CHAPTER 36

Fear and Anxiety: Evolutionary, Cognitive, and Clinical Perspectives

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Very softly down the glade runs a waiting, watching shade,
And the whisper spreads and widens far and near;
And the sweat is on thy brow, for he passes even now—
He is Fear, O Little Hunter, he is Fear!

On thy knees and draw the bow; bid the shrilling arrow go;
In the empty, mocking thicket plunge the spear;
But thy hands are loosed and weak, and the blood has left thy cheek—
It is Fear, O Little Hunter, it is Fear!

Now the spates are banked and deep; now the footless boulders leap—
Now the lightning shows each littlest leaf-rib clear—
But thy throat is shut and dried, and thy heart against thy side
Hammers: Fear, O Little Hunter—this is Fear!
(Kipling, 1895/1983, pp. 176–177)

Fear and anxiety provide recurrent themes for humans pondering their existential predicament. For example, in a theological version, anxiety has been interpreted as resulting from "divine disconnection"—the experience of being separated from God's grace. In existential philosophy, on the other hand, the distress of anxiety is seen as something positive—as the mark of a person exercising his or her freedom and responsibility to choose an authentic life. In a clinical context, the vicissitude of anxiety has been understood as the key to the dynamics of psychopathology, whether it is conceptualized in terms of a learnable drive supporting escape and avoidance, or as the target of psychologically distorting defense mechanisms.

The ubiquity and controversial status of anxiety have made it a central topic for research and reflection. Thus there is a truly voluminous literature on the psychology of anxiety (see, e.g., Barlow, 1988; Rapee, 1996; Tuma & Maser, 1985), only a tiny fraction of which can be represented in this chapter. Its point of departure is that fear and anxiety are emotional phenomena, the elucidation of which has much to gain from being informed by the psychology of emotion. Conversely, empirical data on the clinical phenomena of fear and anxiety provide flushing, shallow breathing, and reports of heart palpitations, intestinal discomforts, and aches and pains) and "cognitive or psychic anxiety" (including, e.g., intrusive and unwanted thoughts, worrying, ruminations, restlessness, and sometimes feelings of muscle tension) (e.g., Buss, 1962; Fenz & Epstein, 1965; Schalling, Cronholm, & Åsberg, 1975).

THE SITUATIONAL CONTEXT OF FEAR AND ANXIETY

Traumatic Situations

Extreme danger jeopardizing one's life (or the lives of close kin) elicits intense fright and may have long-lasting consequences in the form of posttraumatic stress disorder (PTSD). Trauma may also involve natural catastrophes, such as floods or hurricanes destroying one's home or community; or it may involve seeing others being seriously injured or killed as a result of an accident or physical violence. One may be exposed to the trauma alone, as in a rape or an assault, or in a group, as in military combat. If the trauma results in PTSD, the traumatic event is persistently reexperienced (e.g., in the form of "flashbacks"); stimuli or events associated with the trauma are avoided; and the person feels generally numbed with regard to emotions. Common anxiety symptoms experienced by persons suffering from PTSD include sleep and concentration difficulties, irritability or anger outbursts, hypervigilance, and exaggerated startle. Some events, such as torture, frequently result in PTSD, whereas others, such as natural disasters or car accidents, only occasionally result in the disorder (APA, 1994).

Commonly Feared and Potentially Phobic Situations

Survival considerations, either contemporary or in an evolutionary perspective, are relevant for most situational dimensions of human fears. Arrindell, Pickersgill, Merckelbach, Ardon, and Cornet (1991) provided an extensive review of studies factor-analyzing questionnaire data on self-reported fear. After applying strict methodological criteria, they accepted 25 out of 38 published studies for their own analysis. They found that the 194 factors and components identified in these studies could be classified into a structure involving four factors. The first factor was "fears about interpersonal events or situations." It included fears of criticism and social interaction, rejection, conflicts, and evaluation, but also interpersonal aggression and display of sexual and aggressive scenes. The second factor was "fears related to death, injuries, illness, blood, and surgical procedures." This factor had a quite heterogeneous content, incorporating fears of illness, diseases, and disabilities; complaints about physical and mental problems; fears of suicide, homosexuality, and sexual inadequacy; and fears of losing control. Finally, it incorporated fears of contamination, syncope, or other threats to physical health. The third factor, "fear of animals," included common domestic animals; other small, often harmless animals; and creeping and crawling animals such as insects and reptiles. Finally, "agoraphobic fears" was the fourth factor. It involved fear of entering public places (such as stores or shopping malls) and crowds, but also fear of closed spaces (such as elevators, tunnels, theaters, or churches). Furthermore, it involved fears of traveling alone in trains or buses, crossing bridges, and entering open spaces.

All these four factors represent situations of relevance for human evolution (see Seligman, 1971). Human history is replete with examples of how social conflicts that have escalated out of control provide a potentially deadly danger, not to speak of the social threat in terms of the defeat and humiliation they may involve (Öhman, 1986). Thus it comes as no surprise that social interactions are sometimes feared. For fear of death and illness, and associated bodily conditions, there is no need to elaborate the potential survival threat. Although many animals are friendly and sought as companions, there is no question that animals as predators have provided recurrent threats in the evolution of humankind, and it is reasonable to give reptiles a privileged position as the prototypical predators (Öhman, 1986; Öhman, Dimberg, & Öst, 1985). Finally, agoraphobic fears center on the lack of security inherent in separation from safe bases and kin, and the avoidance of places associated with panic and feelings of discomfort.

It is immediately seen that the factors isolated by Arrindell et al. (1991) correspond to four prominent types of phobia: social phobia, blood phobia, animal phobia, and agoraphobia. Furthermore, the second factor incorporates fears often encountered in panic disorder, such as fears or syncope, and in obsessive–compulsive disorder, such as contamination.

Departing from a preparedness perspective on phobias (Seligman, 1971), my colleagues and I (Öhman et al., 1985) have argued that these fear factors may be taken to reflect basic behavioral systems, which have been adaptively shaped by evolution. In particular, we have suggested that social fears resulted from a dominance–submissiveness system, the adaptive function of which was to promote social order by means of facilitating the establishment of dominance hierarchies. Animal fears, on the other hand, are attributed to a predatory defense system, originating in the fear of reptiles by early mammals, and prompting rapid escape from potential predators.

These basically adaptive systems are held to be compromised into producing social and animal phobias when the fear response they engender becomes conditioned to stimuli that actually are harmless in the ecology of modern humans. The basic argument is that evolution has equipped humans with a propensity to associate fear with situations that threatened the survival of their ancestors (Seligman, 1971). The propensity must be based in the genes, and thus genetically based variation in phobias can be expected. This is supported by data from behavioral genetics, which suggest that animal phobias result from the interaction between a genetic component common to all phobias and specific environmental influences (Kendler, Neale, Kessler, Heath, & Eaves, 1992). Hence, although humans in general are prepared to acquire some fears (e.g., snake fears) easily, some individuals must be more prepared than others. Furthermore, whether a phobia is developed or not depends on environmental exposure. We have tested these ideas (see reviews by Dimberg & Öhman, 1996; McNally, 1987; Öhman, 1993) in autonomic conditioning experiments primarily comparing acquisition and resistance to extinction of skin conductance conditioning to potentially phobic (e.g., snakes, spiders, angry faces) and neutral (e.g., flowers, mushrooms, neutral faces, or friendly faces) stimuli. The general, but not invariable, finding has been that responses conditioned to potentially phobic stimuli show enhanced resistance to extinction, compared to responses conditioned to neutral stimuli (Dimberg & Öhman, 1996; McNally, 1987; Öhman, 1993). Examples of data on skin conductance responses from a single cue conditioning paradigm are given in Figure 36.1.

Panic Stimuli

Biologically oriented theorists of panic (e.g., Klein, 1981; Sheehan, 1982) have suggested that panic attacks are spontaneous or endogenously originated. Thus such theorists have held the lack of an eliciting stimulus to be one of the essential characteristics of panic. However, when prospectively assessed, naturally occurring panic attacks typically appear to have precipitants, such as arguments with family members or problems at work (Freedman, Ianni, Ettedgui, & Puthezhath, 1985) or ideations of threat and fear (Hibbert, 1984). The stimuli that elicit panic may be primarily internal—for example, changes in heart rate (Pauli et al., 1991) or other bodily symptoms. Persons suffering from panic disorder, furthermore, appear both to be more sensitive to such stimuli, and to rate them as more dangerous, than controls (Ehlers, 1993). Bodily stimuli, however, are insufficient to produce panic. According to an influential formulation by Clark (1986, 1988), it is only when the bodily stimulation is combined with a catastrophic cognitive interpretation (e.g., an impending heart attack or suffoca-

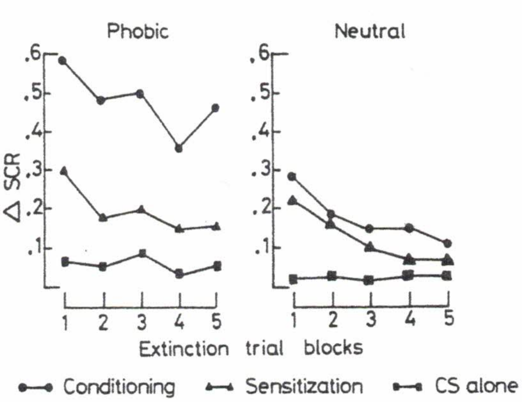

FIGURE 36.1. Extinction of skin conductance responses (SCRs) in subjects who were conditioned to potentially phobic stimuli (pictures of snakes; left panel) or neutral stimuli (pictures of houses; right panel) by receiving them paired with electric shock (unconditioned stimulus). Control subjects had the pictures and the shocks unpaired ("Sensitization") or were only exposed to pictures ("CS alone"). It is obvious that potentially phobic conditioned stimuli resulted in much larger resistance to extinction than did neutral conditioned stimuli. Data from Öhman, Eriksson, and Olofsson (1975).

tion) that panic is elicited. Thus cognitive interpretations may create a vicious circle: The catastrophic interpretation results in more intensely perceived threat and more apprehension; further bodily sensations feed further catastrophic interpretations; and so on (see Pauli et al., 1991; Rapee, 1993). If the symptoms are not given a catastrophic interpretation, however, they may not be associated with fear but with interpretations in terms of bodily problems, as in "panic without fear" (Kushner & Beitman, 1990). In agreement with the cognitive perspective, studies show that the most consistent symptomatic differences between patients with panic disorder and patients with other anxiety disorders pertain not to symptoms but to interpretation of symptoms, such as fears of dying, going crazy, or losing control (Barlow et al., 1985; Borden & Turner, 1989; Rapee, Sanderson, McCauley, & Di Nardo, 1992).

