12. Selective Information Processing, Interoception, and Panic Attacks

A. EHlers, J. MARGRAF, and W. T. ROTH

Introduction

Panic attacks have recently been given a central role in the classification of anxiety disorders (DSM-III-R, Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, third edition-revised, APA 1987). The etiology of these anxiety attacks is controversial. One of their most puzzling and fascinating features for clinicians and researchers is that panic attacks often occur in the absence of any perceived situational triggers. This apparent spontaneity has led researchers to search for possible causes of panic attacks "within" the patient. Many biologically oriented researchers believe that panic attacks represent an "endogenous" form of anxiety resulting from metabolic dysfunction. Detailed critical reviews of these medical models of panic disorder are found in Margraf et al. (1986a) and Margraf and Ehlers (in press).

While medical models presume that yet unknown metabolic dysfunctions of the central nervous system cause spontaneous panic attacks, psychophysiological models assume that internal anxiety triggers can be identified and that panic attacks are a fear response to these internal stimuli. The present paper will focus on these alternative models of panic attacks that have recently received increasing attention and empirical support. After a description of the psychophysiological models, some recent evidence will be reviewed. The need for further specification of the models will be discussed. We will focus on two aspects of possible specifications: the role of selective information processing and the role of interoception in panic attacks. Preliminary results from our laboratories will be presented.

Psychophysiological Models of Panic Attacks

Patients report that many panic attacks occur without any situational triggers. In contrast to medical models, psychophysiological models do not take these reports at face value. It is assumed that anxiety triggers can be identified even for "spontaneous" panic attacks. The role of internal triggers is emphasized. These include body sensations like palpitations or dyspnea, thoughts related to danger, and other cognitive changes such as inability to concentrate or feelings of derealization. Since bodily symptoms are most prominent in panic attacks, most researchers have concentrated on bodily sensations as possible triggers for panic. We will follow this tradition in the present paper. Note, however, that for individual patients cognitive events such as derealization may be more important than bodily cues in triggering panic attacks.

In the past few years, a number of researchers have presented models that explain...
panic attacks as the result of a positive feedback loop between internal stimuli (body sensations or cognitive events) and anxiety responses. To emphasize the close interaction between psychological and physiological responses, we use the term "psychophysiological models" (Margraf et al. 1986a, b). However, other groups have used the terms "cognitive" (Beck et al. 1985; Clark 1986; Rapee, in press) or "psychological" (Barlow 1966; van den Hout and Griez 1983; Rapee, in press) models. The idea that positive feedback can lead to panic attacks had already been presented by Lader and Mathews in 1968. These authors assumed that panic was on a continuum with orienting and defense reactions. They argued that panic attacks result from lack of habituation due to a high level of activation and repeated stimulation. However, they emphasized internal anxiety cues to a lesser extent than current models and did not explain why anxiety attacks are less likely when the patient is accompanied by a friend or spouse.

Psychological models of panic are related to the old concept of "fear of fear" often mentioned in the literature on agoraphobia (Evans 1972; Fenichel 1945; Frankl 1975; Freud 1895; Shands and Schor 1982; Westphal 1871; see Goldstein and Chambless 1978 for the most developed "fear of fear" model and for further references). The concept of "fear of fear" explains the large variety of situations that agoraphobic patients fear. They are not afraid of the situation itself, but they are afraid of having a panic attack. Therefore, they start to avoid situations in which a panic attack is likely or would have severe social or physical consequences. Since the "fear of fear" hypothesis was developed in relation to agoraphobia, the authors were mostly interested in panic attacks occurring in feared situations and avoidance behavior. Thus, they elaborated less on what factors lead to "spontaneous" panic.

Current psychophysiological models which were developed to explain spontaneous as well as situational attacks take into account that some anxiety attacks occur without preceding anxiety and explicitly address fluctuations in symptomatology (e.g., "good" and "bad" days). Thus, they avoid some of the problems of overprediction of the "fear of fear" hypothesis (see also Clark 1988).

