6

Psychophysiological Studies of Emotion and Psychopathology

Serena M. King
Jeanne L. Tsai
Yulia Chentsova-Dutton

Several scholars have written extensively about the general utility of psychophysiological methods in the assessment of psychopathology (Cacioppo & Tassinary, 1990; Iacono, 1991). Physiological techniques allow us to observe changes in the motivational, emotional, or cognitive states of individuals, particularly changes that self-report measures alone may not adequately capture because they occur outside of individuals' awareness or because they may be difficult for the individual to describe. Physiological measurements may also disambiguate equivocal behavioral observations, self-reports, and/or other forms of information about an individual. In clinical settings, physiological measurements may not only assess the physical symptoms associated with a disorder, they may also differentiate among competing psychiatric diagnoses, identify individuals at risk for a particular clinical disorder, and be used as an index of clinical improvement. In understanding psychopathology across cultures, physiological measurements may elucidate whether each clinical disorder has a universal physiological core. In this chapter, we focus on one way psychophysiological methods are increasingly being used in the study of psychopathology: to understand the emotional underpinnings of various clinical disorders.

Although a broad array of definitions of emotion exists, most emotion theorists and researchers agree on two things: (1) that emotions arise in response to an event that bears some significance to the individual, and (2) that emotions involve a subjective experience, a behavioral reaction, and physiological change (Levenson, 1994). Scientists, philosophers, and poets have regarded emotion as an important part of human experience. Thus, it should not be surprising that major forms of psychological and social dysfunction involve deficits in emotional experience and emotional regulation. For some disorders, it is fairly clear that emotional functioning is impaired. For example, depression and the other mood disorders are typically characterized by alterations in the intensity and frequency of emotional episodes, and anxiety disorders are marked by intense fear and worry. However, there are other disorders where the impairments in emotion may be equally significant, although less apparent. For example, schizophrenics have been described as having blunted or flat affect, and individuals with psychopathy often experience little remorse, guilt, or shame for their actions.

In the following sections, we will review the extant literature on the impact of various disorders (e.g., schizophrenia, psychopathy, depres-

sion, and anxiety) on the physiological aspects of emotional functioning. We will then discuss the assessment and treatment implications of these findings and directions for future research. As a way of preparing readers for the subsequent sections on each of the clinical disorders, we first describe common methodological approaches to the study of the physiological concomitants of emotion.

COMMON METHODS IN PSYCHOPHYSIOLOGICAL STUDIES OF EMOTION

In studies of emotion, psychophysiological measures have typically sampled the autonomic nervous system (e.g., heart rate, skin conductance, pulse transmission times to the finger and ear, respiration, finger temperature), and/or the central nervous system (e.g., evoked potentials as measured by the electroencephalogram [EEG], localized brain activity as measured by functional magnetic resonance imaging [fMRI]). Although the nervous system was identified as relevant to emotional responding by American psychologists as early as the 1800s (James, 1884), connections between the mind and body were proposed by Hippocrates and Galen in the first and second centuries (Funder, 2001, p. 203). The psychometric properties of most psychophysiological measures have been described elsewhere (Strube, 1990), as have standard procedures for using these physiological measures (Donchin et al., 1977; Fowles et al., 1981; Fridlund & Cacioppo, 1986; Jennings, Berg, Hutcheson, Obrist, & Turpin, 1981); therefore, these issues will not be discussed here.

A major issue that confronts scientists who employ physiological measures in their studies is one of inference. Because physiological systems are multiply influenced, scientists must design their experiments so that a given physiological change can be attributed to task-relevant changes in the emotional state of the individual. For example, increases in heart rate may be due to the feelings of anger induced by a film (as intended by the researcher), feelings of anxiety about participating in an experiment, or nonemotional behaviors such as taking deep breaths, altering muscle tension in a body part, kicking one's feet, or bouncing in one's chair (presumably not intended by the researcher). To address this concern, researchers will include multiple measures of physiological recording (e.g., electromyographic activity [muscle movement], autonomic measures, and electrocortical measures) as well as nonphysiological measures of emotional responding (e.g., reports of subjective emotional experience and expressive behavior) within a single study, with the hope that convergence and divergence across these measures will allow the accurate interpretation of physiological responses.

The studies that we will discuss in this chapter typically compare study participants' responses during an emotional task with their responses during baseline or another control condition. The emotional tasks include exposure to noxious tones, looking at emotion evoking slides or watching such films, and reliving past emotional episodes in their lives. For example, one common method of studying emotional response is the startle paradigm. Theoretically, the startle paradigm taps a reflex common to humans and animals, evoked by the sudden presentation of an intense, usually noxious, stimulus. Experimentally, the basic startle paradigm entails showing individuals slides of varying emotional valence (negative or positive) and arousal (low or high) (Lang, 1995) and collecting measures of the electromyographic (EMG) activity of the muscle just under the eye (orbicularlis oculi) (Vrana, Spence, & Lang, 1988). While viewing these slides, individuals are exposed to a loud (around 100 dB) burst of white noise (Vrana et al., 1988). Negatively valenced stimuli (e.g., a picture of a gun) produce a potentiated startle response (as measured by EMG activity), whereas pleasantly valenced stimuli (e.g., an erotic picture) produce an attenuated startle response (as shown in Figure 6.1). This pattern has been consistently found across a number of studies with nonclinical populations (Cuthbert, Schupp, Bradley, McManis & Lang, 1998; Patrick, 1994; Vrana et al., 1988).

Other paradigms involve showing participants film clips that are intended to elicit specific emotions (e.g., sadness, amusement) or

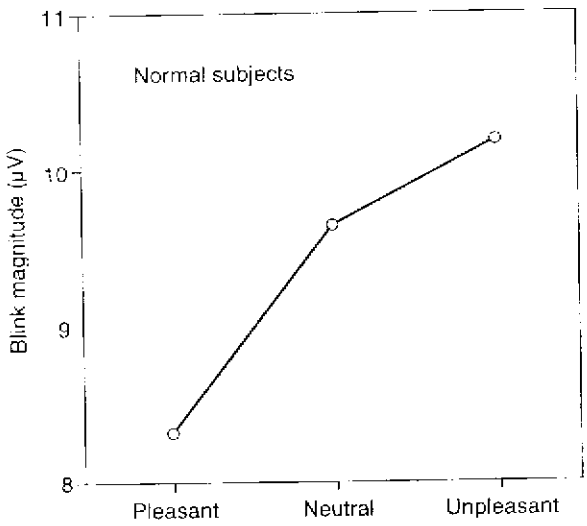

FIGURE 6.1 Mean magnitude of eyeblink startle reaction for normal college students during pleasant, neutral, and unpleasant slides. Startle blink magnitude during unpleasant slides is greater than during neutral slides (startle potentiation). Startle blink magnitude during pleasant slides is smaller than during neutral slides (startle attenuation). From "Emotion and Psychopathy: Startling New Insights," by C. J. Patrick, 1994, *Psychophysiology*, 31, p. 322. Reprinted with the permission of Cambridge University Press.

having them relive emotional episodes in their lives. While participants watch the film clips, their physiological responses are continuously monitored. Changes in mean levels of responding from baseline are then compared across groups (e.g., depressed and nondepressed). Using these paradigms, we have learned much about different clinical disorders and their impact on emotional functioning. We discuss these findings next.

REVEALING THE EMOTIONAL UNDERPINNINGS OF PSYCHOPATHOLOGY

Psychophysiological studies of clinical disorders have contributed enormously to scientists' efforts to elucidate the types of emotional dysfunction that accompany or underlie various clinical disorders. We review the existing literature for five major categories: schizophrenia, psychopathy, alcohol intoxication, depression, and anxiety. This review is not intended to be comprehensive, but to provide readers with an understanding of the general emotional patterns that appear to be related to specific clinical disorders.

Schizophrenia: Disjunction of Response

Flat affect, or the lack of overt emotional expressivity (Neale, Blanchard, Kerr, Kring, & Smith, 1998), has for decades been considered an important aspect of schizophrenia. Only recent empirical investigations, however, have moved beyond clinician's ratings and have used other methods to study more comprehensively whether this characteristic reflects a deeper emotional impairment associated with schizophrenia.