This review suggests that the phasic responses of increased state anxiety seen in phobia, PTSD, and panic typically have identifiable eliciting stimuli. Indeed, panic, phobic fear, and the intense anxiety exhibited by PTSD patients reminded of their trauma appear to result from the activation of one and the same underlying anxiety response. This anxiety response may profitably be viewed as an

> adaptation that evolved to facilitate flight from life-threatening danger. The sudden increase in the rate and strength of cardiac contractions sends extra blood to the muscles, while the gut feels empty and the skin blanches and becomes cool as blood is shunted elsewhere. Rapid and deep breathing increases blood oxygen content. Cooling sweat is secreted, muscles tighten and tremble, and the endocrine system prepares for catabolism (Nesse, Cameron, Curtis, McCann, & Huber-Smith, 1984; Mason, 1968). Intense mental activity is focused on planning escape. When the overwhelming urge to flee is translated into action, all effort is concentrated on escape. The direction of flight is towards home and trusted kin, a behavioral pattern typical of animals that rely on homes and kins for protection. (Nesse, 1987, p. 77S)

Viewed from this evolutionary perspective, it is not the fear/anxiety response in itself that is malfunctional and maladaptive, but the fact that it is triggered in a malfunctional context, as in phobias (see Öhman et al., 1985), or that it may have a dysfunctionally low threshold, as in panic (Klein, 1981, 1993; Nesse, 1987). Furthermore, the full response may be more or less completely triggered in different situations, depending upon the situational context and the overt defense responses it affords (in terms of, e.g., flight, attack, or submission). For example, active escape may be much more functional in animal phobia than in social phobia, which may account for some of the differences between these two types of disorders (Öhman et al., 1985).

THEORETICAL PERSPECTIVE: THE ROLE OF UNCONSCIOUS PROCESSES IN ANXIETY AND FEAR

According to this review, fear/anxiety is rooted in defense responses, which have evolved because they were functional devices to keep people away from potentially deadly contexts. Furthermore, these defenses have been tied to situations involving survival threats, either directly or indirectly, through evolutionary considerations. This evolutionarily inspired stimulus–response analysis, however, is by necessity incomplete, because it does not specify the mechanisms whereby a fear stimulus evokes a response.

A Functional Perspective on Anxiety

Evolved defense responses are of little use unless they are appropriately elicited. To function adequately, they require a perceptual system that can effectively locate threat. Clearly, false negatives (i.e., failing to elicit defense to a potentially hazardous stimulus) are more evolutionarily costly than false positives (i.e., eliciting the response to a stimulus that is in effect harmless). Whereas the former are potentially lethal, the latter, even though distressing to the individual, merely represent wasted energy. In an evolutionary perspective, therefore, it is likely that perceptual systems are biased toward discovering threat. Indeed, this provides an evolutionary reason for why there are anxiety disorders. To guarantee effective defense when life is at stake, the system "plays it safe" by sometimes evoking defense in what turn out to be nonthreatening contexts. Responses of the latter type, of course, seem unnecessary and unreasonable, and may be understood as "irrational anxiety" by both observers and the person. Hence, if anxiety is not (as claimed by existential philosophers) the price for the freeanalysis suggests that anxiety and fear may be activated as a correlate to recruitment of defense responses after merely unconscious analyses of stimuli. As a result of these analyses, threatening stimuli are selected for further conscious, controlled processing. Because they are located by automatic perceptual mechanisms, the person is not necessarily aware of the eliciting stimuli, which may result in episodes of anxiety. Thus what appear from the inside to be "spontaneous" episodes of anxiety may in fact be the results of unconscious stimulation.

Experimental Test of the Model: Unconscious Activation of Phobias

Backward Masking

The central theoretical tenet of this model is that responses of anxiety and fear can be elicited after only a preliminary, unconscious analysis of the stimulus. Its empirical examination, therefore, requires a means of presenting fear stimuli outside of the subject's awareness. Such a means is provided by backward masking. Seminal work by Marcel (1983) demonstrated that subjects appeared to process backwardly masked target stimuli for semantic meaning, even though the intervals between targets and masks were selected to preclude conscious perception of the targets. Backward masking has been regarded as the potentially most fruitful avenue to unconscious perception (Holender, 1986; Öhman, 1999). Thus if backwardly masked fear stimuli were presented to fearful subjects, and still elicited physiological responses suggesting activation of fear/anxiety even though conscious recognition could be ruled out, the theoretical notions advanced here would receive experimental support.

Unconscious Phobic Responses

A colleague and I reported an experiment designed to test this basic assertion (Öhman & Soares, 1994). We selected research participants who feared (above the 95th percentile in the distribution) snakes but not spiders (below the 50th percentile), or vice versa. Participants in the control group feared neither stimulus. In the experiment, participants were exposed to series of pictures of snakes and spiders, with pictures of flowers and mushrooms serving as controls, while skin conductance responses were measured. In the first series, presentations were effectively masked (see Öhman & Soares, 1993) by similar pictures that had been randomly cut to pieces, randomly reassembled, and rephotographed. Thus they were grossly similar to the target stimuli in colors and texture, but they lacked any recognizable central object. A pilot experiment using a forced-choice recognition procedure ascertained that both fearful and nonfearful participants consistently failed to identify the target with the masking parameters used. The masks interrupted presentation of the target stimuli after 30 milliseconds of exposure and remained on for 100 milliseconds during the masked presentation series. In the following series of presentations, the stimuli were presented unmasked. After these series, the participants rated the stimuli for arousal, valence, and control/dominance during separately presented masked and nonmasked rating series.

The upper panels of Figure 36.2 show skin conductance responses to masked (a) and nonmasked (b) presentations of the stimuli. It is evident that the fearful participants responded specifically to their feared stimulus, but did not differ from controls for the other stimulus categories, independently of masking. This enhanced responding to the feared stimulus cannot be attributed to conscious perception. Nevertheless, parallel data were obtained for all three rating dimensions, which suggests that some aspect of the masked stimulation became indirectly available to the conscious system (maybe through bodily feedback?). Thus the fearful participants rated themselves as more disliking, more aroused, and less in control when exposed to masked presentations of their feared stimulus.

The lower panels of Figure 36.2 show data from spontaneous skin conductance responses, a measure closely related to generalized anxiety (e.g., Lader, 1967). Again, the results show enhanced responding in the fearful groups compared to the control group, independently of masking. Thus unconscious exposure to the feared stimuli had effects on anxiety outlasting the specific responses to the stimuli. In other words, it is suggested that nonaware exposure to the feared stimulus resulted in generalized anxiety among fearful subjects.

Conditioning of Unconscious Effects

The data presented in Figure 36.2 provide strong support for the notion of anxiety as elicitable after only preliminary, preattentive, auto-