Psychophysiological models of panic attacks are characterized as follows:

1. In contrast to current medical models (for a review see Margraf et al. 1986a), it is generally assumed that panic attacks are distinct forms of anxiety qualitatively different from fear or anticipatory anxiety.
2. Panic attacks are seen as the result of a positive feedback loop between certain internal cues (body sensations or cognitive events) and the person associated with immediate threat or danger, and the resulting anxiety reaction.
3. It is assumed that psychological interventions that change the person's responses to anxiety symptoms by habituation or reinterpretation are effective in the treatment of panic attacks. This is again in contrast to current medical models that assume that medication is necessary to treat the postulated biological malfunction underlying panic attacks.

Figure 1 shows a schematic presentation of our current understanding of the psychophysiological model. The central part of the schema shows a positive feedback loop (illustrated by black arrows in Fig. 1) leading to a panic attack. Its components are physiological, cognitive, and emotional responses of the panicking person. Note that the positive feedback may start with any of its following components:

- Physiological or cognitive changes occur as a consequence of various causes such as physical effort, drug intake (e.g., caffeine), situational stressors (for example, heat), or emotional responses (e.g., anger or anxiety).
- The person perceives such changes. Note that patients may feel changes in their body sensations even in the absence of physiological changes. For example, patients might feel that their heart rate has accelerated after going to bed because changes in body posture have increased their cardiac awareness.
- The bodily changes or cognitive symptoms are associated with threat or danger. Clark (1986) emphasizes the immediate nature of the anticipated threat. Note that this association can take various forms ranging from conditioning (Goldstein and Chambless 1978; van den Hout and Griez 1983; Barlow 1986; Margraf et al. 1986b) to catastrophic misinterpretations in conscious thoughts (Ottaviani and Beck 1987; Clark 1986; Goldstein and Chambless 1978; Margraf et al. 1986b). The positive feedback may start at this level when situational variables are associated with immediate threat (see below). For example, simple phobias may experience a panic attack when confronted with their phobic stimulus. In agoraphobic patients, the phobic situations are probably only indirectly associated with danger through the association with body sensations (Foa and Kozak 1986).
- The person responds to the perceived threat with anxiety that in turn leads to physiological changes, body sensations, and/or cognitive symptoms (positive feedback). If these symptoms are again perceived and associated with danger, further anxiety increases occur. The patient's anxiety influences these processes. Thus, positive feedback operates at all stages discussed above.

The positive feedback may escalate into a panic attack. Note that is a fast process. It is unclear, at what point anxiety should be called panic. We think that this is a question of severity. We have argued elsewhere that from our laboratory and ambulatory monitoring data, panic attacks do not seem to be an all-or-none phenomenon (Ehlers et al. 1987). Again, the patients' perception and appraisal of their body sensations and of situational variables influence whether they call their anxiety panic (see below).

Panic attacks are time-limited phenomena. The positive feedback model does not explain what terminates a panic attack. Therefore, we have to assume parallel processes that coun-
Thus, the interview and questionnaire data collected so far support the psychophysiological models of panic attacks. However, without further support from experimental studies, the evidence is not conclusive because it depends on the patients' recollections of their panic attacks. In addition, questionnaire and interview studies assess conscious processes while the reported "spontaneity" demonstrates that patients have only limited insight into what triggers panic. Thus, at least some of the processes involved in panic might not be accessible by introspection.

Evidence from Experiments

Recent experimental data support the validity of the questionnaire findings described above. Consistent with the psychophysiological model, the anticipation of anxiety in itself induces anxiety and its physiological concomitants in panic disorder patients to a larger degree than in controls (Ehlers et al. 1988b). Patients and controls had to breathe room air through a mask task on two different occasions. On the 2nd test day they were informed that they would receive carbon dioxide at some point of the inhalation period that could induce anxiety and bodily symptoms. In contrast, they knew that they would only receive room air on the 1st day. Patients showed larger increases in self-reported anxiety and cardiovascular measures only on the 2nd test day, i.e., in anticipation of the anxiety induction procedure.

