSS) (Berkun, 1962) is designed to measure situational (state) effects of stress on the individual. The scale consists of 14 descriptors that the patient can use to identify his or her subjective reactions during a stressful situation. Each of these descriptors comes with an empirically derived numerical weight, which the clinician then uses to generate a subjective stress score.

Profile of Mood States

The Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971) is a factor-analytically derived self-report inventory that measures six identifiable mood or affective states (p. 5):

> Tension–Anxiety Depression–Dejection Anger–Hostility Vigor–Activity Fatigue–Inertia Confusion–Bewilderment

The POMS consists of 65 adjectives, each followed by a 5-point rating scale that the patient uses to indicate the subjective presence of that condition. The instructions ask the patient to use the 65 adjectives to indicate "How you have been feeling

during the past week including today." Other time states have been used, for example: "right now," "today," and for "the past three minutes."

The POMS offers a broader range of state measures for the subjective assessment of stress when compared with the STAI, the AACL-MAACL, and the SSS.

Connor Davidson Resilience Measure

The Connor Davidson Resilience Measure (CD-RISC) (Connor & Davidson, 2003) is a rating scale that assesses resilience. It is composed of 25 items that are rated on a scale of 1–4. Higher scores indicate greater resilience.

Inventory of Complicated Grief Scale

The Inventory of Complicated Grief Scale (Prigerson et al., 1995) is a scale designed to assess symptoms of complicated grief. This scale is designed to assess the complicated bereavement symptoms of depression and anxiety that predict long-term functional impairments. The scale consists of 22 items corresponding to the cognitive, emotional, and behavioral states that are associated with complicated grief. Each of the items is rated on a scale of 1–4, depending on how frequently they experience each of the states.

Generalized Anxiety Disorder Questionnaire

The Generalized Anxiety Disorder Questionnaire (GADQ-IV) (Newman et al., 2002) is a self-report questionnaire that has items that are reflective of the DSM-IV's criteria for Generalized Anxiety Disorder. Most of the items are dichotomous and ask the respondent to give yes/no answers, one item is left open ended and two items utilize a rating scale from one (meaning no distress) to eight (meaning severe distress) to assess distress and impairment.

Beck Anxiety Inventory

The Beck Anxiety Inventory (BAI) (Beck & Steer, 1990) is a widely used 21 item self-report symptom inventory that is used to assess anxiety in adolescents and adults. The items reflect symptoms of anxiety and the respondents are asked to rate them on a scale of 0 (not experiencing symptoms at all) to 3 (severe symptoms). This can be used as a screening or diagnostic tool for Generalized Anxiety Disorder. It can also be used to assess treatment progress.

Symptom Checklist-90 Revised

The Symptoms Checklist 90 (SCL-90-R) (Derogatis, 1994) is a self-report measure assessing symptoms of psychopathology on nine different dimensions and three global indices. The dimensions include anxiety, obsessive-compulsive disorder, phobic anxiety, and paranoid ideation. The SCL-90 consists of 90 items and takes approximately 12–15 min to administer. There is also a shorter form called the Brief Symptom Inventory (BSI; Derogatis, 1993) that is composed of 53 items and provides scores on the same dimensions and global indices as the SCL-90. These tools can both be used to determine treatment progress or treatment outcome.

Screen for Posttraumatic Stress Symptoms

The Screen for Posttraumatic Stress Symptoms (SPTSS) (Carlson, 2001) is a 17-item self-report inventory that assesses the symptoms of PTSD listed in the DSM-IV. Respondents rate each of the items on an 11-point scale indicating frequency of symptoms from 0 (never) to 10 (always). This is recommended for screening of PTSD in both research and clinical settings.

Inventory of Psychosocial Functioning

The Inventory of Psychosocial Functioning is an 87-item self-report measure that assesses functional impairment experienced by active-duty service members and veterans over the past 30 days on a 7-point scale ranging from 1 (“never”) to 7 (“always”). The IPF provides a total score for each of seven subscales (romantic relationships with a spouse or partner, family relationships, work, friendships and socializing, parenting, education, and day-today functioning), and an overall functional impairment score is computed by calculating the mean of the scores for each completed subscale. In a sample of veterans meeting diagnostic criteria for PTSD, their overall mean score on the IPF was 3.86 (SD = 1.06) (Castro, Hayes, & Keane, 2011).