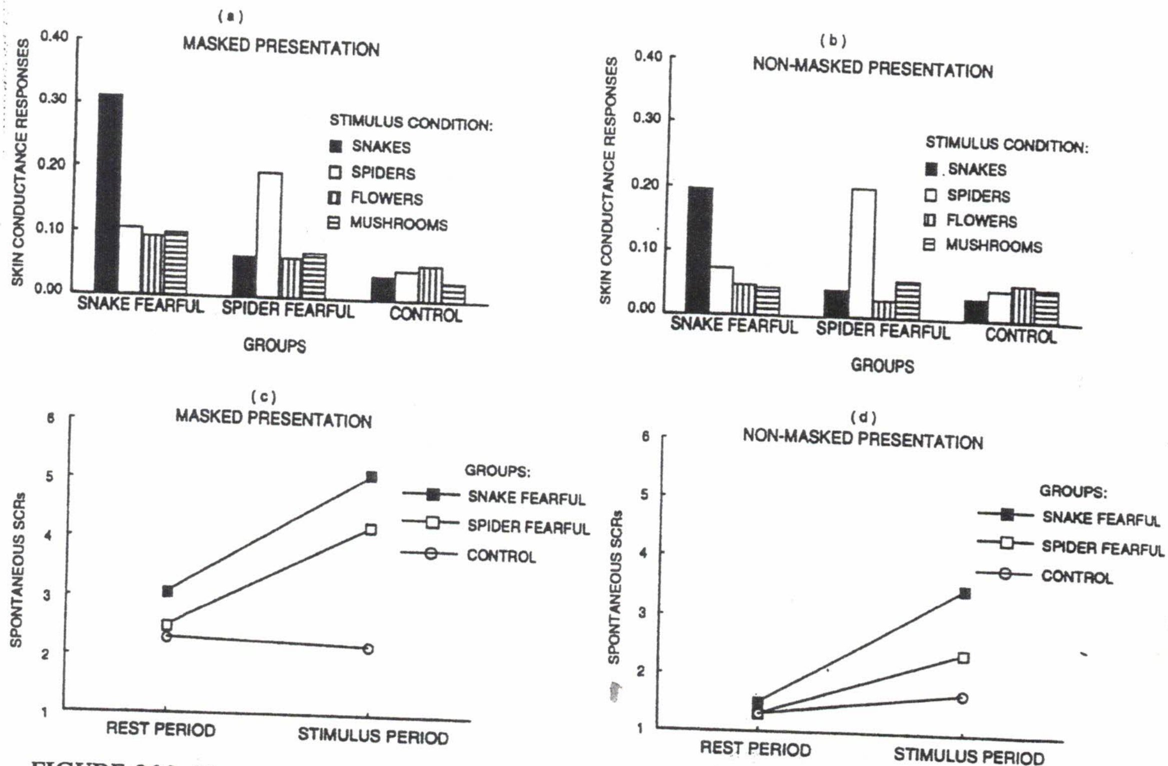

FIGURE 36.2. Upper panels (a and b) show skin conductance responses from snake-fearful, spider-fearful, and nonfearful controls to effectively masked (a) and nonmasked (b) presentations of pictures of snakes, spiders, flowers, and mushrooms. Fearful subjects showed elevated responding to their feared stimulus even if it was prevented from entering conscious perception by backward masking (a). Lower panels (c and d) show spontaneous skin conductance responses (SCRs) in the intervals between stimulation. Whereas controls did not change from rest during stimulation, the fearful subjects showed enhanced spontaneous responding, suggesting that they became anxious after both masked (c) and nonmasked (d) presentations of feared stimuli. Data from Öhman and Soares (1994).

matic, and unconscious analyses of the stimulus. One question raised by these findings concerns the origin of this effect: What is the mechanism whereby fearful subjects acquire these preattentively controlled responses to feared stimuli? Addressing this question, we (Öhman & Soares, 1993; Soares & Öhman, 1993a, 1993b) demonstrated that Pavlovian conditioning to unmasked presentations of fear-relevant stimuli (snakes and spiders) in nonfearful individuals resulted in conditioned skin conductance responses that survived backward masking. Conditioning to fear-irrelevant stimuli (flowers and mushrooms), on the other hand, resulted in more elusive responses that were abolished by masking. Similar data were obtained for another class of fear-relevant stimuli, angry faces, which were masked by neutral faces (Esteves, Dimberg, & Öhman, 1994; Parra, Esteves, Flykt, & Öhman, 1997). Furthermore, we (Esteves, Dimberg, Parra, & Öhman, 1994) reported that skin conductance responses could be conditioned to masked fear-relevant, but not to masked fear-irrelevant, stimuli. That is, after conditioning to masked angry or happy faces, subjects showed enhanced responding to subsequent nonmasked presentations of angry but not happy faces. Similarly, we (Öhman & Soares, 1998) demonstrated unconscious conditioning to masked snakes or spiders, but not to masked flowers or mushrooms. Thus not only could emotional responses be elicited to masked stimuli, but they could also be learned to such stimuli, provided that they were evolutionarily fear-relevant.

I have concluded (Öhman, 1992) that these types of preattentive effects are best interpreted in terms of the ability of biologically fear-relevant stimuli to directly activate physiological responses when such a stimulus automatically captures attention. Thus, consistent with LeDoux's (1990b) findings, the results can more plausibly be attributed to some gross and relatively simple features of the stimuli than to a complete analysis of their meaning. In further agreements with LeDoux's (1996) findings, we (Morris, Öhman, & Dolan, 1998), using brain imaging techniques, were able to demonstrate specific activation of the amygdala by masked conditioned angry faces in human subjects. Moreover, in further confirmation of LeDoux's (1996) tenets, we (Morris, Öhman, & Dolan, 1999) showed that nonconscious activation of the amygdala did not require cortical processing of the eliciting stimulus.

The theory, with its supporting database (Öhman & Soares, 1993, 1994, 1998; Soares & Öhman, 1993a, 1993b), provides a good account of animal phobias, which may be extended to social phobias (Dimberg & Öhman, 1996; Esteves, Dimberg, & Öhman, 1994; Esteves, Dimberg, Parra, & Öhman, 1994; Morris et al., 1998, 1999; Parra et al., 1997). Because phobic stimuli have direct, automatic access to the physiological arousal system, individuals with phobias perceive their bodies in full-swing responding when they consciously locate a phobic stimulus in the surroundings. Consequently, the fear is experienced as inevitable and uncontrollable. This account therefore provides a viable explanation for the "irrationality of phobias"—that is, the dissociation between conscious considerations and fear that is typical of phobias (APA, 1994).

Processing Biases in Anxiety

There is a large literature (see reviews by Mathews & MacLeod, 1994; Mogg & Bradley, 1998) on cognitive functions in anxiety, which documents that anxiety is associated with an attentional bias for focusing on threatening information in the surroundings. As Mathews (1990) has put it, "anxiety and worry are associated with an automatic processing bias, initiated prior to awareness, but serving to attract attention to environmental threat cues, and thus facilitating the acquisition of threatening information" (p. 462). This view is consistent with the functional perspective on anxiety presented above.

Selective Attention Bias in High-Anxiety Persons

To test this theory, Mathews and MacLeod (1986) had patients with generalized anxiety disorder and controls verbally "shadow" (read aloud) stories presented to one ear (the attended channel), while series of unconnected words were presented to the other ear (the rejected channel). At the same time, they were required to respond to visually presented probes by pressing a key. Some series of the words presented to the rejected channel involved threat (e.g., "injury," "disaster," "disease," "accident"), whereas others served as emotionally neutral controls, matched for word length and frequency of occurrence in the language. When questioned after the experiment, the subjects typically remained unaware that words had been presented in the rejected channel, and they did not perform above chance levels in forced-choice recognition tests on threat and control words. Nevertheless, anxious subjects slowed their reaction times to the probes when they occurred with threat as compared to nonthreat words in the rejected channel, whereas the controls did not discriminate these conditions. Similar data were reported by MacLeod, Mathews, and Tata (1986), using a dot probe task in which reaction time probes replaced threatening and nonthreatening words at different locations on a computer screen. According to their results, patients with generalized anxiety disorder showed faster reaction times when the probes replaced a threat word than when they replaced a neutral word (indicating attention to the former), whereas the opposite pattern was shown by the controls.

This basic result was replicated by MacLeod and Mathews (1988) for medical students high and low in trait anxiety. They were tested twice, in periods with or without examination stress, to examine the relative contributions of trait and state anxiety to the previously demonstrated attentional biases. Trait anxiety was related to the bias effect, but it also interacted with state anxiety. As examination time approached and state anxiety increased, high-trait-anxious subjects showed an increased bias to respond faster to examination-relevant threat words, whereas low-trait-anxious subjects showed an increased bias away from these words. Thus trait anxiety was associated with a general attentional bias in the direction of discovering threat, and with rising state anxiety this bias became more specifically geared toward threats associated with the anticipated stressful events. Low-trait-anxious subjects, on the other hand, in general showed a bias away from threat, and as their state anxiety rose before examination, this avoidance bias came to center on threat associated with the impending stress.

To examine the potential preattentive locus of the bias, MacLeod and Rutherford (1992) used a masked version of the Stroop color–word interference task. The latency of color naming of words was examined in participants high and low in trait anxiety, and on half of the trials, the words were masked by letter fragments of the same color. High-trait-anxious participants under examination stress were slower to color-name masked threatening words than were low-trait-anxious participants. On nonmasked trials, on the other hand, both groups showed a tendency specifically to avoid exam-relevant threat words. In other words, stressed highly anxious subjects showed a preattentive generalized bias toward threat, whereas at the conscious, strategic level, they tended specifically to avoid threat related to the source of stress (i.e., the examination). These results have been confirmed by Mogg, Bradley, Williams, and Mathews (1993) and Bradley, Mogg, Millar, and White (1995), using the masked Stroop paradigm, and by Mogg, Bradley, and Williams (1995), using the dot probe task of Mathews et al. (1986). These latter studies, however, examined patients with generalized anxiety disorder and reported an anxiety-driven bias for threat with both masked and nonmasked stimuli, which stand in contrast to the masked generalized bias for, and unmasked specific avoidance of, threatening words reported with highly anxious students by MacLeod and Rutherford (1992). Thus patients with generalized anxiety disorder may show both automatic and strategic vigilance for threat, whereas high-trait-anxious students, even though automatically biased for threat, may be able to cope with anxiety by strategic avoidance of threat stimuli once they become aware of the threatening word content.

Consistent with the data on psychophysiological responding to masked stimuli, this set of data indicates that mildly threatening stimuli capture attention, independently of its current focus. As attention is then automatically switched to the threat, there is competition for processing resources, which is manifested as impeded performance in ongoing, resource-demanding tasks. We (Öhman, Flykt, & Esteves, 1999) combined the two approaches by examining attentional selectivity for pictorial stimuli in a visual search paradigm. The task of the participants was to decide whether a discrepant category was present in a visual display composed of many pictures. Nonfearful participants were faster to locate a discrepant snake or spider among distractors of flowers and mushrooms than vice versa. Furthermore, finding the snake or spider was independent of its location in the display and the number of distractors, which suggests that they were automatically located. This was not true for finding flowers or mushrooms against backgrounds of snakes or spiders. The efficiency in searching for fear-relevant stimuli was further enhanced in subjects selected to be fearful of snakes or spiders. Thus the type of stimuli previously shown to automatically elicit psychophysiological responses also automatically captured attentional in a visual search task, and the attentional bias was enhanced when the target stimulus actually elicited fear.