In another study, panic patients and controls were given false feedback of an abrupt heart rate increase. Only the patients responded with increases in anxiety and physiological arousal (Ehlers et al. 1988a). One patient even experienced a severe panic attack in response to the false feedback (Margraf et al. 1987a). These studies show that a positive feedback loop can be triggered when patients believe that bodily changes have occurred or when they expect to become anxious. Patients were more prone to respond in the direction of positive feedback than controls. These results are in line with those of Clark et al. and Foa (see their chapter in this book) who found that panic patients show larger anxiety responses than controls when reading word pairs of bodily symptoms and catastrophe (Clark) or during imagery (Foa). In addition, Clark et al. used a contextual priming task and found that patients reacted faster to catastrophic misinterpretations of bodily sensations than to neutral interpretations suggesting a bias to interpret bodily symptoms as dangerous.

Further support for the psychophysiological models comes from a research area that used to be primarily associated with medical models of panic. The results from so-called panic induction studies that attempt to provoke anxiety by application of biochemical substances such as sodium lactate or carbon dioxide are consistent with the psychophysiological models (Margraf et al. 1986b; Clark 1986; van den Hout 1988). As all of these procedures induce various unpleasant physical sensations, they can be interpreted as a powerful way to trigger the positive feedback loop described above. Thus, it is not necessary to assume a direct biochemical effect on anxiety to explain the anxiety-inducing effects of pharmacologic panic provocation. The psychophysiological model has the advantage that it explains the variety of procedures that induce panic and, in some cases, have opposite physiological effects (e.g., hyperventilation and lactate infusion induce alkalosis whereas inhalation of 3% CO₂ induces mild acidosis; Ehlers et al. 1986a).

In addition, there are results from panic induction studies that cannot be accounted for by a pure biological interpretation. For example, response to panic induction methods is modified by the subject's expectations. This was demonstrated by manipulating the experimental instructions in healthy subjects (van den Hout and Griez 1982; van der Molen et al. 1986) as well as in panic patients (Margraf et al., in press; Rapee et al., in press). In the study of Margraf et al., patients were more affected by the expectancy manipulation than controls. A recent study from Barlow's laboratory (Sanderson et al.; submitted) demonstrated that the patients' response to carbon dioxide depends on their sense of control. When panic patients thought they had control over CO₂ delivery, few of them panicked although they never used the control mechanism. In contrast, when they knew that they had no control the majority reported a panic attack. Furthermore, medical models cannot explain the case reports of Guttmacher and Nelles (1984) and Shear and Pyer (1987) that patients who had "panicked" during sodium lactate infusion before treatment did not "panic" in response to lactate after successful behavior therapy. Furthermore, they do not explain the efficacy of repeated lactate infusion or CO₂ inhalation in the treatment of panic attacks or other anxiety states (Bonn et al. 1973; Griez and van den Hout 1983). Overall, experimental evidence presented here supports psychophysiological models of panic attacks. In addition, there is a large literature on the relationship of hyperventilation and panic attacks that is in line with a psychophysiological perspective. This literature is reviewed in detail elsewhere (Bass et al., in press; Ley 1987; Rapee, in press).