The Assessment of Coping

The Millon Behavioral Health Inventory (MBHI) (Millon, Green, & Meagher, 1982) assesses the patient’s characterological coping style. The Hassles Scale measures an indirect form of coping within its “uplifts” subscale. Everly created a simple coping inventory for use in conjunction with the National Health Fair (Everly, 1979a; Girdano & Everly, 1986). This checklist can be found in Appendix H.

Finally, perhaps the most popular of the coping indices is the Ways of Coping Checklist developed by Lazarus and Folkman (1984). This 67-item checklist that

assesses an individual's preference for various styles of coping patterns (e.g., defensive coping, information seeking, problem solving) enjoys a considerable empirical foundation and can be found in their 1984 textbook on stress, appraisal, and coping.

In the broadest sense, coping may be viewed as any effort to reduce or mitigate the aversive effects of stress. These efforts may be psychological or behavioral. The scales mentioned sample both domains.

Law of Initial Values

A final point should be made regarding the role of individual differences in the process of measurement. No two patients are exactly alike in their manifestations of the stress response. When measuring psychophysiological reactivity, or any physiological index, the clinician must understand that the patient's baseline level of functioning on any physiological variable affects any subsequent degree of activity or reactivity in that same physiological parameter. This is Wilder's Law of Initial Values (Wilder, 1950). In order to compare an individual's stress reactivity (assuming variant baselines), a statistical correction must be made in order to assure that the correlation between baseline activity and stressful reactivity is equal to zero. Such a correction must be made in order to compare groups as well. Benjamin (1963) has written a very useful paper that addresses the necessary statistical corrections that must be made. She concludes that a covariance model must be adopted in order to correct for the law of initial values, though specific calculations will differ when comparing groups or individuals.[1] It must be remembered that the Law of Initial Values will affect not only the measurement of stress arousal but also stress reduction.

Summary

In this chapter we have described briefly some of the most commonly used methods of measuring the effects of the stress response. The methods described have included physiological and psychological criteria.

[1] One useful formula for correcting for the Law of Initial Values when comparing individuals is the Autonomic Lability Score (ALS; Lacey & Lacey, 1962). The ALS, a form of covariance and therefore consistent with Benjamin's recommendation, is expressed as

$$\mathrm{ALS} = 50 + 10\left[\frac{y_z - x_{zr_{xy}}}{\left(1 - r^{2xy^{0.5}}\right)}\right]$$

where X_z = client's standardized prestressor autonomic level, Y_z = client's standardized poststressor autonomic level, and r_{xy} = correlation for sample between pre- and poststressor levels.

The most important question surrounding the measurement of the stress response is "How do you select the most appropriate measurement criterion?" The answer to this question is in no way clear-cut. Generally speaking, to begin with you should consider the state versus trait measurement criterion issue. Basically, state criteria should be used to measure immediate and/or short-lived phenomena. Trait criteria should be used to measure phenomena that take a longer term to manifest themselves and/or have greater stability and duration. The psychological criteria discussed in this chapter are fairly straightforward as to their state or trait nature. The physiological criteria are somewhat less clear. Some physiological criteria possess both state and trait characteristics. Furthermore, normal values for blood and urinary stress indicators may vary somewhat from lab to lab. Therefore, the clinician should familiarize him- or herself with the lab's standard values. Before using physiological measurement criteria in the assessment of the stress response, the reader who has no background in physiology would benefit from consulting any useful physiology or psychophysiology text (see, e.g., Everly & Sobelman, 1987; Greenfield & Sternbach, 1972; Levi, 1975; Selye, 1976; Stern, Ray, & Davis, 1980). Finally, because no two patients are alike in their response to stressors, the clinician might consider measuring multiple and diverse response mechanisms (or stress axes) in order to increase the sensitivity of any given assessment procedure designed to measure the stress response (see Fig. 5.1).