The Role of Expectancy and Controlled Processing in Anxiety

So far, all the explanatory burden in the discussion of mechanisms of anxiety has been put on early, automatic, and preattentive processing. However, it is clear that these mechanisms are as insufficient to account for anxiety as they are to account for any emotional phenomenon. Theories of emotion have traditionally stressed the role of controlled processing in the generation of emotion (e.g., Lazarus, Kanner, & Folkman, 1980; Mandler, 1975, 1984; Schachter & Singer, 1962). With regard to anxiety, for example, it was noted in the discussion of the stimulus conditions for panic that bodily cues did not elicit attacks unless they were coupled with catastrophic interpretations (e.g., Clark, 1986, 1988). Thus, to approach a complete mechanism-oriented account of anxiety, it is necessary to consider controlled or strategic information processing.

No Panic with Explanation of Symptoms

Anecdotal data reported by Rapee (1986) illustrate the role of cognitions in panic. Although 80% of his patients with panic disorder reported a marked similarity between panic and the symptoms they experienced after hyperventilation, none of them panicked. When questioned, they attributed the lack of panic attacks to the fact that they knew what was causing the symptoms, and that they were in a safe place in case they should panic. Rapee, Mattick, and Murrell (1986) formally tested the hypothesis that a readily available explanation for bodily symptoms would save such patients from panic attacks. They compared patients with panic disorder and social phobia who were given an explanation or no explanation for the physiological effects of CO_2 inhalation. The groups did not differ in reported symptoms, regardless of the explanation given. However, patients with panic disorder who were given no explanation reported more intense symptoms, more intense panic, and more similarity to a "natural" panic attack. In addition, they reported a higher frequency of catastrophic thoughts (e.g., "I am going to die"). The explanation given had no effect on the patients with social phobia, who experienced little panic in any of the explanation conditions. Thus these results show that patients with panic disorder are more vulnerable to anxiety attacks than patients with social phobia when given CO_2 inhalation, and that a readily available explanation of the symptoms can abort the panic attacks. Relating to one of the classical issues in the study of emotion (see Schachter & Singer, 1962), the findings of Rapee et al. (1986) show that unexplained arousal is particularly effective in prompting negatively valenced emotional experience (see Marshall & Zimbardo, 1979; Maslach, 1979).

Effects of Expectations of Control over Symptoms

The role of cognitions in aborting panic was further elucidated by Sanderson, Rapee, and Barlow (1989). Again, CO_2 inhalation was used to induce panic in patients with panic disorder. All patients were instructed that they might experience a range of physical sensations, and a range of emotional states "from relaxation to anxiety," as a result of CO_2 administration. Furthermore, they were all informed that they would be able to adjust the CO_2 mixture by manipulating a dial *if* (and only if) a designated light was illuminated. However, they were urged to stay with the experimenter selected mixture, because that would facilitate assessment. In effect, the dial could not affect the CO_2 mixture, and none of the subjects tried to use it. The light was illuminated for half of the subjects, who thus were given an illusion of control. Subjects in this group reported a much stronger belief in control over CO_2 symptoms than did those in the no-illusion group. Ten subjects panicked during inhalation, 8 of whom were in the no-illusion group. Subjects in this group also reported more symptoms, higher intensity of symptoms, more catastrophic cognitions, and larger similarity of their response to naturally occurring panic attacks than the subjects in the illusion-of-control group. The quite dramatic effect of perceived control over symptoms reported in this study is in accordance with the prominent place of "fear of losing control" in the symptomatology of panic attacks (Barlow et al., 1985; Borden & Turner, 1989; Rapee et al., 1992). Furthermore, it highlights the importance of cognitions in the elicitation of panic.

The Role of Physiological Feedback in Panic

The data reviewed so far have primarily dealt with the panic-inhibitory effects of correct attribution of bodily symptoms to a more or less controllable external source. However, if misattribution of physiological symptoms to uncontrollable sources is focal for panic, then false beliefs about physiological activation should also prompt panic, regardless of the actual physiological changes (Valins, 1972). This hypothesis was tested by Ehlers, Margraf, Roth, Taylor, and Birbaumer (1988). They gave patients with panic disorder and controls feedback about their heart rate by having each heartbeat trigger a tone pip. After a baseline period without feedback, there was a true-feedback period during which the actual heart rate was fed back to the subjects. However, unknown to the participants, at the end of the true-feedback period, control of the feedback was transferred to the experimenter, who produced a 50-beat increase in heart rate over a 30-second period, mimicking the heart rate of an intense panic attack (Cohen, Barlow, & Blanchard, 1985; Lader & Mathews, 1970). When participants who realized that the feedback was false were excluded, the alleged increase in heart rate produced large increases in rated anxiety and excitement, as well as in skin conductance level, in patients but not in controls. Furthermore, whereas the controls showed a significant decrease in heart rate and blood pressure from true to false feedback, the patients showed increases. Thus, in support of the hypothesis, it appeared that the false feedback of a heart rate increase was sufficient to induce an anxiety attack in the patients, whereas the controls appeared more or less unaffected by this manipulation.

Bodily Sensations and Expectancy of Anxiety in Nonfearful Individuals

It appears from the data considered in this section that patients with anxiety disorders have a bias not only to attend to threat in the environment, but also a bias to expect some types of stimuli to signal impending doom. In accordance with this expectation, they appear to react with anxiety and panic to these cues. Because all the studies reviewed so far have used patients with panic disorder as subjects, one may wonder whether the effects reported are specific to them, or whether, with appropriate expectancy inductions, nonfearful individuals could be made to respond like these patients.

This possibility was examined by van der Molen, van den Hout, Vroemen, Lousberg, and Griez (1986). They exposed normal volunteers to lactate infusion or placebo in balanced order. Lactate infusion is a well-established method for experimentally inducing anxiety, and has been specifically related to panic (see Margraf et al., 1986b, and van den Hout, 1988, for reviews). Half of the participants were instructed that "the infusions might cause unpleasant bodily sensations similar to those experienced during periods of anxiety" and that "they might experience anxious effects" (van der Molen et al., 1986, p. 678). The other half of the participants were told that the infusion would evoke feelings of "pleasant tension, such as those experienced during sports, watching an exciting movie, etc." (van der Molen et al., 1986, p. 678). The participants rated their emotional state from –100 (very anxious tension) to +100 (very pleasant excitement). The instructions had no effect in the placebo condition, which resulted in neutral emotional ratings. With lactate infusion, however, participants instructed to expect aversive symptoms and anxiety rated their state as quite negative (–64.3), whereas the participants expecting positive affect showed variable ratings averaging out as emotionally neutral. Thus the results demonstrated that the interaction between bodily cues as induced by lactate infusion, and expectancy of aversive effects, was critical in inducing anxiety. Neither infusion nor instruction per se was sufficient to induce anxiety. This result provides persuasive support for the argument that expectations of negative affect and catastrophic consequences are critical determinants of anxiety and panic attacks, as claimed by cognitive theorists (e.g., Clark, 1986, 1988).

THEORETICAL INTEGRATION

To sum up, I have argued that anxiety originates in biologically evolved defense systems, which are responsible both for acute anxiety attacks (i.e., state anxiety) and, perhaps more indirectly, for more enduring and stable levels of anxiety (i.e., trait anxiety). These defenses are served by information-processing mechanisms centered on the determination of threat and operating at several loci of information processing. In this section, a somewhat speculative integration of the information-processing mechanisms is presented. It is pictorially represented in Figure 36.3.

Feature Detectors

Stimulus information (1 in Figure 36.3) reaches feature detectors, which provide a preliminary segregation of the stimulus before the information is passed on (2) to the significance evaluation system. The important part of this system for alarm/anxiety/fear is that some stimulus features may be directly connected to the arousal system. Thus it is assumed that the alarm reaction, which may eventually surface as an anxiety response, begins to be recruited immediately when the perceptual system encounters a sign of a survival-relevant stimulus.

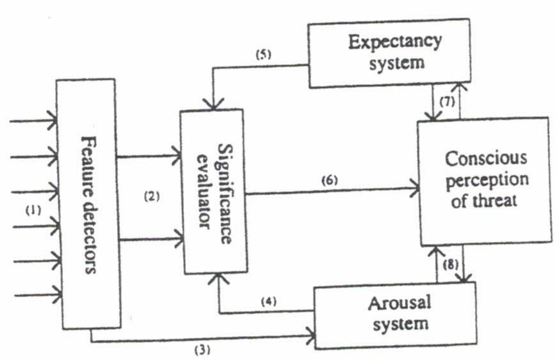

FIGURE 36.3. An information-processing model of the generation of anxiety. See text for explanations.

The feature detectors under discussion here are assumed to operate primarily on physical input. That is, they do not yet interact with memory to provide meaning to the stimulus; rather, they help in the preliminary segregation of the stimulus inflow and the direction of attention toward potentially relevant areas (e.g., Posner, 1980). Primary examples of stimulus features that may directly trigger the arousal system include high intensity and rapid rise times, provoking startle.

In attentional terms, this system may be set to discover potential threat in the environment by a filtering mechanism, giving priority to biologically important stimuli (see Öhman, 1992, for documentation of this point). Such stimuli, then, have the dual effect of immediately activating the arousal system (3) and being selected for preferential treatment by the significance evaluation system (2), which passes information directly on to the conscious perception system (6). The former of these routes may be strongly automatic, in the sense that a particular feature reflexively elicits autonomic responses. The route from feature detectors to the significance evaluator, however, may be subject to competition between stimulus features, which, as emphasized by Mathews and Mackintosh (1998), is an assumption that appears necessitated by the data on attentional bias in anxiety.