Do schizophrenics' emotional experiences and physiological responses match their lack of emotional expression? To answer this question, Ann Kring and her colleagues (Kring, Kerr, Smith, & Neale, 1993) conducted several studies in which they showed schizophrenics and nonschizophrenics (matched by age and education) film clips each intended to elicit a specific emotional state (i.e., neutral, happy, sad, and fear/disgust). During the film clips, participants' skin conductance responses and heart rate were measured. After each film clip, participants completed a self-report measure of their emotional experience. The researchers coded the facial emotional behavior of the participants during the film clip to examine whether the schizophrenic patients did indeed demonstrate emotional blunting. Across their studies, Kring and her colleagues found that while schizophrenics show *less* facial expression during both positive and negative film clips than comparison subjects, they report experiencing *more* emotion and actually demonstrate *more* skin conductance activity than their normal counterparts. The heart rate data were mixed, and therefore were more difficult to interpret. A recent study by Curtis and colleagues (1999) found that schizophrenia patients with

and without flat affect do not differ from controls in their startle responding to positive and negative slides or in their ratings of the slides, lending yet further support for the notion that flat affect belies the emotional experience of schizophrenics.

In their follow-up studies, Kring and colleagues have found that the disjunction among physiology, self-report, and expressive behavior cannot be attributed to deficits in social skills (Salem & Kring, 1999). Kring and colleagues (Neale et al., 1998) have proposed two possible explanations for the disjunction of emotional response that appears to accompany schizophrenia. First, it is possible that schizophrenics are inhibiting or suppressing their emotional expressions. For instance, studies by Gross and his colleagues (e.g., Gross & Levenson, 1993) found that suppression of emotional expression results in increased physiological activity but does not alter reported subjective experience—a pattern of responding that is similar to that of schizophrenics. Second, it is possible that schizophrenics are processing the emotional stimuli to the degree that they know what emotion they should feel, but not to a degree that alters their expressive behavior or physiology. In this case, increases in skin conductance activity may be a response to the situation rather than an emotion-specific response. The findings of Curtis and co-workers (1999) argue against this latter possibility; however, only future studies of the impact of schizophrenia on emotional functioning will determine which of these possible explanations for the disjunction of response holds true.

Psychopathy: Disruptions in Inhibition and Reward

Psychopaths often appear to be unusually charming and pleasant. Researchers consistently describe psychopaths as being unable to experience emotion strongly (Lykken, 1957), as being emotionally detached from their social environments, and as lacking empathy for others (Cleckley, 1982). Given the questionable nature of psychopaths' self-reports, psychophysiological studies of emotion provide objective evidence for emotional dysfunction in psychopathy.

To understand the specific nature of emotional dysfunction in psychopathy, many researchers have drawn on Gray's (1975) notion that two opposing systems—the behavioral activation system and the behavioral inhibition system—guide behavior. The behavioral activation system (BAS) is responsible for approach and reward-seeking behaviors, such as engaging in sexual activity, whereas the behavioral inhibition system (BIS) is responsible for withdrawal (aversive) behaviors, such as fleeing from potential predators. According to Gray, the BIS suppresses approach behaviors that may have negative consequences for the organism. For example, the BIS may inhibit risky sexual behavior. This process is referred to as "passive avoidance," or the inhibition of reward-seeking behavior due to punishment that is contingent on that behavior (Fowles, 1988). Fowles (1988) suggests that psychopaths may not learn passive avoidance due to deficits in their BIS. Mapping this onto behavioral manifestations of psychopathy, a weak inhibitory system may explain psychopaths' "emotional detachment," or their hyporesponses to certain forms of punishment.

A number of studies have tested the notion that psychopaths have weak inhibitory systems and show dampened responses to fearful stimuli. Normally, individuals would show increases in arousal in response to a fearful stimulus, demonstrating activation of the BIS. Due to hypothesized deficits in their BIS, psychopaths may not show increases in arousal, and instead, may demonstrate dampened or attenuated heart rate and skin conductance responses during the presentation of fearful stimuli. Studies using a variety of different emotion-induction approaches (e.g., startle, auditory tones) have generally supported this view (Hare, 1968; Ogloff & Wong, 1990; Patrick, 1994; see Arnett, 1997, for review), although one study did not (Patrick, Bradley, & Lang, 1993). Similarly, when psychopathy was measured as a continuous variable, individuals with high psychopathy scores demonstrated smaller increases in skin conductance activity than those individuals with

lower psychopathy scores in response to a 120-dB tone (Hare & Craigen, 1974; Hare, Frazelle & Cox 1978).

Studies using the slide-viewing startle paradigm have also supported the notion that psychopaths suffer from deficits in their BIS, and more specifically, in their fear response. Psychopaths have shown attenuated startle responses (magnitude of eye blink as measured by EMG) to unpleasant slides compared with nonpsychopaths, who demonstrated potentiated startle responses to the same slides (Patrick et al., 1993). Group differences did not emerge during the pleasant slides. Moreover, a subgroup of psychopaths with extremely high scores on emotional detachment was found to have the most attenuated startle responses during the unpleasant slides. Because there is ample evidence that fear potentiation is associated with trait measures of high fearfulness (Cook, Davis, Hawk, Spence, & Gautier, 1992), these data suggest that psychopaths are low on the dimension of fearfulness. To determine if this response patterning is specific to psychopaths, Patrick (1994) compared startle patterns of psychopathic, emotionally detached offenders and of antisocial groups of criminals. Startle response was measured in these groups during *anticipation* of a noxious blast of white noise. Only psychopaths and emotionally detached subjects demonstrated attenuated startle responses in anticipation of the blast, supporting the idea that this unique startle pattern is specific to psychopaths (i.e., not simply criminals or antisocial individuals) and that the pattern may be related to the emotional deficits that plague psychopathic individuals.

Ogloff & Wong (1990) found that the effect of psychopathy on physiological responding during the anticipation of an aversive stimulus may depend on the strategies available to avoid the stimulus. In one condition participants were able to prevent the burst of white noise by pressing a button (simulating active avoidance), whereas in the other condition participants were not able to prevent it (simulating passive avoidance). Compared with nonpsychopaths, psychopaths showed no increases in skin conductance activity and greater increases in heart rate in the passive avoidance condition. There were no differences between the groups during the active avoidance condition.

More recent theories suggest that in addition to deficits in inhibition, psychopaths may have an overactive BAS. For example, psychopathic individuals show greater heart rate responses when rewarded with money after successfully pressing a button a certain number of times within an allotted time period (Arnett, Smith & Newman, 1997). Arnett and colleagues (1997) suggest that psychopaths may have a "motivational focus" for reward; as a result, they may see their victims only as barriers to obtaining whatever reward they are pursuing, and they may feel no remorse or guilt for doing everything they can to obtain such rewards.

In summary, findings from autonomic studies have generally supported classic notions about the emotional impairments that characterize psychopathy. That is, studies of psychopaths have found that they show underarousal in response to fearful stimuli and overarousal to rewarding stimuli compared with normal controls. Future studies should include other measures of physiological responding to shed more light on the emotional underpinnings of psychopathy.

Alcohol Intoxication: Dampened Emotional Responding and Restricted Attention

Individuals expect that ingesting alcohol will reduce their stress and tension (Bennett, Janca, Grant, & Sartorius, 1993). Is this what actually happens when individuals ingest alcohol? Using basic dimensional (arousal and valence) models of emotion, Stritzke, Patrick, and Lang (1995) tested the hypothesis that alcohol acts nonselectively to dampen the impact of aversive stimuli on overall responding. Their findings supported this hypothesis: When individuals were exposed to both positive and negative slides (matched for arousal level), participants who were moderately intoxicated demonstrated dampened startle and skin conductance responses to all stimuli, not just the aversive ones. Curtin, Lang, Patrick, and Stritzke (1998) conducted a follow-up study to examine the specific mechanisms by which alcohol intoxication

dampened emotional responses. They exposed individuals to a threatening condition (one in which they expected to receive shocks) and a safe condition (one in which they expected not to receive shocks). Consistent with the findings of Stritzke and colleagues (1995), across both conditions, individuals who were moderately intoxicated showed dampened startle responses compared with those who were not, but for both groups, startle response was greater in the threatening condition than in the safe condition. Startle potentiation during the threatening condition disappeared for the intoxicated individuals only when a distracting stimulus (positively valenced and highly arousing slides) was also present. For the individuals who were not intoxicated, the presence of the distracting stimulus did not alter the startle response. These findings support theories that alcohol intoxication results in "myopia," or limited attention to stressful stimuli in cognitively demanding situations (Steele & Josephs, 1990).

Despite growing evidence that alcohol ingestion affects emotional responding in measurable and predictable ways, few studies have examined how *alcoholism* affects emotional responding. A future step may be to combine the methodological sophistication of studies such as Curtin (1998) and Stritzke (1995) and their colleagues to examine individuals who have alcoholism and those at risk for the disorder.