Having provided these closing points, let us review the major issues discussed within this chapter:

1. It has been argued that the single most important aspect of empirical investigation is the process of the *measurement* of relevant variables. This is true of investigations into the nature of human stress as well.
2. The Social Readjustment Rating Scale (Holmes & Rahe, 1967), the Life Experiences Survey (Sarason et al., 1978), and the Hassles Scale (Kanner et al., 1981) are all self-report inventories that assess the patient's exposure to critical "life events." Collectively, these scales do not measure stress; rather, they assess the patient's exposure to stressors. Stressor scales are correlated with stress arousal because the physiology of adaptation to novel or challenging stimuli is also the physiology of the stress response.
3. The Derogatis Stress Scale (Derogatis, 1980) and the Millon Behavioral Health Inventory (Millon, et al., 1982) represent scales designed to assess the patient's cognitive–affective status. The stress response is thus assessed indirectly through the measurement of cognitive–affective states known to be highly associated with stress arousal. It has been argued that such assessments may well be the most efficient, practical, and cost-effective way of assessing stress arousal. All of these scales also include symptom indices.
4. Albeit an important clinical phenomenon, the assessment of propensities for limbic efferent discharge (limbic hypersensitivity phenomenon) is extremely difficult. Subcortical electroencephalography is a crude measure at best. Electrodermal and general psychophysiological reactivity may be the best options currently available for the assessment of neurological triggering mechanisms of the human stress response.

5. Numerous measurement options exist for the assessment of the physiological stress response itself (if deemed appropriate).
   (a) The neural stress axes may be assessed via electrodermal measures, electromyographic measures, as well as cardiovascular measures (heart rate, peripheral blood flow, blood pressures).
   (b) The neuroendocrine stress axis can be measured via the assessment adrenal medullary catecholamines.
   (c) The assessment of the endocrine stress axes is most commonly conducted via the assessment of cortisol.
6. The assessment of target-organ effects of pathogenic stress arousal can be conducted via standard physical medicine examination or the use of self-report inventories such as the Seriousness of Illness Rating Scale (Wyler et al., 1968) to measure *physical effects. Psychological effects* may be assessed via self-report scales such as the MMPI, the MCMI, the 16-PF, IES-R, PENN, SASRQ, CGO, the TAS, the STAI, the AACL, the SSS, and the POMS.
7. Coping is an important potential mediating variable. It may be assessed via the MBHI; the Hassles Scale, a coping scale developed by Everly (1979a) for the US Public Health Service; or the Ways of Coping Checklist (Lazarus & Folkman, 1984).

References

Alexander, A. B. (1975). An experimental test of the assumptions relating to the use of EMG: biofeedback as a general relaxation training technique. *Psychophysiology, 12*, 656–662.

Anderson, B., Hovmoller, S., Karlsson, C., & Svensson, S. (1974). Analysis of urinary catecholarnines. *Clinica Chimica Acta, 51*, 13–28.

Andreassi, J. (1980). Psychophysiology. New York: Oxford University Press.

Arnarson, E., & Sheffield, B. (1980, March). *The generalization of the effects of EMG and temperature biofeedback*. Paper presented at the Annual Meeting of the Biofeedback Society of America, Colorado Springs, CO.

Beck, A. T., & Steer, R. A. (1990). Manual for the Beck anxiety inventory. San Antonio, TX: Psychological Corporation.

Benjamin, L. (1963). Statistical treatment of the law of the initial values in autonomic research. *Psychosomatic Medicine, 25*, 556–566.

Berkun, M. (1962). Experimental studies of psychological stress in man. *Psychological Monographs, 76* (Whole No. 534).

Bio-Science. (1982). *Bio-Science handbook*. Van Nuys, CA: Author.

Budzynski, T. (1979, November). *Biofeedback and stress management.* Paper presented at the Johns Hopkins Conference on Clinical Biofeedback, Baltimore, MD.

Budzynski, T., & Stoyva, J. (1969). An instrument for producing deep muscle relaxation by means of analog information feedback. *Journal of Applied Behavioral Analysis, 2*, 231–237.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegren, A., & Kaemmer, B. (1989). *Manual for the restandardized Minnesota Multiphasic Personality Inventory: MMPI-2. An administrative and interpretive guide*. Minneapolis: University of Minnesota Press.

Cardena, E., & Spiegel, D. (1993). Dissociative reactions to the Bay Area Earthquake. *The American Journal of Psychiatry, 150*, 474–478.