Evidence from Treatment Studies

There has been controversy between proponents of medical and psychophysiological models whether panic attacks can be successfully treated without giving medication. This question has only recently been addressed by systematic treatment studies. In the large literature on treatment of agoraphobia, for a long time, little emphasis was placed on panic attacks. While the efficacy of exposure therapy in the reduction of agoraphobic avoidance behavior was clearly demonstrated—with follow-ups of up to 9 years (e.g., Burns 1983; Emmelkamp and Kuipers 1979; Fiegenbaum 1986; Goldstein 1982; Hand et al. 1974, 1986; Mathews et al. 1977; McPherson et al. 1980; Michelson et al. 1983; Munby and Johnston 1980), few studies have assessed the effect of the treatment procedures on panic attacks. In their 1984 review, Jacob and Rapport identified only eight studies that reported results on panic attacks. Six of these studies found improvement in panic frequency and/or severity after exposure treatment. Since the focus was agoraphobic avoidance behavior, measures of panic attacks were often unsatisfactory and the results should only be considered preliminary. More recent studies that used better assessment of panic attacks usually found positive effects of exposure to anxiety-provoking situations on panic frequency and intensity (Teich et al. 1983; Michelson et al. 1985; Lelliot et al. 1987; Marchione et al. 1987). No effect was found in the study by Aronson et al. (1985). In the last few years a number of single-case (Griez and van den Hout 1983; Rapee 1985a; Waddell et al. 1984) and group studies (Barlow et al. 1984; Bonn et al. 1984; Clark et al. 1985; Griez et al. 1985; Griez and van den Hout 1986b; Ost 1983; Saikovskis et al. 1988; Sartory 1985; Sokol-Kesler and Beck, cited from Ottavani and Beck 1987) have been presented that demonstrated the efficacy of cognitive-behavioral treatments of panic attacks. Treatment components included education about panic attacks and treatment rationale, exposure to feared internal and external stimuli, cognitive therapy, controlled expiration, methods to increase...
vagal tone, and relaxation techniques. The most common outcome criteria were
diary reports of panic frequency and intensity. All studies found marked and
stable improvement or complete remission. Follow-ups ranged from 3 months to
2 years. Interestingly, most studies found further gains during follow-up. For fur­
ther evidence from more recent treatment studies see the chapters by Barlow, Clark
et al., Fiegenbaum, and Shear et al. in this book.

Although some of the studies used very small samples, the consistency of the results is impressive. Thus, there is suf­
cient evidence to conclude that cognitive-behavioral treatments are effective in the treatment of panic attacks. However, treatment effectiveness can only be seen
as an indirect support of the psychophysi­
ological model. First, the treatment mechanism remains unclear. Second, even with a purely metabolic etiology of panic attacks psychological treatments could be effective. However, the efficacy of psychological treatments clearly
contradicts the assumption of proponents of medical models that medication is neces­
sary to treat panic attacks.

Possible Specification 1:
Role of Interoception

In the psychological literature on panic attacks we often read that panic patients suffer from interoceptive fears. Psychophysi­
ological models usually start with explaining what happens after the patient has perceived certain changes in his body or cognitive functioning. Thus, little is said about the perceptual process itself.

As we will outline here, we think that it is worthwhile to study the process of bodily changes in this population.

There are several reasons to study interoception in persons with panic attacks: First, individual differences in interocep­
tion may be one factor involved in the
etiology of panic disorder. One problem with the psychophysiological model is that usually people have only limited awareness of their bodily functions like cardiac or gastrointestinal activity, especially if these processes are within their normal range of functioning. We therefore have to specify conditions that influence whether a panic patient experiences body sensations. It is conceivable that panic patients differ from other people in their ability to detect changes in their bodily functions like heart rate increases or arrhythmias. Individual differences in interoception could influence the probability of people developing concerns about their heart as well as the probability of positive feedback in the sense of the psychophysiological model. It is also possible that persons who have experienced spontaneous panic attacks will begin to monitor their internal state more closely (e.g., by taking their pulse repeatedly or by shifting their attentional focus). Again, this could increase the probability of positive feedback. Thus, interoception could be involved in the etiology and/or maintenance of panic attacks.