Depression: Electrodermal Hypoarousal and Dampened Positive Affect

The clinical literature suggests that depression increases the experience of negative emotion and decreases the experience of positive emotion. Consistent with these reports, empirical evidence suggests that depression *diminishes* the experience and expression of positive emotion during positive emotional events. Across a variety of tasks intended to elicit pleasant emotional states (e.g., imagining or recalling positive events, watching funny films or pleasant slides, tasting sweet drinks, or smiling on command), depressed individuals report experiencing *less* positive emotion (Berenbaum & Oltmanns, 1992; Gehricke & Shapiro, 1998; Schwartz et al., 1976a, 1976b) and display *less* facial behavior (Schwartz et al., 1976a, 1976b) and/or *fewer* positive facial expressions (e.g., happiness) (Berenbaum, 1992; Berenbaum & Oltmanns, 1992; Jaeger, Borod, & Peslow, 1986; Jones & Pansa, 1979; Triosi & Moles, 1999) than their nondepressed counterparts.

The few exceptions to the general pattern of attenuated positive emotion in depression are studies that demonstrate *no* differences between depressed and nondepressed groups in expressive facial behavior, as measured by electromyographic methods (Gehricke & Shapiro, 1998; Oliveau & Willmuth, 1979). These discrepancies may be due to different means of eliciting emotion and to the different muscular sites at which EMG activity was recorded. For example, Gehricke & Shapiro asked participants to imagine *situations* in which they might feel positive emotions; this may be a less effective means of eliciting emotion than having participants reexperience an emotion or inducing emotion through film clips. Oliveau & Willmuth measured corrugator activity (muscle movement above the eyebrows) only, despite findings that zygomatic major activity (muscle movement around the mouth) may be more appropriate to study in the context of positive emotion, and findings that demonstrate differences in zygomatic major activity between depressed and nondepressed persons (Schwartz et al. 1976a, 1976b).

What about responses to negative events? Studies of the effects of depression on emotional reactivity during negative emotional events have yielded a mixed pattern of results. While one study found that depression *augments* the reported experience of sadness (Schwartz et al., 1976a), several others found that depression *does not affect* the reported experience of sadness and other negative emotions (e.g., disgust) (Berenbaum & Oltmanns, 1992; Gehricke & Shapiro, 1998; Schwartz et al., 1976b). Similarly, depressed individuals either display *more* (Berenbaum, 1992; Greden, Genero, Price, Ferberg, & Levine, 1986; Jaeger et al., 1986; Schwartz et al., 1976; Teasdale & Bancroft, 1977; Troisi & Moles, 1999) or *similar* amounts of facial behavior/numbers of facial expressions as their nondepressed counterparts during negative emo-

tional events (Berenbaum & Oltmanns, 1992; Oliveau & Willmuth, 1979; Schwartz et al., 1976a, 1976b). Studies in which depressed participants express more negative emotion than nondepressed participants primarily use imagery tasks to elicit emotion. Therefore, it is possible that in these studies, group differences are due to differences in the ability to generate emotional images or in the intensity of images generated by depressed and nondepressed groups. To test this notion, Berenbaum and Oltmanns (1992) used standardized stimuli (aversive tastes and film clips) to induce negative emotion in their participants. They found *no* differences between depressed and nondepressed individuals' reported emotions and negative facial expressions in response to these stimuli.

With the exception of studies that measure expressive behavior via electromyography, most of the studies described above did not include physiological measures. However, a large body of research has compared the electrodermal and cardiovascular responses of depressed and nondepressed groups in response to stressful stimuli (e.g., mental arithmetic, auditory tones, bursts of loud noise). These studies have found that depressed individuals show *less* electrodermal reactivity than do nondepressed individuals (Donat & McCollough, 1983; Dawson, Schell, and Catania, 1977; Greenfield, Katz, Alexander, & Roessler, 1963; Lader & Wing, 1969; McCarron, 1973; Noble & Lader, 1971; Zuckerman, Persky, & Curtis, 1968). It has been suggested that such reductions in electrodermal reactivity reflect the state of low arousal, withdrawal, and internal preoccupation that characterizes depression (Dawson et al., 1977); however, studies by Iacono and colleagues (Iacono et al., 1983) have found that even remitted unipolar and bipolar depressives show the same electrodermal patterns, suggesting that these patterns are markers of risk for depression (see below). Future studies that include individuals with histories of depression and that administer emotional tasks will be able to determine whether electrodermal hyporesponding is not only a marker of depression, but also an index of dampened emotional reactivity.

Studies of the effects of depression on changes in cardiovascular reactivity in response to stressful stimuli (e.g., mental arithmetic, auditory tones, bursts of loud noise) yield a less consistent pattern of results. While some studies suggest that depressed individuals demonstrate less cardiovascular reactivity in response to stressful stimuli than do nondepressed individuals (Dawson et al., 1977), others find no differences between the groups (Zuckerman et al., 1968). Moreover, researchers argue as to whether found differences in cardiovascular reactivity are due to the state of withdrawal and internal preoccupation that characterizes depression (Dawson et al., 1977) or to variables that are often confounded with depression, such as medication use and anxiety (Iacono et al., 1983). As with studies of electrodermal reactivity in depressed groups, few studies have examined whether depression affects the cardiovascular reactivity of individuals in response to stimuli that target specific emotions and that more closely simulate emotional situations in everyday life.

To this end, Tsai, Pole, Levenson, and Muñoz (under review) compared the cardiovascular and electrodermal responses of depressed and nondepressed Spanish-speaking Latinas to sad and amusing film clips. They found that across the film clips, depressed Latinas showed smaller changes in skin conductance activity but no differences in various measures of cardiovascular response (e.g., heart rate, finger pulse amplitude, pulse transmission time to the ear) compared with normal controls, which is consistent with the above literature. These physiological findings occurred against a backdrop of differences in positive expressive behavior and reports of anger and contempt: depressed Latinas smiled less and reported experiencing more anger and contempt than did their nondepressed peers. Future studies should examine whether these findings are specific to depression and whether they hold for other cultural groups and types of depression.

Anxiety Disorders

Physiological hyperarousal (e.g., accelerated heart rate, lightheadedness, exaggerated startle

response, muscle tension) is a central feature of many of the anxiety disorders, including posttraumatic stress disorder, generalized anxiety disorder, phobias, and panic disorder (American Psychiatric Association, 1994). Psychophysiological studies of individuals with anxiety disorders have focused on two main questions: (1) Does hyperarousal occur specifically in response to the threatening stimulus? (2) What are the particular elicitors of an anxious episode? In answering these questions, these studies have often revealed other important aspects of anxiety disorders. Given the wide variety of anxiety disorders, we consider each one separately.

Phobias: Conscious and Nonconscious Stimulus-specific Fear

A phobia is the fear-mediated avoidance of an object or situation that disrupts normal daily functioning. Not surprisingly, evidence suggests that when phobics are exposed to the object(s) of their fear, their physiological responding increases. For example, in vivo exposure to animals produced reports of fear as well as increases in heart rate and blood pressure in animal phobics (Grey, Sartory, and Rachman, 1979; Nesse et al., 1985). Individuals with driving phobia demonstrated higher heart rates and increased respiratory minute volume compared with normal controls during driving tasks (Sartory, Roth & Kopell, 1992).

Davidson, Marshall, Tomarken, and Henriques (2000) and Hofmann, Newman, Ehlers, and Roth (1995) also found that compared with normal controls, individuals with a phobia of speaking in public demonstrated increased heart rate and blood pressure during speech challenge tasks, as well as greater right-sided EEG activity (Levin et al., 1993). Greater right-sided EEG activation and increases in heart rate were associated with increases in self-reports of anxiety in this sample. These findings are consistent with those that link right-sided EEG asymmetry to the experience of withdrawal-related emotions such as fear (Davidson, 1998). Other research suggests that among individuals with phobia, those with specific phobias may demonstrate even greater physiological arousal than those with generalized forms of phobia, despite the fact that the latter group actually reports more fear and distress during a speech task (Heimberg, Hope, Dodge, & Becker, 1990; Levin et al., 1993). For example, Hofmann and colleagues (1995) found that although individuals who had avoidant personality disorder and generalized social phobias reported more anxiety than those without avoidant personality disorder but with a specific phobia of public speaking during a speech task, the latter group demonstrated higher heart rate responses and smaller T-wave amplitudes (associated with greater sympathetic cardiac activity) than did the former group. Both groups reported more anxiety than normal controls. These findings suggest that individuals with discrete social phobias and those with generalized social anxiety may base their reports of anxiety on different things. For individuals with discrete social phobias, reports may be based on actual physiological response, whereas for individuals with generalized social anxiety, reports may be more influenced by their expectation of an anxiety-provoking event.