There are good data available to suggest that a direct link between features and autonomic responding is operating in phobias (Öhman & Soares, 1994) and that it may result from conditioning (Öhman & Soares, 1993; Esteves, Dimberg, & Öhman, 1994). It may also be speculated that this mechanism is operating in panic attacks. Barlow et al. (1985) reported clinical impressions suggesting that panic attacks are "usually associated with mild exercise, sexual relations, sudden temperature changes, stress, or other cues that alter physiological functioning in some discernible way, albeit outside the patient's awareness" (p. 327). If we accept Klein's (1993) argument that panic reflects the activation of an alarm system evolved to respond to suffocation hazard and prompting rapid escape from potentially deadly locations, it appears reasonable to presume that critical cues for this system would activate feature detectors that are sensitive to specific bodily stimuli. These stimuli either would result in unconditioned activation (e.g., increased CO_2 concentration in the inhaled air, lack of oxygen) or would reflect conditioned activation of alarm (e.g., heart rate increases). In both cases, however, the process would rapidly and automatically trigger the arousal system, independently of the person's awareness of the threat stimulus. A similar argument can perhaps be made for PTSD, where the eliciting event is so threatening that it provides traumatic conditioning to associated cues, which in this way may acquire power to activate the arousal system automatically. Few data are available to support the conditioning scenario postulated here for panic disorder or PTSD; however, it is clear that a severely traumatic event (such as a failure to get air) is an extremely efficient unconditioned stimulus for persistent conditioned fear responses (Sanderson, Laverty, & Campbell, 1963).

The Significance Evaluator

The significance evaluator automatically assesses stimuli for relevance. In contrast to the feature detectors, which are selective for input in terms of a stimulus set or a filtering mechanism, this system operates by a response set or a pigeonholing mechanism (Broadbent, 1970). That is to say, its selection is "top-down" or schema-driven, in the sense that it is set by the expectancy system (5) to look for particular categories of input. This implies that its operation is predicated on a full-meaning analysis of the stimuli. This is the locus of the bias to discover threat among persons high in anxiety (e.g., Mathews, 1990; Mogg & Bradley, 1998; see review above). Thus, as part of interrelated memory systems, memorial representations of moods (Bower, 1981) or emotional responses (Lang, 1984) may prime memory areas focused on threat; as a result, the expectancy system sets the significance evaluator to respond to threat words, for example. When threat words are discovered, the conscious perception system (6) is called, and this call may result in competition with other ongoing tasks, such as responding to visual probes (MacLeod et al., 1986; Mathews & MacLeod, 1986). Such competition implies that the significance evaluator requires cognitive resources, and thus that it is at least partly a controlled processing system. Nevertheless, it operates preattentively, without any necessary conscious access (Öhman, 1992; see also Spinks & Kramer, 1991). Thus it is assumed that there is controlled processing that is not consciously available.

The significance evaluator has been viewed as central to the elicitation of orienting responses (Bernstein & Taylor, 1979; Öhman, 1979). As the significance evaluator activates the conscious perception channel (through 6) so that the eliciting stimulus (e.g., the threat) is consciously perceived, an orienting response is elicited through activation of the arousal system (8). It should perhaps be explicitly noted that the view presented here, associating the orienting response with conscious perception (Öhman, 1992), represents a change from my previous view (Öhman, 1979), which assumed that orienting responses are activated by a direct link from the significance evaluator to the arousal system. The basic argument for this change is that backward masking, which blocks the target stimulus from entering conscious perception (i.e., path 6), also blocks skin conductance responses to conditioned stimuli, unless they are biologically fear-relevant. Biologically fear-relevant conditioned stimuli, on the other hand, are presumed to activate the arousal system through path 3, and thus skin conductance responses to such stimuli survive masking (see Öhman, 1992). The important implication for anxiety is that nonconscious discovery of potential threat through the significance evaluator does not result in activation of the arousal system unless it results in conscious perception of threat. Thus the enhanced skin conductance responses to relevant threat words presented in the rejected channel of a shadowing task for subjects with obsessive–compulsive disorder (Foa & McNally, 1986) should be attributed to switches of attention between channels (Dawson & Schell, 1982; Trandel & McNally, 1987). Similarly, enhanced skin conductance responses to relevant threat words presented against a noisy background to veterans with PTSD should be attributed to conscious perception (McNally et al., 1987).

The Arousal System

Although the significance evaluator is assumed to have no effect, or only a weak effect, on the arousal system, this latter system is assumed to be able to "tune up" the significance evaluator (4). Thus increased arousal is assumed to enhance the biases of the significance evaluator, perhaps in a way analogous to the multiplicative relation between drive and habit in Hullian learning theory. This assumption explains the effect of state anxiety on attentional bias (e.g., MacLeod & Mathews, 1988; MacLeod & Rutherford, 1992).

The arousal system also provides critical input to the conscious perception system, which has been taken as the distinctive feature of emotional experience (Mandler, 1975). For example, the studies by Ehlers et al. (1988) and Pauli et al. (1991) demonstrated the power of such perceived input to generate anxiety in anxiety-prone individuals. However, it is important to note that the relationship between the arousal system and the conscious perception system is bidirectional. Thus, as threat and danger are consciously perceived, the arousal system is recruited to provide metabolic support for the more or less vigorous coping actions that may become necessary.

Although the arousal system is presented as a unitary system here, this is of course a gross oversimplification. The core of the system is Cannon's (1929) emergency reaction—that is, a sympatho-adrenal mobilization of bodily resources for vigorous action—but the character of the resulting physiological response is modulated by stimulus factors, available action alternatives, and the particular action chosen, as well as by characteristics of the individual (see Öhman, Hamm, & Hugdahl, 2000).

The Expectancy System

The expectancy system relies on the organization of emotion in memory. Following Lang (1984), it is assumed that memory for emotional episodes can be represented by interconnected nodes comprising stimulus, response, and meaning information. Such networks can be activated by matching input information, but because nodes are assumed to coactivate one another within the network, a partial match involving only a few nodes is sufficient to put the system into an activated state. However, the more complete the match, the stronger the activation (Lang, 1984). When activated, the system can be viewed as biasing the significance evaluator to respond to information matching active memory nodes (5). Furthermore, such matching provides information for the conscious perception system, which in turn keeps the memory foundation of the expectancy system in continual activation—maintaining, as it were, the bias to discover threat. In agreement with Mathews (1990), it is assumed that this biasing of the significance evaluator occurs at a nonconscious level of information processing.

However, the expectancy system has a dual role in generating fear and anxiety. Not only does it bias the processing of incoming information, but it also provides the context for the interpretation of inputs to the conscious perception system (see Mandler, 1975, 1984). At this level, the influence of the expectancy system occurs at a consciously reportable level, where it is more appropriate to use the term "expectancy" than in the case of biasing the significance evaluator. Thus interpretation of input from the significance evaluator (5) and the arousal system (8) by the conscious perception system occurs in continuous interaction with the expectancy system and its associated memory. This is the basis for the effects of expectancies on panic that have been discussed previously.

Perceived Threat and Coping

The conscious perception system is merely an aspect of a much broader system, whether we call it the "mind" elucidated as the "cognitive–interpretive system" (Mandler, 1975), "consciousness" (Posner, 1978), "control processing" (Schneider et al., 1984), or the "central capacity-limited channel" (Öhman, 1979). For present purposes this system has two central functions: (1) to integrate information input from the arousal system, the significance evaluator, and the expectancy system; and (2) to select an action alternative to cope with the perceived threat. When Epstein's (1972) distinction between anxiety and fear is adhered to, the latter function becomes critical for deciding the emotional effect of the perceived threat. If its nature is such that avoidance or escape provides successful coping with the threat, the result is fear. If there is no such possibility, or if attempted escape is interfered with, the result is anxiety (Epstein, 1972).

CONCLUDING DISCUSSION

To recapitulate, it has been argued that responses of fear and anxiety originate in an alarm system shaped by evolution to protect creatures from impending danger. This system is biased to discover threat, and it results in a sympathetically dominated response as a support of potential flight or fight. This response system can be triggered from three different levels of information processing, the first two of which are inaccessible to introspection. The first level concerns a direct link to an arousal system from elementary feature detectors geared to respond to biologically relevant threats. Thus the arousal system becomes collaterally and automatically activated with the activation of further information-processing stages, whose functioning may be influenced by the arousal. The second level concerns a schema-driven nonconscious bias to discover threat in the environment, which delivers information to conscious perception, but has no effect or only a weak effect on physiological arousal. The third level concerns the direct effect of expectancy and physiological arousal on the cognitive–interpretive activity resulting in perceived threat. In this concluding section of the chapter, some implications of this view of anxiety are discussed.

The Nature of the Unconscious Effects

Freud (e.g., 1900/1953), of course, believed that anxiety has an unconscious origin, residing in the interaction between bodily and instinctual energies on the one hand, and various psychological defense mechanisms on the other. Similarly, according to the present scheme, unconscious activation of bodily systems in interaction with an interpretive conscious system plays a pivotal role in the generation of anxiety. Indeed, the correspondence with classic psychodynamic notions can be pushed a step further by noting that the present scheme, in a way, does not operate with one but with two different types of unconscious. Freud distinguished between what might be termed a "drive unconscious" and a "repressed unconscious" (see Power & Brewin, 1991), which in some respects would correspond to the unconscious effects of the feature detectors and the significance evaluator, respectively, in the present model. Furthermore, in some other respects the former would correspond to the "collective unconscious" postulated by Jung (1953) to encompass the cumulative experience of the human species. In the model (Figure 36.3), this role is played by feature detectors, which have been shaped to respond particularly strongly to features associated with recurrent threats to well-being in the evolution of humankind. Elsewhere (Öhman, 1986), I have suggested that reptiles provided an archaic prototype for threats emanating from predation pressure, and that this may explain the human tendency to equip the embodiments of evil with bestial features. Thus Jung's notion of "archetypes" can in fact be reinterpreted in terms of biological preparedness (e.g., Seligman, 1971).