Second, interoception is important for the treatment of panic. The psychological interventions include self-control techniques such as controlled breathing (Saikovskis et al. 1986), stimulation of vagal tone (Sartory 1985), or relaxation (Barlow et al. 1984; Giltin et al. 1985; Ošt 1988). These techniques require the patients' ability to monitor their bodily state and to decide when to use the techniques. The same applies to medication prescribed on a prn basis. Third, interoception may be a precursor of panic attacks. Little is known about predictors of long-term outcome in panic patients or persons with infrequent panic attacks. The ability of a person to detect changes in bodily functions may be one factor influencing prognosis.

In the following, we will discuss cardiac awareness as an example of the possible role of interoception in panic disorder. Cardiac awareness is especially relevant since palpitations are the most commonly reported symptom of panic attacks. In a recent diary study, palpitations were reported in 68% of 175 recorded attacks (Margraf et al. 1987). Do patients with panic disorder differ from controls in their cardiac awareness? If we look at the patients' self-reports, the answer is yes. Panic patients report to be much more aware of sensations from their heart than nonanxious control subjects (King et al. 1985).

There is a lack of studies corroborating self-reports by objective tests of cardiac perception. The literature on interocep­
tion shows that untrained subjects show sensitivities that are not present in panic patients (Katkin 1985). There is some, although inconsis­
tent, evidence that persons with high state anxiety show more heart beat perception (Schandy 1981) and that persons with good cardiac perception are emotionally more responsive than poor perceivers (Schandy 1983; Katkin 1985). However, it was generally found that correlations between self-reported awareness of bodily function and objectively measured cardiac sensitivity are very small (for example, the psychophysical model is especially relevant). However, we cannot draw conclusions from this study unequivocally about cardiac awareness in panic patients because both anxiety neurotics and agoraphobics experience panic attacks.

A recent study by Harbauer-Raum (1987) is more definitive. She assessed 27 patients with cardiac neurosis, a diagnosis that probably represents a subgroup of panic disorder (Buller et al. 1987). In the laboratory, these patients performed better at a heart beat perception task (counting their heart beats without taking their pulse) than 16 normal controls. They were also better at controls and patients with mitral valve prolapse (n = 27) in detecting cardiac arrhythmias occurring during 24-h ECG monitoring. The latter result was subsequently refined by Stalman et al. (1987) who found that patients with cardiac neurosis (n = 25) noticed cardiac arrhythmias better than control subjects (n = 14) and patients with hypochondria­s (n = 5) or autonomic neuropathy (n = 12). Cardiac neurotics detected ventricular extrasystoles but not tachycardias better than patients with mitral-valve prolapse (n = 23).

It is uncertain whether these results can be generalized to the entire population of panic disorder patients. The diagnosis of cardiac neurosis is given to a patient group much more homogeneous with respect to their interpretation of cardiac sensations. Furthermore, not all panic patients are primarily concerned about their cardiac function. There are other reasons to believe that such a generaliza­tion may not be valid. Most importantly, we found a discrepancy between patients'
reports of palpitations in the majority of panic attacks and concurrent heart rate recordings. Based on the patients' descriptions of panic attacks, we usually assume that panic attacks are accompanied by abrupt and large increases in heart rate as observed in single cases (Cohen et al., 1985; Lader and Mathews 1977; Margraf et al., 1987a). Taylor et al. (1986), however, found that this only applies to a subgroup of panic attacks. More recently, we recorded heart rates and levels of physical activity in a larger sample of 44 naturally occurring panic attacks using a portable microcomputer (Margraf et al., 1987b). We found that, on the average, spontaneous panic attacks were not accompanied by heart rate increases. During panic attacks occurring in feared situations, mild heart rate elevations were observed compared to a matched period 24 h later, but these elevations were already present during the 15 min preceding the attacks and may be accounted for by increased levels of physical activity.