Does heightened physiological arousal in response to a fearful stimulus require conscious processing? In a seminal study, Öhman and Soares (1994) found that viewing pictures of spiders and snakes elicited higher levels of skin conductance activity among spider and snake phobics, respectively, compared with normal controls. Their findings held even when the pictures were presented very briefly and were masked to prevent conscious recognition of the images. Moreover, higher skin conductance responses were specific to particular phobias; that is, snake phobics reacted to pictures of snakes but not spiders, and vice versa. Thus, physiological methods have revealed that for phobics, stimulus-specific fear responses occur almost instantaneously, even before individuals are able to report viewing the object of their fear.

Panic Disorder: Internal and External Elicitors of Fear

One of the greatest contributions made by psychophysiological studies is in our understand-

ing of panic disorder. Initially, individuals with panic disorder were thought to hyperventilate more easily than normal controls because of decreased levels of carbon dioxide in their blood. However, researchers found that the mechanism of panic attacks is quite different: individuals with panic disorders appear to be more rather than less sensitive to arterial carbon dioxide than are normal controls (Gorman et al., 1988). Building on these findings, the experimental administration of carbon dioxide to panic patients has become a useful tool in studying what triggers a panic attack.

Not surprisingly, studies that have compared the emotional responses of individuals with panic disorder with those of normal controls have found that subjects with panic disorder demonstrate higher heart rates when exposed to panic-inducing situations, such as crowded streets and shopping malls (Khawaja, Oei, and Evans, 1993). In addition, *internal* physical sensations associated with panic attacks also appear to be powerful elicitors of fear for individuals with panic disorder. For example, Goetz and colleagues (1993) monitored individuals' physiological responses during in vivo panic attacks that occurred in response to placebo infusion and found that the panic attacks were preceded by a 10% increase in heart rate and increases in minute ventilation and tidal volume during the two minutes prior to the beginning of the attack. Others (Gorman et al., 1988) have found that after administration of carbon dioxide, individuals with panic disorder demonstrate panic responses more often than those with other anxiety disorders, also suggesting that the co-occurrence of internal and external elicitors of fear may be specific to panic.

Generalized Anxiety Disorder: Nonspecific Hypervigilance

Generalized anxiety disorder (GAD) is associated with persistent and excessive anxiety and worry, accompanied by symptoms of heightened arousal, such as restlessness and muscle tension. Although only a handful of studies have focused on the emotional impairments associated with generalized anxiety disorder, existing studies generally support clinical descriptions of this disorder. For example, subjects with GAD demonstrate faster heart rates compared to controls during baseline, and worrying is associated with increases in physiological responding (Thayer et al., 1996). Thayer and colleagues (2000) also found that compared to controls, individuals with GAD demonstrated greater heart rate acceleration in response to novel threatening stimuli and slower habituation in response to novel neutral stimuli. In the same study, the authors found that after a number of trials, participants with GAD demonstrated conditioned anticipatory heart rate deceleration in response to threat words, suggesting that they are able to employ strategies to protect themselves against anticipated threatening stimuli (e.g., via motivated inattention or internally directed attention).

Posttraumatic Stress Disorder: Trauma-specific Fear, Slower Habituation to Novel Stimuli

The DSM-IV (American Psychiatric Association, 1994, p. 428) describes posttraumatic stress disorder (PTSD) as a cluster of "characteristic symptoms following exposure to an extreme traumatic stressor." Thus, it is not surprising that compared with other male combat veterans, those with PTSD demonstrate increased heart rate, skin conductance, and blood pressure responses (Keane et al., 1998; Orr, Pitman, Lasko & Herz, 1993) and exhibit higher levels of plasma norepinephrine (Blanchard, Kolb, Prins, Gates, & McCoy, 1991) when audiovisually presented with trauma-related stimuli or when asked to imagine trauma-related scenarios. Another study compared the physiological responses of World War II and Korean War veterans with and without PTSD during script-driven imagery tasks (Orr et al., 1993). The scripts varied in content. Although the two groups did not differ in their self-reported emotions, they did differ in their physiological responses (heart rate, skin conductance, and electromyographic responses) during the script that included personal combat material, a highly trauma-relevant stimulus. The physiological responses of the two groups did not differ during

scripts that included personal trauma material unrelated to PTSD or during standard combat scripts, suggesting that for veterans with PTSD, increased arousal occurs only in response to personally traumatic material related to their PTSD. Heightened physiological arousal in response to trauma-related cues may be a result of classic aversive conditioning (Pitman, 1988).

Findings from other studies also suggest that veterans with PTSD do not show increased physiological arousal to non-trauma-related material. For example, Casada, Amdur, Larsen, and Liberzon (1998) and Orr, Meyerhoff, Edwards, and Pitman (1998) found that during generic stressful and emotion-eliciting tasks (e.g., standing up from a reclining position, performing a mental arithmetic task, placing a hand in ice water, and viewing negatively valenced and arousing slides), PTSD and non-PTSD combat veterans did not differ in their physiological responses, as measured by heart rate and systolic and diastolic blood pressure. Interestingly, this pattern of results has been found to differ for nonveterans with PTSD. That is, research on non-combat-related PTSD indicates that personally significant traumatic scripts that are unrelated to the cause of the PTSD (e.g., an individual who has a traumatic memory of a traffic accident, but whose PTSD occurred in response to sexual assault) may elicit heightened physiological response (Shalev, Gelpin, Orr, & Pitman, 1997). The differences in specificity between civilian and veteran groups may be due to factors such as duration of the PTSD or the severity of the trauma. More research is clearly needed to identify the source of these different findings.

The DSM-IV also lists exaggerated startle response as a key feature of PTSD, suggesting that individuals with PTSD respond more strongly to unexpected stimuli than do normal controls. Researchers have examined whether this clinical feature of PTSD maps on to participants' behavior in the laboratory by measuring startle responses to sudden and unexpected bursts of noise. For example, Orr, Lasko, Shalev, and Pitman (1995) administered a 95-dB burst of white noise to Vietnam veterans with and without PTSD. They found that veterans with PTSD showed increased muscular startle response and had skin conductance responses whose magnitude returned to baseline at slower rates than those of non-PTSD controls. Other studies (Metzger et al., 1999; Shalev et al., 2000) showed startle response in survivors of non-combat trauma, such as accidents, terrorist attacks, and childhood sexual abuse. Similarly, civilians with PTSD showed greater heart rate acceleration, and their skin conductance responses took longer to return to baseline than did those of trauma survivors without PTSD. Studies using startle eyeblink data support these findings (Shalev et al., 2000), although one study did not find a difference in eyeblink magnitude and habituation between subjects with PTSD and normal controls (Metzger et al., 1999). However, this disparity in findings may be due to the fact that Metzger and colleagues (1999) included all women (whereas other studies have either included only men or both men and women), and because their control group appeared to be different from those of other studies. In general, the majority of studies on PTSD report differences in the eyeblink responses of PTSD and non-PTSD individuals.

PTSD-related increases in responses to startling stimuli hold across threatening and safety conditions. For example, veterans with PTSD demonstrated increases in startle reactions in response to both threatening (anticipation of electric shock) and safety cues (no anticipation of electric shock), whereas normal controls did not (Grillon & Morgan, 1999; Grillon, Morgan, Davis, & Southwick, 1998). Grillon and Morgan (1999) found that by a second study session (which occurred several days later), veterans with PTSD began to show different startle responses to the threatening and safe stimuli. Thus, the lack of differentiation in startle responses to safe and threatening cues may reflect delays in learning rather than chronic deficits in differentiation.

In summary, the effects of PTSD on emotional responding appear to vary for different types of PTSD (combat related versus non-combat related). For veterans, PTSD increases emotional responding to PTSD-related trauma material only, whereas for civilians, PTSD in-

creases emotional responding to both PTSD and non-PTSD-related trauma material. For both groups, however, PTSD appears to alter responses to novel stimuli and to increase the amount of time needed to differentiate between threatening and safe stimuli.

The findings discussed above illustrate how psychophysiological studies have advanced our knowledge about various clinical disorders, and in some cases, have dispelled misconceptions about the disorders that were based solely on behavioral observations or self-reports. In the following sections, we focus on the ways in which these findings and others can be used in clinical settings.

PHYSIOLOGICAL PATTERNS ASSOCIATED WITH RISK FOR PSYCHOPATHOLOGY

In addition to disorder-related alterations in emotional functioning, physiological methods have been used to identify risk for psychopathology. These physiological patterns have the potential to identify individuals that are at risk for various disorders, allowing the implementation of relevant interventions that might prevent the full expression of the disorder in later life. In the studies reviewed above, comparisons were made between individuals who did and did not have a given disorder. In studies of physiological patterns associated with increased risk for a disorder, none of the participants currently have the disorder itself; instead, comparisons are made between individuals who either do or do not have a family history of the relevant disorder. In the next section, we discuss physiological patterns that are associated with increased risk for the onset of various clinical disorders and that appear to be relevant to emotional functioning.