The other mechanism of nonconscious bias for responding to threat, residing in the significance evaluator and the expectancy system, suggests some of the Freudian notions of the unconscious. Because this bias represents schema-driven effects dependent on memorial organization (e.g., Lang, 1984), it will reflect the individual's unique personal experience. Depending on the history of the individual, he or she may respond to some potential threat cues rather than others. Furthermore, what is extremely threatening to one individual may be completely innocuous to another, because the corresponding memorial node may not be connected to nodes in memory structures related to threat. Thus effects similar to those of the classical Freudian defenses may be interpretable in these terms. Furthermore, one may even argue that the schema-driven unconscious threat-biasing system may sometimes be pitted against the feature-driven detection system in a way strongly reminiscent of the interactions between defenses and drives in psychoanalytic theory. For example, if bodily cues activate the arousal system, but the significance evaluator is biased against responding to these cues in terms of threat, then something similar to the phenomenon seen in patients with "panic without fear" (Kushner & Beitman, 1990) should result. However, whether this should be viewed as anxiety counteracted by defenses, or simply as lack of anxiety, appears a moot point. The important insight here is rather that phenomena similar to those described by psychoanalysts are readily interpretable in terms of the current model (see Power & Brewin, 1991, for further discussion of the communality between traditional psychoanalytic theories and contemporary cognitive science theories). These interpretations, furthermore, are preferable to the psychoanalytic ones because they are backed up by a scientific literature based on rigorous theorizing and controlled data, rather than based on anecdotal observations from uncontrolled case studies (see Grünbaum, 1984).

The Relationship between Fear and Anxiety

The model depicted in Figure 36.3 implies that there are in effect two types of anxiety, which both differ from fear. Following Lang (1984), emotions can be viewed as action sets—as readinesses to respond in particular ways. Fear, then, is viewed as an emotional response related to avoidance and escape. Although such responses may be primed by the feature detectors and the significance evaluator, which may recruit metabolic support for vigorous action, the eventual overt responses are taken to occur after conscious perception of the threat. When such responses are blocked, fear is changed into anxiety (Epstein, 1972). However, if anxiety is construed as "unresolved fear" or "undirected arousal" related to perceived threat (Epstein, 1972), then the model implies that there is a more basic type of anxiety than the one resulting when avoidance motives become frustrated. This type of anxiety results from the unconscious input to the conscious perception system from the significance evaluator and the arousal system. Because the source of this input is not necessarily available to consciousness, the resulting state of undirected arousal is experienced as anxiety, or perhaps more precisely as "undirected alarm." The person knows that something is wrong, but cannot pinpoint any clear reason for it. Anxiety in this sense, then, is entirely dependent on the unconscious mechanisms, whereas the anxiety resulting from interference with avoidance is more readily appraised at the conscious level as originating in the external world or in personal shortcomings.

The "alarm" or "primary" anxiety may be channeled or "resolved" into fear, if escape is selected as the action option after a complete conscious and controlled processing of the stimulus situation. Normally, of course, the eliciting stimulus is consciously perceived simultaneously with the arousal of anxiety, as attention is directed to the preattentively located threat. It is only when the attention shift fails to locate the stimulus that preattentively elicited unconscious anxiety is left in the system without any apparent stimulus for its explanation. This may occur when the stimulus is very faint and thus fails to be perceived; when the preattentive mechanisms falsely locate a threat that is not confirmed by controlled processing; or when several more or less simultaneous stimuli (e.g., emanating from the body) mask one another. In any of these cases, an emotional state perhaps best characterized as "anxious alarm" should be the experienced result, and this state should be clearly different from anxiety generated by failed escape or avoidance.

Implications for Anxiety Disorders

The model depicted in Figure 36.3 permits the various anxiety disorders to be viewed as resulting from different emphases within the same information-processing structures.

As already noted, phobias and panic disorder are taken as resulting from the automatic activation of the arousal system by specific features located by the feature detectors. This activation provides a surge of physiological arousal, cues from which become available to the conscious perception system. Therefore, these two types of disorders should be similar in the sense that they both reflect increases in sympathetically mediated arousal. However, whereas the information reaching the conscious perception system from the arousal system is quite similar in phobias and panic disorder, the information arriving via the significance evaluator is radically different. In the case of a phobia, the source of the physiological arousal is attributed to some factor in the external world; in the case of panic, the arousal is attributed to an enemy from within. As everyone who has considered security management knows, the former case is much easier to handle than the latter. An external enemy can be met with barricades, attack, or defensive withdrawal, depending on situational factors and the relative balance of power between the threatened and the threatener. In phobias, this balance is typically interpreted as supporting flight. However, an enemy within has crossed the defensive barricades, which makes the impending danger acute. Flight is not an option, and the risks of being overwhelmed and of capitulation become acute. Therefore, catastrophic interpretations of the situation are readily invited. In this way, both the similarities and the differences in symptomatology between phobias and panic disorder can be accounted for.

If Klein's (1993) interpretation of panic as the result of an alarm system responding to suffocation threat is accepted, then a route to the understanding of agoraphobia is opened. In agoraphobia the suffocation alarm may be conditioned to environmental cues, which then become avoided. However, if the alarm response rather is conditioned to bodily cues (such as heart rate increases), then the person may end up with apparently spontaneous panic attacks, without obvious situational triggers.

In PTSD, there is an original trauma that totally recruits the individual's defense responses, often for quite protracted periods of time and at overwhelming intensities (e.g., in combat). As a result, cues may be conditioned to recruit the arousal system automatically, as in phobia and panic. In PTSD, however, the subsequent stage—cognitive preoccupation with the trauma, partly mediated through the expectancy system and the significance evaluator—appears to take on a more prominent role than in phobias and panic disorder, leading to physiological activation not only automatically but also through conscious mediation (via worries and ruminations).

Generalized anxiety disorder, finally, appears to lack arousal activation via the feature detectors; it seems to be primarily driven by the expectancy system and the bias to discover threats (Rapee, 1991). The physiological effects that are needed to turn the worry into anxiety (Mathews, 1990) are likely to be recruited through activation of the arousal system from the cognitive perception system. Thus in this disorder the expectancy–significance–perception loop appears to play the primary role and the nonconscious activation of arousal a secondary role in the problem.

The perspective provided here views phobias and panic disorders as physiologically driven, and generalized anxiety disorder as cognitively driven, with PTSD at a somewhat intermediate position between the two groups. Thus, within a unitary theoretical frame, it is possible to deal quite effectively with important aspects of the different anxiety disorders. However, whereas this account appears quite successful in dealing with the symptomatology of the disorders, it is relatively silent on the issue of etiology. Conditioning is given a role in several disorders—and, elaborated as biologically prepared learning, it may be decisive for phobias (Öhman et al., 1985)—but there is at present no information in the model to suggest why some persons develop panic disorder and others generalized anxiety disorder. In particular, the origin of the cognitively driven ruminations in generalized anxiety disorder remains a mystery.

Anxiety, Emotion, and Cognition

If the interaction between physiological cues and cognitive–interpretive activity is taken as the hallmark of emotional phenomena (e.g., James, 1884; Mandler, 1975), then fear and anxiety as described in this chapter are prima facie emotional phenomena. It has been documented that bodily cues provide some of the most important experienced symptoms in fear and anxiety, and that measurable physiological responses are prominent correlates of fear and anxiety both in the laboratory and when ambulatorily monitored in everyday life. There are good data suggesting that perceived bodily changes are critical to anxiety attacks (e.g., Pauli et al., 1991), but it is equally clear that such bodily changes are neither necessary nor sufficient for anxiety to be experienced. The anxiety is typically evoked in particular types of situations, the nature of which is such that an evolutionary origin appears a quite straightforward interpretation of their potency.

With regard to theory, an interactional perspective stresses the inextricable interplay among physiological activation, cognitive processes, and emotional responding from the very moment an effective stimulus makes contact with the relevant sensory organ. Thus it is clear that some emotional effects occur immediately upon presentation of an effective stimulus (LeDoux, 1990b, 1996; Öhman & Soares, 1994), providing some justification for the claim that "affects precedes inference" (Zajonc, 1980). However, it is equally clear that cognitions stemming from nonconscious biases play pervasive roles in the interpretation of threats (e.g., Mathews, 1990), in the volitional appraisal of the stimulus, and in deliberations about potential response alternatives. Thus, as a final point, the literature reviewed in this chapter is taken to suggest that it is hardly meaningful to ask oneself whether cognition is necessary for emotion. We appear to have reached a stage in the knowledge of fear and anxiety at which the meaningful question is how the interaction between emotion and cognition occurs, and some tentative answers to this fundamental question may already be at hand.

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REFERENCES

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arrindell, W. A., Pickersgill, M. J., Merckelbach, H., Ardon, M. A., & Cornet, F. C. (1991). Phobic dimensions: III. Factor analytic approaches to the study of common phobic fears: An updated review of findings obtained with adult subjects. *Advances in Behaviour Research and Therapy, 13*, 73–130.

Barlow, D. H. (1988). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press.

Barlow, D. H., Vermilyea, J., Blanchard, E. B., Vermilyea, B. B., Di Nardo, P. A., & Cerny, J. A. (1985). The phenomenon of panic. *Journal of Abnormal Psychology, 94*, 320–328.