Carlson, E. B. (2001). Psychometric study of a brief screen for PTSD: Assessing the impact of multiple traumatic events. *Assessment, 8*(4), 431–441.
Castro, F., Hayes, J. P., & Keane, T. M. (2011). Issues in assessment of PTSD within the military culture. In B. A. Moore & W. E. Penk (Eds.), *Treating PTSD in military personnel. A clinical handbook* (pp. 23–41). New York, NY: Guilford.
Cattell, R. B. (1972). *The sixteen personality factor*. Champaign, IL: Institute for Personality and Ability Testing.
Cattell, R. B., & Scheier, I. (1961). *The meaning and measurement of neuroticism and anxiety*. New York, NY: Ronald Press.
Cohen, J. (1984). The benefits of meta-analysis. In J. Williams & R. Spitzer (Eds.), *Psychotherapy research* (pp. 332–339). New York, NY: Guilford.
Connor, K. M., & Davidson, J. R. T. (2003). Development of a new resilience scale: The Connor-Davidson Resilience Scale (DC-RISC). *Depression and Anxiety, 18*(2), 76–82.
Damon Corporation. (1981). *Evaluation of adrenocortical function*. Needham Heights, MA: Author.
Derogatis, L. (1977). *The SCL-90-R: Administration, scoring and procedures manual 1*. Baltimore, MA: Clinical Psychometric Research.
Derogatis, L. (1980). *The Derogatis stress profile*. Baltimore, MA: Clinical Psychometric Research.
Derogatis, L. R. (1993). *Brief Symptom Inventory (BSI): Administration, scoring, and procedures manual*. Minneapolis, MN: National Computer Systems.
Derogatis, L. R. (1994). *SCL-90-R: Symptom Checklist-90-R: Administration, Scoring & Procedures Manual* (3rd ed.). Minneapolis, MN: National Computer Systems.
Donaldson, S., Donaldson, M., & Snelling, L. (2003). SEMG Evaluations: An overview. *Applied Psychophysiology and Biofeedback, 28*(2), 121–127.
Edelberg, R. (1972). Electrical activity of the skin. In N. Greenfield & R. Sternbach (Eds.), Handbook of Psychophysiology (pp. 367–418). New York: Holt, Rinehart & Winston.
Euler, U. S. V., & Lishajko, F. (1961). Improved techniques for the fluorimetric estimation of catecholamines. *Acta Physiologica Scandinavia, 51*, 348–355.
Everly, G. S., Jr. (1979a). *Strategies for coping with stress: An assessment scale*. Washington, DC: Office of Health Promotion, Department of Health and Human Services.
Everly, G. S., Jr., Davy, J. A., Smith, K. J., Lating, J. M., & Nucifora, F. C., Jr. (2011). A defining aspect of human resilience in the workplace: A structural modeling approach. *Disaster Medicine and Public Health Preparedness, 5*(2), 98–105.
Everly, G. S., Jr., & Sobelman, S. H. (1987). *The assessment of the human stress response: Neurological, biochemical, and psychological foundations*. New York, NY: American Management Systems Press.
Everly, G. S., Jr., Welzant, V., Machado, P. & Miller, K. (1989). *The correlation between frontalis muscle tension and sympathetic nervous system activity.* Unpublished research report.
Field, T. (2009). *Complementary and alternative therapies research*. Washington, DC: American Psychological Association.
Fiske, D. W. (1983). The meta-analysis revolution in outcome research. *Journal of Consulting and Clinical Psychology, 51*, 65–70.
Freedman, R., & Papsdorf, J. (1976). *Generalization of frontal EMG biofeedback training to other muscles*. Paper presented to the 7th Annual Meeting of the Biofeedback Society, Colorado Springs.
Gellhorn, E. (1964a). Motion and emotion. *Psychological Review, 71*, 457–472.
Gellhorn, E., & Loofburrow, G. (1963). *Emotions and emotional disorders*. New York, NY: Harper & Row.
Girdano, D. A., & Everly, G. S., Jr. (1986). *Controlling stress and tension* (2nd ed.). Englewood Cliffs, NJ: Prentice-Hall.
Glaus, K., & Kotses, H. (1977). *Generalization of conditioned frontalis tension.* Paper presented to the 8th Annual Meeting of the Biofeedback Society. Orlando, FL.
Glaus, K., & Kotses, H. (1978). *Generalization of conditioned frontalis tension: a closer look.* Paper presented to the 9th Annual Meeting of the Biofeedback Society. Albuquerque, NM.