Alcohol Abuse and Dependence

The degree to which an individual finds alcohol to be anxiolytic (or anxiety reducing) may depend on whether she or he is at risk for alcoholism. Physiological patterns that have been associated with risk for alcoholism include electrodermal underresponding in anticipation of punishment, increased physiological responding to stressors while sober, and greater alcohol-induced dampened responses to stress. For example, Finn, Kessler, and Hussong (1994) found that individuals with a positive family history of alcoholism were electrodermally less responsive to conditioned signals for an electric shock than those without a family history of alcoholism; this response was also related to a history of drinking problems. Individuals with family histories of alcoholism showed higher heart rates during electric shock while sober than those without (Finn, Earleywine, & Pihl, 1992; Finn & Pihl, 1987), and alcohol consumption led to a marked decrease in stress reactivity in high but not in low risk groups (Finn & Pihl, 1987, 1988; Levenson, Oyama, & Meek, 1987). Moreover, among those with a family history of alcoholism, individuals with a multigenerational history as opposed to a single generational history showed greater reduction in stress reactivity after alcohol consumption (Finn & Pihl, 1988). Finn, Kessler, and Hussong (1994) suggested that these patterns may indicate that individuals at risk for alcoholism underrespond to signals for punishment, increasing the frequency of extreme drinking behavior.

Depression

Physiological patterns that appear to be associated with risk for depression include frontal hemispheric asymmetry in EEG responding and skin conductance underresponding. For example, Davidson and colleagues have found that greater right frontal activation (compared with left frontal activation) during baseline is related to increased susceptibility to negative affect, whereas greater left frontal activation (compared with right frontal activation) during baseline is related to increased susceptibility to positive affect in normal samples (Tomarken, Davidson, Wheeler, & Doss, 1992). For example, Tomarken, Davidson, and Henriques

(1990) found that relative right frontal activation during baseline was associated with greater levels of negative emotional responding to negative film clips. Frontal asymmetry is not associated with a broad dimension of general emotional reactivity (Tomarken et al., 1992). Recent findings suggest that left and right frontal activation may be related to approach and withdrawal tendencies rather than to positive and negative affect (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997).

What implications do these findings have for depression? A number of studies have demonstrated relative left frontal hypoactivation in depressed persons (see Davidson, 1998, for review; Henriques & Davidson, 1991; Schaeffer, Davidson, & Saron, 1983). Less left frontal activation appears to differentiate previously depressed subjects from healthy controls (Henriques & Davidson, 1990), suggesting that it may reflect a vulnerability to or residue of depression. Moreover, there is some evidence that EEG asymmetry patterns may differentiate depression from anxiety (Keller et al., 2000). Asymmetry scores have demonstrated excellent internal consistency, adequate test-retest stability over three weeks (Tomarken et al., 1992), some degree of familial transmission, and some utility in identifying individuals at risk for the disorder (Field, Fox, Pickens, & Nawrocki, 1995), suggesting it may be a physiological marker for depression (Iacono & Ficken, 1989). It is important to note, however, that some studies have not found differences in frontal activation in depressed and nondepressed individuals (Reid, Duke, & Allen, 1998). Although it has been proposed that methodological differences make it difficult to compare findings across studies (Davidson, 1998), only future studies will determine if this is the case.

In addition to EEG asymmetry, skin conductance nonresponding has been found to be a marker for both unipolar and bipolar depression. For example, Iacono and colleagues (1983) found that individuals with a history of unipolar and bipolar depression who were not currently depressed showed dampened skin conductance responses compared with normal controls in response to a series of tones, during breathing exercises, and during a rest period.

Posttraumatic Stress Disorder

Scientists have found that physiological responding during baseline may predict posttraumatic stress disorder. For example, Shalev and co-workers (1998) assessed self-report and physiological responses of civilian trauma survivors upon their arrival at the emergency room of a hospital and followed them for several months after their initial visit. Those individuals who had higher heart rates during rest four months after the trauma originally occurred met criteria for PTSD. Other studies have used psychophysiological parameters to differentiate between individuals with current diagnoses of PTSD and normal controls (with and without a personal history of trauma). Increases in heart rate, blood pressure, EMG, and skin conductance levels during neutral and anxiety-eliciting tasks resulted in 64–80% of correct classification (Blanchard, Kolb, & Prins, 1991; Keane et al., 1998). Although future research is needed, findings from existing studies suggest that psychophysiological measures can be a useful addition to self-report measures in establishing diagnosis of PTSD. Thus, psychophysiological measurements, used in concert with other sources of information, may prove to be invaluable in identifying not only individuals who are at risk for a disorder, but also individuals who may require additional treatments to ensure a smooth recovery.

THE USE OF PSYCHOPHYSIOLOGICAL METHODS DURING TREATMENT

Psychophysiological recordings may be used in a number of ways during the course of treatment. First, the changes in emotional responding reported in the beginning of this chapter might be used to index clinical improvement. For example, to assess the effectiveness of treatments for phobia, measures of skin conductance activity may be obtained at any time during the course of treatment to determine whether the

patient continues to fear the relevant stimulus even without being aware of having been exposed to it (as found by Öhman and Soares, 1994). Second, physiological measures may identify individuals who are the most responsive to particular treatments. For example, increased heart rate during therapy among patients with anxiety disorders has been shown to be associated with success in therapy (Lang et al., 1998). Pitman and colleagues (1996) also found that increases in heart rate during the first exposure session were associated with greater decreases in the intrusive symptoms of PTSD during the course of flooding therapy. Physiological methods may also be used to ensure patient compliance during treatment. For example, in a study of image flooding therapy for PTSD, significant increases in within-session habituation were observed during the initial sessions for heart rate, skin conductance and EMG activity, and number of body movements (Pitman et al., 1996). These physiological data resembled findings based on self-report ratings, providing evidence that patients were processing emotional content during the initial sessions of exposure therapy. Finally, physiological measures may also be used to test the effectiveness of specific therapeutic interventions. For example, Teasdale and Bancroft (1977) and Teasdale and Rezin (1978) examined the effects of cognitive therapy techniques, changing the content of thoughts and thought-stopping, respectively, on emotional responding in depressed patients. Although they found that manipulating thought content altered facial corrugator activity, thought-stopping did not, shedding some light on which cognitive techniques are useful (or not) in altering the moods of depressed patients.

Given the important data that psychophysiological measures provide, it is somewhat surprising that psychophysiological measures are not used more in the treatment of clinical disorders. It is our hope that as our knowledge about the emotional underpinnings of various forms of psychopathology increases, researchers and clinicians alike will begin to examine the utility of such psychophysiological methods in clinical settings.

OTHER SOURCES OF VARIATION IN PHYSIOLOGICAL RESPONDING

When interpreting physiological differences between clinical and normal groups, it is critical to consider whether such differences may be due to developmental, sex, or racial differences in physiological responding rather than to the presence of a clinical disorder. For example, psychophysiological measures are subject to maturational changes in earlier life, and therefore may reflect growth. In the case of startle responding, modulation abilities do not mature until 8 years of age (Ornitz, Guthrie, Kaplan, Lane, & Norman, 1986). Since there has been interest in using startle as a measure of psychophysiological risk in childhood disorders (Grillon, Dierker, & Merikangas, 1997), maturational differences in a measure over development may be important to consider. Maturational changes occur at later stages of the human life span as well. For example, in one of the few studies to use multiple measures of emotional responding in old age, Tsai, Levenson, and Carstensen (2000) found that older adults demonstrated smaller changes in cardiovascular responding than did younger adults during sad and amusing film clips. These physiological differences occurred against a backdrop of no age differences in self-reports of emotional experience or expressive behavior. Thus, decreases in physiological responding in old age should not be interpreted as reductions in the capacity to experience emotions. Of course, nonpsychophysiological measures of emotion are subject to maturational changes as well. For instance, some evidence suggests that prior to the end of the first year of life, reliable assessments of emotional expressions are extremely difficult to obtain (Camras & Izard, 1991), and infant expressions of basic emotions such as anger and surprise may not be direct analogues to adult expressions (see Camras, 1994).

Physiological responses may also vary by sex. For example, autonomic responding (e.g., cardiovascular activity) is influenced by hormonal changes associated with different stages of the menstrual cycle for women (Kaplan, Whitsett,

& Robinson, 1990; Little & Zahn, 1974; Litschauer, Zauchner, Huemer, & Kafra-Luetzow, 1998). This factor and others related to physiological differences between men and women should be considered when one interprets group differences in physiological responding.