In addition, results from other studies cast doubt on a high visceral awareness of panic disorder patients. In our studies, patients tended to report physical symptoms indiscriminately (Ehlers et al., 1986b). Other studies have shown that there is a substantial overlap between hyperventilation and panic disorder (see reviews of Bass et al., in press; Ley 1987; and Rapee, in press). In this literature, it is usually assumed that the patients are unaware of their hyperventilation, i.e., show poor interoception in this respect. Thus, previous research on interoception in disorders related to panic attacks has shown inconsistent if not contradictory findings. We have therefore started a research program designed to study cardiac awareness in carefully diagnosed patients with panic disorder and persons with infrequent panic attacks. We combine a cross-sectional with a longitudinal study. Preliminary results of the cross-sectional part are presented below.

The first two studies conducted at Stanford University compared patients with panic disorder and normal controls that were free of any history of psychiatric disorders. Subjects were asked to do a heart-rate perception test at the beginning and end of a psychophysiological laboratory assessment. Subjects were instructed to match the rate of a train of tone pips to their heart rate without actually taking their pulse. The task was similar to a procedure developed by Porges and Raskin (1969). Subjects were given 1 min to complete the test using a dial with a range from 1 to 10. In the first study, the dial was set at 5 (equalling 60 bpm) before the subject started. Between the first and second test, subjects received 6 min of true and 2 min of false heart rate feedback as described in Ehlers et al. (1988a). The results from this study (based on 24 patients and 24 controls) are shown in Fig. 1. Performance in the heart rate perception test was assessed by the absolute difference between the real and the estimated heart rate (in beats per minute, bpm). In study 1, there was no overall difference in accuracy between the groups. However, controls but not patients improved their estimates after receiving heart rate feedback. The vast majority of subjects in both the patient and control groups underestimated their heart rates. We were concerned that the setting of the dial might have biased the subjects' estimations. Therefore, we repeated the experiment starting with a dial setting of 120 bpm. In this study, no heart rate feedback was given. The preliminary analysis of 22 patients and 18 controls is shown in Fig. 2 (study 2). There were no significant group or time effects or interactions in the ANOVA. As in study 1, the majority of patients underestimated their heart rates. Thus, the studies showed that both patients and controls are generally inaccurate in their heart rate estimations. If we compare the results of studies 1 and 2, however, it appears that patients were more stable in their judgements than controls. Controls were more influenced by the heart rate feedback and by the setting of the dial. When the two protocols were compared in an overall ANOVA, a significant group x protocol interaction was found. Thus, although patients are inaccurate in their heart rate estimations, they seem to be less influenced by external information in making these judgements. One reason for this may be that they rely more on internal information than controls.

Before final conclusions are drawn from these studies, we have to bear in mind, however, that the test of cardiac perception has a number of disadvantages. First, it involves matching of an external and an internal signal. Thus, the task is more complex and requires further stages of information processing beyond accurate interoception (for example, the subject's abilities to monitor two signals at the same time, to compare two rhythms, to compare between two modes of perception, and/or to remember rhythms). Second, the task involves motor activity that in itself has an impact on the subject's heart rate.

We have started a new study at Philipps University (Marburg) using different methods to assess heart rate perception. One of the tests we use is the "heart rate tracking" method described by Schanbacher (1981). Subjects are asked to count their heart beats repeatedly during signalled intervals of 35, 25, and 45 s without taking their pulse and without knowledge of the number of seconds in these intervals. This task was used by Harbauer-Raum (1987), who found superior heart rate perception in patients with cardiac neurosis as described above. Preliminary analyses of this test showed a trend towards a higher percentage of panic patients classified as good cardiac perceivers compared to infrequent paniciers and normal controls. There was no indication of better cardiac perception in infrequent paniciers. Thus, while panic patients insist that they are very aware of cardiac sensations, it is unclear whether these sensations reflect objectively measurable changes in cardiac function. The evidence collected so far suggests that good cardiac perception is only found in a subgroup of panic patients. In these patients, however, excessive awareness of cardiac sensations...
may be one of the factors contributing to the maintenance of the disorder.