Greenfield, N., & Sternbach, R. (1972). *Handbook of psychophysiology*. New York, NY: Holt, Rinehart & Winston.

Hall, J. E. (2011). *Guyton and Hall Textbook of Medical Physiology* (12th ed.). Philadelphia, PA: Saunders Elsevier.

Hammarberg, M. (1992). Penn Inventory for posttraumatic stress disorder: Psychometric properties. *Psychological Assessment, 4*, 67–76.

Hegstrand, L. R., & Eichelman, B. (1981). Determination of rat brain tissue catecholamines using liquid chromatography with electrochemical detection. *Journal of Chromatography, 22*, 107–111.

Holmes, T. H., & Rahe, R. (1967). The social readjustment rating scale. *Journal of Psychosomatic Research, 11*, 213–218.

Ide, B. A. (1996). Psychometric review of family disruption from Illness Scale. In B. H. Stamm (Ed.), *Measurement of stress trauma, and adaptation*. Lutherville, MD: Sidran Press.

Jacobs, D. S., Demott, W., Finly, P., Horvat, R., Kasten, B., & Tilzer, L. (1994). *Laboratory test handbook* (3rd ed.). Cleveland: Lexi-Comp.

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

Jacobson, E. (1970). *Modern treatment of tense patients*. Springfield, IL: Charles C. Thomas.

Janoff-Bulman, R. (1996). Psychometric review of World Assumption Scale. In B. H. Stamm (Ed.), *Measurement of stress, trauma, and adaptation*. Lutherville, MD: Sidran Press.

Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *Journal of Behavioral Medicine, 4*, 1–39.

Katzung, B. G. (1992). *Basic and clinical Pharmacology* (5th ed.). Norwalk, CT: Lange.

Lacey, J., & Lacey, B. (1962). The law of initial value in the longitudinal study of autonomic constitution. *Annals of the New York Academy of Sciences, 98*, 1257–1290.

Lake, C. R., Ziegler, M., & Kopin, L. (1976). Use of plasma norepinephrine for evaluation of sympathetic neuronal function in man. *Life Sciences, 18*, 1315–1326.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Levi, L. (1975). *Emotions: Their parameters and measurement*. New York: Raven Press.

Malmo, R. B. (1966). Studies of anxiety. In C. Spielberger (Ed.), *Anxiety and behavior* (pp. 157–177). New York: Academic Press.

Malmo, R. B. (1975). *On emotions, needs, and our archaic brain*. New York: Holt, Rinehart & Winston.

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.

McClelland, D. C., Ross, G., & Patel, V. (1985). The effect of an academic examination on salivary norepinephrine and immunoglobulin levels. *Journal of Human Stress, 11*, 52–59.

McNair, D., Lorr, M., & Droppleman, L. (1971). *Profile of mood states manual*. San Diego: Educational and Industrial Testing Service.

McNeil, F. (1996). Psychometric review of Common Grief Response Questionnaire. In B. H. Stamm (Ed.). *Measurement of stress, trauma, and adaptaion*. Lutherville, Maryland; Sidran Press.

Miehlke, A. (1973). *Surgery of the facial nerve*. Philadelphia: Saunders.

Miller, L., & Smith, A. (1982). *The Stress Audit Questionnaire*. Boston: Neuromedical Consultants.

Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.

Millon, T. (1983). *Millon Clinical Multiaxial Inventory manual* (3rd ed.). Minneapolis: National Computer Systems.

Millon, T., Green, C. J., & Meagher, R. B. (1982). *Millon Behavioral Health Inventory manual* (3rd ed.). Minneapolis: National Computer Systems.

Millon, T., Millon, C., Davis, R., & Grossman, S. (2009). *Millon Clinical Multiaxial Inventory–III manual* (4th ed.). Minneapolis, MN: National Computer Systems.

Monroe, S. (1983). Major and minor life events as predictors of psychological distress. *Journal of Behavioral Medicine, 6*, 189–206.

Newman, M. G., Zuellig, A. R., Kachin, K. E., Constantino, M. J., Przeworski, A., Erickson, T., & Cashman-McGrath, L. (2002). Preliminary reliability and validity of the Generalized Anxiety Disorder Questionnaire-IV: A revised self-report diagnostic measure of generalized anxiety disorder. *Behavior Therapy, 33*(2), 215–233.