Bernstein, A. S., & Taylor, K. W. (1979). The interaction of stimulus information with potential stimulus significance in eliciting the skin conductance orienting response. In H. D. Kimmel, E. H. van Olst, & J. F. Orlebeke (Eds.), *The orienting reflex in humans*. Hillsdale, NJ: Erlbaum.

Borden, J. W., & Turner, S. M. (1989). Is panic a unique emotional experience? *Behaviour Research and Therapy, 27*, 263–268.

Bower, G. H. (1981). Mood and memory. *American Psychologist, 36*, 129–148.

Bradley, B. P., Mogg, K., Millar, N., & White, J. (1995). Selective processing of negative information: Effects of clinical anxiety, concurrent depression, and awareness. *Journal of Abnormal Psychology, 104*, 532–536.

Broadbent, D. E. (1970). Stimulus set and response set: Two kinds of selective attention. In D. M. Mostofsky (Ed.), *Attention: Contemporary theory and analysis*. New York: Appleton-Century-Crofts.

Buss, A. H. (1962). Two anxiety factors in psychiatric patients. *Journal of Abnormal and Social Psychology, 65*, 426–427.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage*. New York: Appleton-Century-Crofts.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*, 461–470.

Clark, D. M. (1988). A cognitive model of panic attacks. In S. Rachman & J. D. Maser (Eds.), *Panic: Psychological perspectives*. Hillsdale, NJ: Erlbaum.

Cohen, A. S., Barlow, D. H., & Blanchard, E. B. (1985). Psychophysiology of relaxation-associated panic attacks. *Journal of Abnormal Psychology, 94*, 96–101.

Dawson, M. E., & Schell, A. M. (1982). Electrodermal responses to attended and nonattended significant stimuli during dichotic listening. *Journal of Experimental Psychology: Human Perception and Performance, 8*, 315–324.

Dimberg, U., & Öhman, A. (1996). Behold the wrath: Psychophysiological responses to facial stimuli. *Motivation and Emotion, 20*, 149–182.

Ehlers, A. (1993). Interoception and panic disorder. *Advances in Behaviour Research and Therapy, 15*, 3–21.

Ehlers, A., Margraf, J., Roth, W. T., Taylor, C. B., & Birbaumer, N. (1988). Anxiety induced by false heart rate feedback in patients with panic disorder. *Behaviour Research and Therapy, 26*, 1–11.

Epstein, S. (1972). The nature of anxiety with emphasis upon its relationship to expectancy. In C. D. Spielberger (Ed.), *Anxiety: Current trends in theory and research* (Vol. 2). New York: Academic Press.

Esteves, F., Dimberg, U., & Öhman, A. (1994). Automatically elicited fear: Conditioned skin conductance responses to masked facial expressions. *Cognition and Emotion, 8*, 393–413.

Esteves, F., Dimberg, U., Parra, C., & Öhman, A. (1994). Nonconscious associative learning: Pavlovian conditioning of skin conductance responses to masked fear-relevant facial stimuli. *Psychophysiology, 31*, 375–385.

Fenz, W. D., & Epstein, S. (1965). Manifest anxiety: Unifactorial or multifactorial composition. *Perceptual and Motor Skills, 20*, 773–780.

Foa, E. B., & McNally, R. J. (1986). Sensitivity to feared stimuli in obsessive–compulsives: A dichotic listening analysis. *Cognitive Therapy and Research, 10*, 477–485.

Freedman, R. R., Ianni, P., Ettedgui, E., & Puthezhath, N. (1985). Ambulatory monitoring of panic disorder. *Archives of General Psychiatry, 42*, 244–255.

Freud, S. (1953). The interpretation of dreams. In J. Strachey (Ed. & Trans.) *The standard edition of the complete psychological works of Sigmund Freud* (Vols. 4 and 5). London: Hogarth Press. (Original work published 1900)

Grünbaum, A. (1984). *The foundation of psychoanalysis: A philosophical critique*. Berkeley: University of California Press.

Hibbert, G. A. (1984). Ideational components of anxiety: Their origin and content. *British Journal of Psychiatry, 144*, 618–624.

Holender, D. (1986). Semantic activation without conscious identification in dichotic listening, parafoveal vision, and visual masking: A survey and appraisal. *Behavioral and Brain Sciences, 9*, 1–66.

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

Jung, C. G. (1953). Two essays on analytical psychology. In C. G. Jung, *Collected works* (Vol. 7). New York: Pantheon.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). The genetic epidemiology of phobias in women: The interrelationship of agoraphobia, social phobia, situational phobia and simple phobia. *Archives of General Psychiatry, 49*, 273–281.

Kipling, R. (1983). *The second jungle book*. London: Macmillan. (Original work published 1895)

Klein, D. F. (1981). Anxiety reconceptualized. In D. F. Klein & J. Rabkin (Eds.), *Anxiety: New research and changing concepts*. New York: Raven Press.

Klein, D. F. (1993). False suffocation alarms, spontaneous panics, and related conditions. *Archives of General Psychiatry, 50*, 306–317.

Kushner, M. G., & Beitman, B. D. (1990). Panic attacks without fear: An overview. *Behaviour Research and Therapy, 28*, 469–479.

Lader, M. H. (1967). Palmar skin conductance measures in anxiety and phobic states. *Journal of Psychosomatic Research, 11*, 271–281.

Lader, M., & Marks, I. (1973). *Clinical anxiety*. London: Heinemann.

Lader, M., & Mathews, A. (1970). Physiological changes during spontaneous panic attacks. *Journal of Psychosomatic Research, 14*, 377–382.

Lang, P. J. (1968). Fear reduction and fear behavior: Problems in treating a construct. In J. M. Shlien (Ed.), *Research in psychotherapy* (Vol. 3). Washington, DC: American Psychological Association.

Lang, P. J. (1978). Anxiety: Toward a psychophysiological definition. In H. S. Akiskal & W. L. Webb (Eds.), *Psychiatric diagnosis: Explorations of biological predictors*. New York: Spectrum.

Lang, P. J. (1984). Cognition in emotion: Concept and action. In C. E. Izard, J. Kagan, & R. B. Zajonc (Eds.), *Emotions, cognition, and behavior*. New York: Cambridge University Press.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang, R. F. Simons, & M. T. Balaban (Eds.), *Attention and orienting: Sensory and motivational processes*. Mahwah, NJ: Erlbaum.

Lazarus, R. S., Kanner, A. D., & Folkman, S. (1980). Emotions: A cognitive–phenomenological analysis. In R. Plutchik & H. Kellerman (Eds.), *Emotion: Theory, research, and experience. Vol. 1. Theories of emotion*. New York: Academic Press.

LeDoux, J. E. (1989). Cognitive–emotional interactions in the brain. *Cognition and Emotion, 3*, 267–289.

LeDoux, J. E. (1990a). Fear pathways in the brain: Implications for a theory of the emotional brain. In P. F. Brain, S. Parmigiani, R. J. Blanchard, & D. Mainardi (Eds.), *Fear and defence*. London: Harwood.

LeDoux, J. E. (1990b). Information flow from sensation to emotion: Plasticity in the neural computation of stimulus value. In M. Gabriel & J. Moore (Eds.), *Learning and computational neuroscience: Foundations of adaptive networks*. Cambridge, MA: MIT Press.

LeDoux, J. E. (1996). *The emotional brain*. New York: Simon & Schuster.

MacLeod, C., & Mathews, A. (1988). Anxiety and the allocation of attention to threat. *Quarterly Journal of Experimental Psychology, 40A*, 653–670.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*, 15–20.

MacLeod, C., & Rutherford, E. M. (1992). Anxiety and the selective processing of emotional information: Mediating roles of awareness, trait and state variables, and personal relevance of stimulus materials. *Behaviour Research and Therapy, 30*, 479–491.

Mandler, G. (1975). *Mind and emotion*. New York: Wiley.

Mandler, G. (1984). *Mind and body: Psychology of emotion and stress*. New York: Norton.

Marcel, A. (1983). Conscious and unconscious perception: An approach to the relations between phenomenal experience and perceptual processes. *Cognitive Psychology, 15*, 238–300.

Margraf, J., Ehlers, A., & Roth, W. T. (1986a). Biological models of panic disorder and agoraphobia: A review. *Behaviour Research and Therapy, 24*, 553–567.

Margraf, J., Ehlers, A., & Roth, W. T. (1986b). Sodium lactate infusions and panic attacks: A review and critique. *Psychosomatic Medicine, 48*, 23–51.

Marshall, G. D., & Zimbardo, P. G. (1979). Affective consequences of inadequately explained physiological arousal. *Journal of Personality and Social Psychology, 37*, 970–988.