Across a number of studies, mean levels of physiological responding during emotional events do not seem to vary for some cultural groups. For example, the directed facial action task (a task in which participants are instructed to make facial configurations that resemble prototypical emotional expressions) has been shown to be effective in altering autonomic activity and subjective reports of emotional experience among American subjects. Levenson, Ekman, Heider, and Friesen (1992) studied the physiological responses and subjective reports of emotion during the directed facial action task among Minangkabau men (an ethnic group from West Sumatra). These findings were compared with data previously obtained from a North American sample. The results suggested that while the patterns of physiological response differed for specific emotional configurations, the patterns were consistent for the two cultural groups. Similarly, no differences have been found in mean levels of physiological responding between individuals of Chinese or European descent during emotional conversations with their romantic partners about areas of conflict in their relationships (Tsai & Levenson, 1997), while watching sad and amusing film clips (Tsai, Levenson, & Carstensen, 2000), or while describing past anger-provoking incidents aloud (Drummond and Quah, 2001). In Tsai and Levenson (1997) and Levenson and coworkers (1992), differences were found in reports of emotional experience, suggesting that in certain situations, culture may influence interpretation of physiological response. Although much more research is needed in this area—particularly studies that include non-Asian groups—existing studies suggest that culture exerts little influence on physiological responding during emotional events.

Other differences that may characterize groups, however, may alter physiological responding. For example, several studies have documented lower levels of skin conductance responding among African Americans compared with their Caucasian counterparts (e.g., Davis & Cowles, 1989). These differences have been attributed to properties of lighter and darker skin pigmentation (e.g., relationship between number of sweat glands and skin conductance) (Johnson & Landon, 1965). These findings suggest the importance of identifying physical sources of differences in physiological responding so as not to misinterpret group differences that may be due to physical rather than to emotional or psychopathological variation. For example, the lower levels of skin conductance responding due to skin pigmentation should not be misinterpreted as a greater risk for depression.

FUTURE RESEARCH DIRECTIONS

Clearly, there is much room (and need) for future research that explores the psychophysiology of emotion and psychopathology. Building on the work described in this chapter, future studies should include more diverse comparison groups (e.g., individuals with different clinical disorders, individuals of different cultural backgrounds) to examine the generalizability of the findings. Most of the studies described here did not include groups with disorders other than the one of interest, and therefore it is unclear whether the found deficits in emotional functioning are specific to the clinical disorder or instead reflect a more general impairment in emotional functioning. Future studies are also needed to examine how physiological deficits in emotional functioning change over the course of the disorder. For example, in the case of schizophrenia, does the disjunction of emotional responding become more intense as the level of dysfunction increases? Studies that expand their measurement of physiological responding to include more than mean levels of responding (e.g., patterning) may reveal even more ways in which emotional functioning is

impaired with various disorders. With the onset of innovative physiological techniques (e.g., fMRI), the future holds even more direct ways of measuring physiological processes that we once could only infer (e.g., localization of brain activity and function). Finally, studies that experiment with and evaluate the use of physiological techniques in clinical settings are sorely needed. These studies can best ensure that the knowledge we have gained about physiology, emotion, and psychopathology is improving the lives of individuals afflicted with mental illness.

CONCLUSION

In this chapter we have reviewed the literature that examines the effects of various forms of psychopathology on emotional functioning, using psychophysiological methods. We have described the potential clinical uses of such findings and reviewed other studies that have actually used psychophysiological methods to assess risk for developing a disorder, to predict recovery, and to monitor clinical improvement. Finally, we have discussed possible sources of variance in physiological responding that are not related to psychopathology, such as age, sex, and skin pigmentation. It is our belief that physiological measures—while subject to measurement error and misinterpretation—can provide important and useful information about the emotional state of individuals when obtained properly and in the context of other methods (self-report, behavioral observation). More specifically, we believe that physiological methods have and will continue to reveal central features of various clinical disorders as they relate to emotion. This knowledge is critical to the treatment of individuals who suffer from various forms of mental illness worldwide.

ACKNOWLEDGMENTS

The authors would like to thank David Lake, Bill Iacono, Mark Kramer, and Steve Malone for their helpful comments on earlier drafts of this manuscript.

REFERENCES

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Arnett, P. A. (1997). Autonomic responsivity in psychopaths: A critical review and theoretical proposal. *Clinical Psychology Review, 17*(8), 903–936.

Arnett, P. A., Smith, S. S., & Newman, J. P. (1997). Approach and avoidance motivation in incarcerated psychopaths. *Journal of Personality and Social Psychology, 72*, 1413–1428.

Bech, P. (1992). Symptoms and assessment of depression. In E. S. Paykel (Ed.), *Handbook of Affective Disorders* (pp. 3–13). New York: Guilford.

Bennett, L. A., Janca, A., Grant, B. F., & Sartorius, N. (1993). Boundaries between normal and pathological drinking: A cross-cultural comparison. *Alcohol, Health and Research World, 17*(3), 190–195.

Berenbaum, H. (1992). Posed facial expression of emotion in schizophrenia and depression. *Psychological Medicine 22*(4), 929–937.

Berenbaum, H., & Oltmanns, T. F. (1992). Emotional experience and expression in schizophrenia and depression. *Journal of Abnormal Psychology, 101*, 37–44.

Blanchard, E. B., Kolb, L. C., & Prins, A. (1991). Psychophysiological responses in the diagnosis of posttraumatic stress disorder in Vietnam veterans. *Journal of Nervous and Mental Disease, 179*(2), 97–101.

Blanchard, E. B., Kolb, L. C., Prins, A., Gates, S., & McCoy, G. C. (1991). Changes in plasma norepinephrine to combat-related stimuli among Vietnam veterans with posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 179*(6), 371–373.

Cacioppo, J. T., & Tassinary, L. G. (1990). *Principles of psychophysiology: Physical, social, and inferential elements*. New York: Cambridge University Press.

Camras, L. A. (1994). Two aspects of emotional development: Expression and elicitation. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion* (pp. 347–351). New York: Oxford University Press.

Camras, L. A., C., M., & Izard, C. (1991). The development of facial expressions in infancy. In R. Feldman & B. Rime (Eds.), *Fundamentals of nonverbal behavior* (pp. 73–105). New York: Cambridge University Press.

Casada, J. H., Amdur, R., Larsen, R., & Liberzon, I. (1998). Psychophysiologic responsivity in posttraumatic stress disorder: Generalized hyperresponsiveness versus trauma specificity. *Biological Psychiatry, 44*, 1037–1044.

Cleckley, H. (1982). *The mask of sanity*. St. Louis: C.V. Mosby Company.

Cook, E. W., Davis, T. L., Hawk, L. W., Spence, E. L., & Gautier, C. H. (1992). Fearfulness and startle potentiation during aversive visual stimuli. *Psychophysiology, 29*, 633–645.

Cuthbert, B. N., Schupp, H. T., Bradley, M., McManis, M., & Lang, P. J. (1998). Probing affective pictures: Attended startle and tone probes. *Psychophysiology, 35*, 344–347.

Curtin, J. J., Lang, A. R., Patrick, C. J., & Stritzke, W. G. K. (1998). Alcohol and fear-potentiated startle: The role of competing cognitive demands in the stress-reducing effects of intoxication. *Journal of Abnormal Psychology, 107*(4), 547–557.

Curtis, G. E., Lebow, B., Lake, D. S., Katsanis, J., & Iacono, W. G. (1999). Acoustic startle reflex in schizophrenia patients and their first-degree relatives: Evidence of normal emotional modulation. *Psychophysiology, 36*(4), 469–475.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35*, 607–614.

Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry, 47*, 85–95.

Davis, C., & Cowles, M. (1989). Some sources of variance in skin conductance. *Canadian Journal of Psychology, 43*, 97–103.

Davison, G. C., & Neale, J. M. (1994). *Abnormal psychology (6th ed.)*. New York: John Wiley & Sons.

Dawson, M. E., Schell, A., & Catania, J. (1977). Autonomic correlates of depression and clinical improvement following electroconvulsive shock therapy. *Psychophysiology, 14*, 569–578.

Donat, D. C. & McCullough, J. P. (1983). Psychophysiological discriminants of depression at rest and in response to stress. *Journal of Clinical Psychology, 39*, 315–320.

Donchin, E., Callaway, E., Cooper, R., Desmedt, J. E., Goff, W. R., Hillyard, S. A., & Sutton, S. (1977). Publication criteria for studies of evoked potentials (EP) in man. *Progress in Clinical Neurophysiology, 1*, 1–11.

Drummond, P. D., & Quah, S. H. (2001). The effect of expressing anger on cardiovascular reactivity and facial blood flow in Chinese and Caucasians. *Psychophysiology 38*, 2190–2196.

Field, T., Fox, N. A., Pickens, J., & Nawrocki, T. (1995). Relative frontal EEG activation in 3-6-month old infants of "depressed" mothers. *Development and Psychopathology, 4*, 67–80.