Nucifora, F., Hall, R., & Everly, G. S., Jr., (2011). Reexamining the role of the traumatic stressor and the trajectory of posttraumatic distress in thewake of disaster. *Disaster Medicine and Public Health Preparednes,s* . 5 *(supp.2):* S172–175.

Prigerson, H. G., Maciejewski, P. K., Reynolds III, C. F., Bierhals, A. J., Newsom, J. T., Fasiczka, A., Miller, M. (1995). Inventory of complicated grief: A scale to measure maladaptive symptoms of loss. *Psychiatric Research, 59*(1–2), 65–79.

Peek, C. J. (2003). A primer of biofeedback instrumentation. In M. S. Schwartz, F. Andrasik (Eds.), *Biofeedback: A Practitioner's Guide* (pp.43-87). New York: Guilford.

Rosenberg, S., Hayes, J., & Peterson, R. (1987). Revising the Seriousness of Illness Rating Scale. *International Journal of Psychiatry in Medicine, 17*, 85–92.

Rubin, L. R. (1977). *Reanimation of the paralyzed face*. St. Louis, MD: Mosby.

Sarason, I., Johnson, J., & Siegel, J. (1978). Assessing the impact of life changes. *Journal of Consulting and Clinical Psychology, 46*, 932–946.

Schwartz, M. S., & Andrasik, F. (2003). *Biofeedback: A practitioner's guide* (3rd ed.). New York: Guilford.

Schwartz, G., Fair, P., Mandel, M., Salt, P., Mieske, M., & Klerman, G. (1978). Facial electromyography in the assessment of improvement in depression. *Psychosomatic Medicine, 40*, 355–360.

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

Shagass, C., & Malmo, R. (1954). Psychodynamic themes and localized muscular tension during psychotherapy. *Psychosomatic Medicine, 16*, 295–313.

Shalev, A., Peri, T., Canetti, L., & Schreiber, S. (1996). Predictors of PTSD in injured survivors of trauma: A prospective study. *American Journal of Psychiatry, 153*, 219–225.

Smith, M. L., Glass, G. V., & Miller, T. I. (1980). *The benefits of psychotherapy*. Baltimore: Johns Hopkins University Press.

Spielberger, C. D., Gorsuch, R., & Luchene, R. (1970). *The STAI manual*. Palo Alto, CA: Consulting Psychologists Press.

Stamm, B. H. (1996). *Measurement of stress, trauma, and adaptation*. Lutherville, MD: Sidran Press.

Stern, R., Ray, W., & Davis, C. (1980). *Psychophysiological recording*. New York: Oxford University Press.

Stoyva, J. M. (1979). Musculoskeletal and stress-related disorders. In O. Pomerleau & J. Brady (Eds.), *Behavioral medicine* (pp. 155–176). Baltimore: Williams & Wilkins.

Taylor, J. (1953). A scale for manifest anxiety. *Journal of Abnormal and Social Psychology, 45*, 285–290.

UCLA Laboratory for Stress Assessment and Research. (n.d.). Stress and adversity Invetory (STRAIN). Retreived from: http://www.uclastresslab.org/products/strain–stress-and-adversity-inventory

Weil, J. (1974). *A neurophysiological model of emotional and intentional behavior*. Springfield, IL: Charles C. Thomas.

Weiss, D., & Marmar, C. (October, 1993). *The impact of debriefings on emergency services personnel workers: Effects of site and service*. San Antonio, Texas: International Society for Traumatic Stress Studies Annual Meeting.

Wilder, J. (1950). The law of initial values. *Psychosomatic Medicine, 12*, 392–401.

Wolfe, J., & Kimerling, R. (1998). Assessment of PTSD and gender. In J. Wilson & T. M. Keene (Eds.), *Assessing psychological trauma and PTSD*. New York: Plenum.

Wyler, R. A., Masuda, M., & Holmes, T. H. (1968). Seriousness of illness rating scale. *Journal of Psychosomatic Research, 11*, 363–374.

Zuckerman, M. (1960). The development of an affect adjective checklist for the measurement of anxiety. *Journal of Consulting Psychology, 24*, 457–462.

Zuckerman, M., & Lubin, B. (1965). *Manual for the Multiple Affect Adjective Checklist*. San Diego: Educational and Industrial Testing Service.