Possible Specification 2: Role of Selective Information Processing

Several studies using methods developed in experimental cognitive psychology have demonstrated that anxiety patients show an attentional bias toward threat cues related to their respective disorders. Burgess et al. (1981) used a dichotic listening task and found that six patients with social phobia or agoraphobia detected fear-relevant words (like “public speaking” or “shopping alone”) in the unattended channel better than control groups of six nonanxious subjects and 12 psychology students with high scores on phobia scales. Similarly, in a study of Foa and McNally (1986), 11 obsessive-compulsive patients detected fear-relevant words like “foes” in the unattended channel better than control words and also showed larger skin conductance responses to these words. These differences disappeared after successful behavior therapy, indicating that the effects were not due to differences in familiarity with the stimulus material.

The pattern of results is in line with those of Watts et al. (1986a), who assessed reaction times of spider phobics and normal controls in a modified Stroop color-naming task. Before, but not after treatment, spider phobics showed a larger interference when color-naming words related to spiders such as “creepy” or “crawling.” In contrast, interference was similar in both groups for the standard Stroop test (color words) or other control words. Deficits in the patients’ recognition memory were less clear (Watts et al. 1986b). The subjects were shown dead spiders and had to recognize them later. Patients gave fewer correct answers for big spiders only. In a second experiment, this result was only partly replicated.

In a study of Streblow et al. (1985), phobic patients (type of phobia was not specified) and matched controls were presented slides showing three objects. Their task was to focus their attention on the central item and decide whether this object belonged to a previously given category. The central and/or peripheral objects were either neutral (such as plants, musical instruments) or phobia-relevant (such as insects). The reaction time analysis showed that untreated phobics, but not treated phobics or controls, identified phobia-relevant targets faster than neutral ones. Treated and untreated phobics were more distracted by phobia-relevant peripheral objects. In the case of negative decisions, phobics reacted more slowly when central or peripheral phobic objects were presented. In a second experiment, subjects had to decide whether words had been previously presented to them. In a second experiment, untreated patients recognized phobia-relevant objects faster than control words. In addition, untreated phobics showed longer reaction times when they had to signal that a phobia-relevant word had not been previously presented.

Most relevant to the study of panic disorder is a series of experiments of Mathews and MacLeod, and coworkers on information processing in generalized anxiety disorders. Like panic patients, these patients suffer from severe anxiety not triggered by phobic situations. In reaction-time paradigms such as the Stroop color-naming task (Mathews and MacLeod 1985), dichotic listening (Mathews and MacLeod 1986), or a visual probe detection task combined with word reading (MacLeod et al. 1986a), generalized anxiety patients showed an attentional bias for material representing social threat (words like “stupid,” “hated”) or physical threat (for example, “paralyzed” or “disease”). While patients shifted their attention to emotionally threatening material, controls tended to shift attention away from such material (MacLeod et al. 1986). The simultaneous presentation of threatening material interfered with the patients’ task performance more with the patients’ task performance than with that of controls (Mathews and MacLeod 1985, 1986). Interestingly, these attentional shifts could occur without the subjects’ awareness: Patients could not recall or recognize the threat words better than controls (Mathews and MacLeod 1985, 1986) although they had responded to them differently.

In contrast to the consistent findings on attentional bias, evidence for memory bias in generalized anxiety is less clear. Mogg et al. (1987) studied active recall and recognition memory for positive and threatening adjectives that were either presented as self-relevant or relevant to another person. Previous findings in depression and anxiety patients (P. B. Mathews and MacLeod 1985, 1986) indicate that patients differ in their recall of threatening material less well than controls. This finding is consistent with the trends found by Watts et al. (1986b) in patient phobics, and contrary to Mogg et al.’s predictions.