Finn, P. R., Farleywine, M., & Pihl, R. O. (1992). Sensation seeking, stress reactivity and alcohol dampening discriminate the density of a family history of alcoholism. *Alcoholism: Clinical and experimental research, 16*, 585–590.

Finn, P. R., Kessler, D. N., & Hussong, A. M. (1994). Risk for alcoholism and classical conditioning to signals for punishment: Evidence for a weak behavioral inhibition system? *Journal of Abnormal Psychology, 103*(2), 293–301.

Finn, P. R., & Pihl, R. O. (1987, Aug.). Men at high risk for alcoholism: The effect of alcohol on cardiovascular response to unavoidable shock. *Journal of Abnormal Psychology, 96*(3).

Finn, P. R., & Pihl, R. O. (1988, Dec.). Risk for alcoholism: A comparison between two different groups of sons of alcoholics on cardiovascular reactivity and sensitivity to alcohol. *Alcoholism: Clinical & Experimental Research, 12*(6).

Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology, 25*(4), 373–391.

Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurement. *Psychophysiology, 18*, 232–239.

Fridlund, A. J. & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. *Psychophysiology, 23*, 567–589.

Funder, D. (2001). *The personality puzzle*. New York: W.W. Norton.

Gehricke, J. G., & Shapiro, D. (1995). *Flat affect and social disengagement in depression: Facial and autonomic activity in response to social and solitary imagery*. Paper presented at the 38th Annual Meeting of the Society for Psychophysiological Research, Denver.

Goetz, R. R., Klein, D. F., Gully, R., Kahn, J., Liebowitz, M. R., Fyer, A. J., & Gorman, J. M. (1993). Panic attacks during placebo procedures in the laboratory. *Archives of General Psychiatry, 50*, 280–285.

Gorman, J. M., Fyer, M. R., Goetz, R., Askanazi, J., Liebowitz, M. R., Fyer, A. J., Kinney, J., & Klein, D. F. (1988). Ventilatory physiology of patients

with panic disorder. *Archives of General Psychiatry, 45,* 31–39.

Gray (1975). *Elements of a two process theory of learning.* New York: Academic Press.

Greden, J. F., Genero, N., Price, L., Feiberg, M., & Levine, S. (1986). Facial electromyography in depression. *Archives of General Psychiatry, 43,* 269–274.

Greenfield, N. S., Katz, D., Alexander, A. A., & Roessler, R. (1963). The relationship between physiological and psychological responsivity: Depression and galvanic skin response. *Journal of Nervous and Mental Disease, 136,* 535–539.

Grey, S., Sartory, G., & Rachman, S. (1979). Synchronous and desynchronous changes during fear reduction. *Behavior Research & Therapy, 17*(2), 137–147.

Grillon, C., Dierker, L., & Merikangas, K. R. (1997). Startle modulation in children at risk for anxiety disorders and/or alcoholism. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 925–932.

Grillon, C., & Morgan, C. A. (1999). Fear-potentiated startle conditioning to explicit and contextual cues in Gulf war veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology, 108*(1), 134–142.

Grillon, C., Morgan, C. A., Davis, M., & Southwick, S. M. (1998). Effects of experimental context and explicit threat cues on acoustic startle in Vietnam veterans with posttraumatic stress disorder. *Biological Psychiatry, 44,* 1027–1036.

Gross, J. J., & Levenson, R.W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64,* 970–986.

Hare, R. (1968). Psychopathy, autonomic functioning and the orienting response. *Journal of Abnormal Psychology, 73*(3), 1–24.

Hare, R. D., & Craigen, D. (1974). Psychopathy and physiological activity in a mixed-motive game situation. *Psychophysiology, 11*(2), 197–206.

Hare, R. D., Frazelle, J., & Cox, D. N. (1978). Psychopathy and physiological responses to threat of an aversive stimulus. *Psychophysiology, 15*(2), 165–172.

Harmon-Jones, E., & Allen, J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology, 106*(1), 158–163.

Heimberg, R. G., Hope, D. A., Dodge, C. S., & Becker, R. E. (1990). DSM-III-R subtypes of social phobia: Comparison of generalized social phobics and public speaking phobics. *Journal of Nervous and Mental Disease, 178*(3), 172–179.

Henriques, J. B., & Davidson, R. J. (1990). Regional brain asymmetries discriminate between previously depressed and healthy control subjects. *Journal of Abnormal Psychology, 99*(1), 22–31.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology, 100*(4), 535–545.

Hofmann, S. G., Newman, M. G., Ehlers, A., & Roth, W. T. (1995). Psychophysiological differences between subgroups of social phobia. *Journal of Abnormal Psychology, 104*(1), 224–231.

Iacono, W. G. (1991). Psychophysiological assessment of psychopathology. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3*(3), 309–320.

Iacono, W. G., & Ficken, J. W. (1989). Research strategies employing psychophysiological measures: Identifying and using psychophysiological markers. In G. Turpin (Ed.), *Handbook of Clinical Psychophysiology* (pp. 45–70). New York: Wiley.

Iacono, W.G., Lykken, D., Peloquin, L., Lumry, A., Valentine, R. H., & Tuason, V. (1983). Electrodermal activity in euthymic unipolar and bipolar affective disorders. *Archives of General Psychiatry, 40,* 557.

Jaeger, J., Borod, J. C., & Peslow, E. (1986). Facial expression of positive and negative emotions in patients with unipolar depression. *Journal of Affective Disorders, 11,* 43–50.

James, W. (1884). What is an emotion? *Mind, 9,* 188–205.

Jennings, J. R., Berg, W. K., Hutcheson, J. S., Obrist, P. A. & Turpin, G. (1981). Publication guidelines for heart rate studies in man. *Psychophysiology, 18,* 226–231.

Johnson, L., & Landon, M. (1965). Eccrine sweat gland activity and racial differences in resting skin conductance. *Psychophysiology, 1,* 322–329.

Jones, J. H., & Pansa M. (1979). Some nonverbal aspects of depression and schizophrenia occurring during the interview. *Journal of Nervous and Mental Disease, 167,* 402–409.

Kaplan, B. J., Whitsett, S. F., & Robinson, J. W. (1990). Menstrual cycle phase is a potent confound in psychophysiology research. *Psychophysiology, 27,* 445–450.

Keane, T. M., Kolb, L. C., Kaloupek, D. G., Orr, S. P., Blanchard, E. B., Thomas, R. G., Hsieh, F. Y., & Lavori, P. W. (1998). Utility of psy-

chophysiological measurement in the diagnosis of posttraumatic stress disorder: Results from a Department of Veterans Affairs cooperative study. *Journal of Consulting and Clinical Psychology, 66*(6), 914–923.

Keller, J., Nitschke, J. B., Bhargava, T., Deldin, P., Gergen, J. A., Miller, G. A., & Heller, W. (2000). Neuropsychological differentiation of depression and anxiety. *Journal of Abnormal Psychology, 109*(1), 3–10.

Khawaja, N. G., Oei, T. P. S., & Evans, L. (1993). Comparison between the panic disorder with agoraphobia patients and normal controls on the basis of cognitions, affect and physiology. *Behavioral and Cognitive Psychotherapy, 21*, 199–217.

Kring, A. M., Kerr, S. L., Smith, D. A., & Neale, J. M. (1993). Flat affect in schizophrenia does not reflect diminished subjective experience of emotion. *Journal of Abnormal Psychology, 102*(4), 507–517.

Lader, M. H., & Wing, L. (1969). Physiological measures in agitated and retarded depressed patients. *Journal of Psychiatric Research, 7*, 89–100.

Lang, P. J. (1995). The emotion probe. *American Psychologist, 50*(5), 372–385.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention and the startle reflex. *Psychological Review, 97*, 377–395.

Lang, P. J., Cuthbert, B. N., & Bradley, M. M. (1998). Measuring emotion in therapy: Imagery, activation, and feeling. *Behavior Therapy, 29*, 655–674.

Levenson, R.W. (1994). Human emotions: A functional view. In P. Ekman and R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 123–130). New York: Oxford University Press.

Levenson, R. W., Ekman, P., Heider, K., & Friesen, W. V. (1992). Emotion and autonomic nervous system activity in the Minangkabau of West Sumatra. *Journal of Personality and Social Psychology, 62*, 972–988.

Levenson, R. W., Oyama, O. N., & Meek, P. S. (1987). Greater reinforcement from alcohol for those at risk: Parental risk, personality risk and sex. *Journal of Abnormal Psychology, 96*(3), 242–253.