Maslach, C. (1979). Negative emotional biasing of un-

explained arousal. *Journal of Personality and Social Psychology, 37*, 953–969.
Mason, J. W. (1968). Overall hormonal balance as a key to endocrine functions. *Psychosomatic Medicine, 30*, 791–808.
Mathews, A. (1990). Why worry?: The cognitive function of anxiety. *Behaviour Research and Therapy, 28*, 455–468.
Mathews, A., & Mackintosh, B. (1998). A cognitive model of selective processing in anxiety. *Cognitive Therapy and Research, 22*, 539–560.
Mathews, A., & MacLeod, C. (1986). Discrimination of threat cues without awareness in anxiety states. *Journal of Abnormal Psychology, 95*, 131–138.
Mathews, A., & MacLeod, C. (1994). Cognitive approaches to emotion and emotional disorders. *Annual Review of Psychology, 45*, 25–50.
McNally, R. J. (1987). Preparedness and phobias: A review. *Psychological Bulletin, 101*, 283–303.
McNally, R. J., Luedke, D. L., Besyner, J. K., Peterson, R., Bohm, K., & Lips, O. J. (1987). Sensitivity to stress-relevant stimuli in posttraumatic stress disorder. *Journal of Anxiety Disorders, 1*, 105–116.
Mogg, K., & Bradley, B. P. (1998). A cognitive–motivational analysis of anxiety. *Behaviour Research and Therapy, 36*, 809–848.
Mogg, K., Bradley, B. P., & Williams, R. (1995). Attentional bias in anxiety and depression: The role of awareness. *British Journal of Clinical Psychology, 34*, 17–36.
Mogg, K., Bradley, B. P., Williams, R., & Mathews, A. (1993). Subliminal processing of emotional information in anxiety and depression. *Journal of Abnormal Psychology, 102*, 304–311.
Morris, J. S., Öhman, A., & Dolan, R. J. (1998). Conscious and unconscious emotional learning in the human amygdala. *Nature, 393*, 467–470.
Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating "unseen" fear. *Proceedings of the National Academy of Sciences, 96*, 1680–1685.
Nesse, R. M. (1987). An evolutionary perspective on panic disorder and agoraphobia. *Ethology and Sociobiology, 8*, 73S–83S.
Nesse, R. M., Cameron, O. G., Curtis, G. C., McCann, D. S., & Huber-Smith, M. J. (1984). Adrenergic function in panic anxiety patients. *Archives of General Psychiatry, 41*, 320–332.
Öhman, A. (1979). The orienting response, attention, and learning: An information processing perspective. In H. D. Kimmel, E. H. van Olst, & J. F. Orlebeke (Eds.), *The orienting reflex in humans*. Hillsdale, NJ: Erlbaum.
Öhman, A. (1986). Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analyses of emotion. *Psychophysiology, 23*, 123–145.
Öhman, A. (1992). Orienting and attention: Preferred preattentive processing of potentially phobic stimuli. In B. A. Campbell, H. Haynes, & R. Richardson (Eds.), *Attention and information processing in infants and adults: Perspectives from human and animal research*. Hillsdale, NJ: Erlbaum.
Öhman, A. (1993). Stimulus prepotency and fear: Data and theory. In N. Birbaumer & A. Öhman (Eds.), *The organization of emotion: Cognitive, clinical and psychophysiological perspectives*. Toronto: Hogrefe.
Öhman, A. (1996). Preferential preattentive processing of threat in anxiety: Preparedness and attentional biases. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders*. New York: Guilford Press.
Öhman, A. (1999). Distinguishing unconscious from conscious emotional processes: Methodological considerations and theoretical implications. In T. Dalgleish & M. Power (Eds.), *Handbook of cognition and emotion*. Chichester, England: Wiley.
Öhman, A., Dimberg, U., & Öst, L.-G. (1985). Animal and social phobias: Biological constraints on learned fear responses. In S. Reiss & R. R. Bootzin (Eds.), *Theoretical issues in behavior therapy*. New York: Academic Press.
Öhman, A., Eriksson, A., & Olofsson, C. (1975). One-trial learning and superior resistance to extinction of autonomic responses conditioned to potentially phobic stimuli. *Journal of Comparative and Physiological Psychology, 88*, 619–627.
Öhman, A., Flykt, A., & Esteves, F. (1999). *Emotion drives attention: Beware! There are snakes in the grass!* Manuscript submitted for publication.
Öhman, A., Flykt, A., & Lundqvist, D. (2000). Unconscious emotion: Evolutionary perspectives, psychophysiological data, and neuropsychological mechanisms. In R. Lane & L. Nadel (Eds.), *The cognitive neuroscience of emotion* (pp. 296–327). New York: Oxford University Press.
Öhman, A., Hamm, A. O., & Hugdahl, K. (2000). Cognition and the autonomic nervous system: Orienting, anticipation, and conditioning. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 533–575). New York: Cambridge University Press.
Öhman, A., & Soares, J. J. F. (1993). On the automaticity of phobic fear: Conditioned skin conductance responses to masked fear-relevant stimuli. *Journal of Abnormal Psychology, 102*, 121–132.
Öhman, A., & Soares, J. J. F. (1994). "Unconscious anxiety": Phobic responses to masked stimuli. *Journal of Abnormal Psychology, 103*, 231–240.
Öhman, A., & Soares, J. J. F. (1998). Emotional conditioning to masked stimuli: Expectancies for aversive outcomes following nonrecognized fear-relevant stimuli. *Journal of Experimental Psychology: General, 127*, 69–82.
Parra, C., Esteves, F., Flykt, A., & Öhman, A. (1997). Pavlovian conditioning to social stimuli: Backward masking and the dissociation of implicit and explicit cognitive processes. *European Psychologist, 2*, 106–117.
Pauli, P., Marquardt, C., Hartl, L., Nutzinger, D. O., H"lzl, R., & Strian, F. (1991). Anxiety induced by cardiac perceptions in patients with panic attacks: A field study. *Behaviour Research and Therapy, 29*, 137–145.
Posner, M. I. (1978). *Chronometric explorations of mind*. Hillsdale, NJ: Erlbaum.
Posner, M. I. (1980). Orienting and attention. *Quarterly Journal of Experimental Psychology, 32*, 3–25.

Power, M., & Brewin, C. R. (1991). From Freud to cognitive science: A contemporary account of the unconscious. *British Journal of Clinical Psychology, 30*, 289–310.

Rapee, R. M. (1986). Differential response to hyperventilation in panic disorder and generalized anxiety disorder. *Journal of Abnormal Psychology, 95*, 24–28

Rapee, R. M. (1991). Generalized anxiety disorder: A review of clinical features and theoretical concepts. *Clinical Psychology Review, 11*, 419–440.

Rapee, R. M. (1993). Psychological factors in panic disorder. *Advances in Behaviour Research and Therapy, 15*, 85–102.

Rapee, R. M. (Ed.). (1996). *Current controversies in the anxiety disorders*. New York: Guilford Press.

Rapee, R. M., Mattick, R., & Murrell, E. (1986). Cognitive mediation in the affective component of spontaneous panic attacks. *Journal of Behavior Therapy and Experimental Psychiatry, 17*, 245–253.

Rapee, R. M., Sanderson, W. C., McCauley, P. A., & Di Nardo, P. A. (1992). Differences in reported symptom profile between panic disorder and other DSM-III-R anxiety disorders. *Behaviour Research and Therapy, 30*, 45–52.

Sanderson, R. S., Laverty, R. S., & Campbell, D. (1963). Traumatic conditioned responses acquired during respiratory paralysis. *Nature, 196*, 1235–1236.

Sanderson, W. C., Rapee, R. M., & Barlow, D. H. (1989). The influence of an illusion of control on panic attacks induced via inhalation of 5. 5% carbon dioxide-enriched air. *Archives of General Psychiatry, 46*, 157–162.

Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review, 69*, 379–399.

Schalling, D., Cronholm, B., & Åsberg. M. (1975). Components of state and trait anxiety as related to personality and arousal. In L. Levi (Ed.), *Emotions: Their parameters and measurement.* New York: Raven Press.

Schneider, W., Dumais, S. T., & Shiffrin, R. M. (1984). Automatic and control processing and attention. In R. Parasuraman & D. R. Davies (Eds.), *Varieties of attention*. Orlando, FL: Academic Press.

Seligman, M. E. P. (1971). Phobias and preparedness. *Behavior Therapy. 2*, 307–320.

Sheehan, D. V. (1982). Panic attacks and phobias. *New England Journal of Medicine, 307*, 156–158.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84*, 127–190.

Soares, J. J. F., & Öhman, A. (1993a). Backward masking and skin conductance responses after conditioning to non-feared but fear-relevant stimuli in fearful subjects. *Psychophysiology, 30*, 460–466.

Soares, J. J. F., & Öhman, A. (1993b). Preattentive processing, preparedness, and phobias: Effects of instruction on conditioned electrodermal responses to masked and non-masked fear-relevant stimuli. *Behaviour Research and Therapy, 31*, 87–95.

Spielberger, C. D. (1972). Anxiety as an emotional state. In C. D. Spielberger (Ed.), *Anxiety: Current trends in theory and research* (Vol. 1). New York: Academic Press.

Spinks, J., & Kramer, A. (1991). Capacity views of human information processing: Autonomic measures. In J. R. Jennings & M. G. H. Coles (Eds.), *Handbook of cognitive psychophysiology: Central and autonomic nervous system approaches*. Chichester, England: Wiley.

Trandel, D. V., & McNally, R. J. (1987). Perception of threat cues in post-traumatic stress disorder: Semantic processing without awareness? *Behaviour Research and Therapy, 25*, 469–476.

Tuma, A. H., & Maser, J. D. (Eds.). (1995). *Anxiety and the anxiety disorders*. Hillsdale, NJ: Erlbaum.

Valins, S. (1972). The perception and labelling of bodily changes as determinants of emotional behavior. In P. Black (Ed.), *Physiological correlates of emotion*. New York: Academic Press.

van den Hout, M. A. (1988). The explanation of experimental panic. In S. Rachman & J. D. Maser (Eds.), *Panic: Psychological perspectives*. Hillsdale, NJ: Erlbaum.

van der Molen, G. M., van den Hout, M. A., Vroemen, J., Lousberg, H., & Griez, E. (1986). Cognitive determinants of lactate-induced anxiety. *Behaviour Research and Therapy, 24*, 677–680.

Zajonc, R. B. (1980). Feeling and thinking: Preferences need no inferences. *American Psychologist, 35*, 151–175.