Levin, A. P., Saoud, J. B., Gorman, J. M., Fyer, A. J., Crawford, R., & Liebowitz, M. R. (1993). Responses of generalized and discrete social phobias during public speaking challenge. *Journal of Anxiety Disorders, 7*, 207–221.

Litschauer, B., Zauchner, S., Hnemer, K.-H., & Kafka-Luetzow, A. (1998, Mar.–Apr.). Cardiovascular, endocrine, and receptor measures as related to sex and the menstrual cycle phase. *Psychosomatic Medicine, 60*(2).

Little, B. C., & Zahn, T. P. (1974). Changes in mood and autonomic functioning during the menstrual cycle. *Psychophysiology, 11*, 579–590.

Lykken, D. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Clinical Psychology, 55*, 6–10.

Metzger, L. J., Orr, S. P., Berry, N. J., Ahern, C. E., Lasko, N. B., & Pitman, R. K. (1999). Physiological reactivity to startling tones in women with posttraumatic stress disorder. *Journal of Abnormal Psychology, 108*(2), 347–352.

McCarron, L. T. (1973). Psychophysiological discriminants of reactive depression. *Psychophysiology, 10*, 225–229.

Neale, J. M., Blanchard, J. J., Kerr, S., Kring, A. M., & Smith, D. A. (1998). Flat affect in schizophrenia. In W. F. Flack, Jr. & J. D. Laird (Eds), Emotions in psychopathology: Theory and research (pp. 353–364). New York: Oxford University Press.

Noble, P., & Lader, M. (1971a). Depressive illness, pulse rate, and forearm blood flow. British *Journal of Psychiatry, 119*, 261–266.

Noble, P., & Lader, M. (1971b). The symptomatic correlates of the skin conductance changes in depression. *Journal of Psychiatric Research, 9*, 61–69.

Öhman, A., & Soares, J. J. F. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology, 103* (2), 231–240.

Ogloff, J. R., & Wong, S. (1990, June). Electrodermal and cardiovascular evidence of a coping response in psychopaths. *Criminal Justice & Behavior, 17*(2).

Oliveau, D., & Willmuth, R. (1979). Facial muscle electromyography in depressed and non-depressed hospitalized subjects: A partial replication. *American Journal of Psychiatry, 136*, 548–550.

Ornitz, E. M., Guthrie, D., Kaplan, A. R., Lane, S. J., & Norman, R. J. (1986). Maturation of startle modulation. *Psychophysiology, 23*(6), 624–634.

Orr, S. P., Lasko, N. B., Shalev, A. Y., & Pitman, R. K. (1995). Physiological responses to loud tones in Vietnam veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology, 104*(1), 75–82.

Orr, S. P., Meyerhoff, J. L., Edwards, J. V., & Pitman, R. K. (1998). Heart rate and blood pressure resting levels and responses to generic stressors in

Vietnam veterans with posttraumatic stress disorder. *Journal of Traumatic Stress, 11*(1), 155–164.

Orr, S. P., Pitman, R. K., Lasko, N. B., & Herz, L. R. (1993). Psychophysiological assessment of posttraumatic stress disorder imagery in World War II and Korean combat veterans. *Journal of Abnormal Psychology, 102*(1), 152–159.

Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology, 31*, 319–330.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of Abnormal Psychology, 102*(1), 82–92.

Peri, T., Ben-Shakir, G., Orr, S. P., & Shalev, A. Y. (2000). Psychophysiological assessment of aversive conditioning in posttraumatic stress disorder. *Biological Psychiatry, 47*, 512–519.

Pitman, R. K. (1988). Post traumatic stress disorder, conditioning, and network theory. *Psychiatric Annals, 18*, 221–223.

Pitman, R. K., Orr, S. P., Altman, B., Longpre, R. E., Poire, R. E., Macklin, M. L., Michaels, M. J., & Steketee, G. S. (1996). Emotional processing and outcome of imaginal flooding therapy in Vietnam veterans with chronic posttraumatic stress disorder. *Comprehensive Psychiatry, 37*(6), 409–418.

Reid, S. A., Duke, L. M., & Allen, J. J. B. (1998). Resting frontal electroencephalographic asymmetry in depression: Inconsistencies suggest the need to identify mediating factors. *Psychophysiology, 35*, 389–404.

Salem, J., & Kring, A. (1999). Flat affect and social skills in schizophrenia: Evidence for their independence. *Psychiatry Research, 87*, 159–167.

Sartory, G., Roth, W. T., & Kopell, M. L. (1992). Psychophysiological assessment of driving phobia. *Journal of Psychophysiology, 6*, 311–320.

Schaeffer, C. E., Davidson, R. J., & Saron, C. (1983). Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. *Biological Psychiatry, 7*, 753–762.

Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., & Klerman, G. L. (1976a). Facial muscle patterning to affective imagery in depressed and nondepressed subjects. *Science, 192*, 489–491.

Schwartz, G. E., Fair, P. L., Salt, P., Mandel, M. R., & Klerman, G. L. (1976b). Facial expression and imagery in depression: An electromyographic study. *Psychosomatic Medicine, 38*, 337–347.

Shalev, A. Y., Gelpin, E., Orr, S. P., & Pitman, R. K. (1997). Psychophysiological assessment of mental imagery of stressful events in Israeli civilian posttraumatic stress disorder patients. *Comprehensive Psychiatry, 38*(5), 269–273.

Shalev, A. Y., Peri, T., Brandes, D., Freedman, S., Orr, S. P., & Pitman, R. K. (2000). Auditory startle response in trauma survivors with posttraumatic stress disorder: A prospective study. *American Journal of Psychiatry, 157*(2), 255–261.

Shalev, A. Y., Sahar, T., Freedman, S., Peri, T., Glick, N., Brandes, D., Orr, S. P., & Pitman, R. K. (1998). A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder. *Archives of General Psychiatry, 55*, 553–559.

Strube, M. (1990). Psychometric principles: From physiological data to psychological constructs. In J. Cacioppo and L. Tassinary (Eds.), *Principles of psychophysiology: Physical, social, and inferential elements* (pp. 34–57). New York: Cambridge University Press.

Stritzke, W. G. K., Lang, A. R., & Patrick, C. J. (1996). Beyond stress and arousal: A reconceptualization of alcohol-emotion relations with reference to psychophysiological methods. *Psychological Bulletin, 120*(3), 376–395.

Stritzke, W. G. K., Patrick, C. J., & Lang, A. R. (1995). Alcohol and human emotion: A multidimensional analysis incorporating startle probe methodology. *Journal of Abnormal Psychology, 104*(1), 114–122.

Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist, 45*, 921–933.

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8*(3), 204–210.

Teasdale, J. D. & Bancroft, J. (1977). Manipulation of thought content as a determinant of mood and corrugator electromyographic activity in depressed patients. *Journal of Abnormal Psychology, 86*, 235–241.

Teasdale, J. D. & Rezin, V. (1978). Effect of thought-stopping on thoughts, mood, and corrugator EMG in depressed patients. *Behavior Research and Therapy, 16*, 97–102.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39*(4), 255–266.

Thayer, J. F., Friedman, B. H., Borkovec, T. D., Johnsen, B. H., & Molina, S. (2000). Phasic heart period reactions to cued threat and nonthreat stimuli in generalized anxiety disorder. *Psychophysiology, 37*(3), 361–368.

Tomarken, A. J., Davidson, R. J., & Henriques, J. B. (1990). Resting frontal brain asymmetry predict

affective responses to films. *Journal of Personality and Social Psychology, 4*, 791–801.

Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Doss, R. C. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *Journal of Personality and Social Psychology, 62*(4), 676–687.

Troisi, A., & Moles, A. (1999). Gender differences in depression: An ethological study of nonverbal behavior during interviews. *Journal of Psychiatric Research, 33*(3), 243–250.

Tsai, J. L., & Levenson, R. W. (1997). Cultural influences on emotional responding: Chinese American and European American dating couples during interpersonal conflict. *Journal of Cross-Cultural Psychology, 28*, 600–625.

Tsai, J. L., Levenson, R. W., & Carstensen, L. L. (2000). Autonomic, expressive, and subjective responses to emotional films in older and younger Chinese American and European American adults. *Psychology and Aging, 15*, 684–693.

Tsai, J. L., Pole, N., Levenson, R. W., & Muñoz, R. F. (under review). The effects of depression on the emotional responses of Spanish-speaking Latinas.

Vrana, S. R., Spence, E. L., & Lang, P. L. (1988). The startle probe response: A new measure of emotion? *Journal of Abnormal Psychology, 97*(4), 487–491.

Zuckerman, M., Persky, H., & Curtis, G. C. (1968). Relationships among anxiety, depression, hostility, and autonomic variables. *Journal of Nervous and Mental Disease, 146*, 481.