The findings in information processing in anxiety disorders can be summarized as follows: Patients with anxiety disorders detected anxiety-relevant information in unattended material better or faster than neutral information (Burgess et al. 1981; Foa and McNally 1986; MacLeod et al. 1986; Mathews and MacLeod 1986; Streblow et al. 1985), while control groups did not show this response pattern. When anxiety patients were presented with task-irrelevant but anxiety-relevant information, smaller interference with task performance was found than in controls, even when subjects were told to ignore this information (Watts et al. 1986a; Mathews and MacLeod 1985, 1986; Streblow et al. 1985). One interesting aspect concerns the type of stimulus material used: The studies in phobics of obsessive-compulsive patients used individualized material “tailed” to the specific content of the patients’ fears, whereas the studies of the Mathews group used more general threat words that probably be considered threatening by most subjects. While one might argue that the findings using individualized stimulus material were due to the fact that the material was irrelevant (not threatening) to the controls, but relevant (threatening) to the patients, this does not hold for the attentional bias found in generalized anxiety disorder.

Memory processes are probably less strongly affected than attentional processes in anxiety disorders. The studies cited above did not show differences in recognition memory for unattended material (Mathews and MacLeod 1985, 1986) even though differences in attention were found. If there is a memory bias, it operates in a different direction than the attentional bias. It appears that anxious or phobic subjects may remember threatening material less well (Watts et al. 1986b; Mogg et al. 1987). Note that these authors used measures of the quality of recognition (number of correct answers or measures of discrimination), whereas Streblow et al. (1985) assessed the speed of recognition. In the latter study, phobic patients showed faster recognition of phobia-relevant words in the verification condition and slower recognition in the negative decision case. Unfortunately, hit/false alarm rates were not reported in the Streblow et al. study, and positive and negative decisions are combined in the data reported by Mogg et al. and Watts et al. Thus, the data from these different studies cannot be directly compared. Negative decisions seem to be a more complex task requiring more accurate and complex encoding of the material. It appears that the overall pattern of findings is consistent with the idea of poorer processing of threat material in anxiety disorders. The mechanisms of impaired processing such as poor focused
attention, superficial processing, physiological defense reactions, or inhibition by cognitive avoidance strategies remain to be studied (cf. Mogg et al. 1987; Watts et al. 1986b). Overall, different biases seem to operate at different stages of information processing in anxiety disorders. Anxiety-relevant stimuli recruit attentional resources, but seem to be poorly processed.

The results reported here are directly relevant to the study of panic disorder. They might help in explaining the occurrence of panic attacks in the apparent absence of situational triggers. If panic disorder patients show similar attentional biases to threat cues as generalized anxiety patients, it is possible that information processing of threat cues without the patient's awareness is involved in triggering panic (cf. Mathews and MacLeod 1986). In terms of the psychophysiological model presented in Fig. 1, this would mean that we would have to assume an interaction between the perception of an internal or external cue and its association with danger. Cues related to threat would more easily be detected.

We have started a research program designed to study attentional bias in panic patients and infrequent panicers (see also the chapter by Margraf and Ehlers in this book). In a first series of experiments (Ehlers et al. in press), we used a modified Stroop color-naming task similarly to the study of Mathews and MacLeod (1985). Subjects were asked to color-name each of the cards was taken (in s). After the test, subjects answered a recognition questionnaire containing the 72 words shown on the cards plus 72 other (distractor) words. Table 1 shows a comparison of 24 patients with panic disorder and 24 controls. The time it took subjects to color-name the experimental conditions (threat vs control word), and type of threat (physical vs separation vs embarrassment), we found significant group x condition (P < 0.005) and type x condition (P < 0.05) interactions. Further analyses showed that the group x condition interaction resulted from larger interference with threat words in the patient group. However, significantly larger interference was only found for physical threat, not for the two social threat conditions.

In another study comparing nonclinical panicers and controls, we also found larger interference in panicers when color-naming physical threat words than in controls. Responses to neutral control words and standard Stroop task (color words) were similar in both groups. In neither study, a difference in recognition memory between the groups was found.

Table 1: Stroop color-naming task. Mean time (s) and standard deviations

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical threat words</td>
<td>76.3</td>
<td>70.8</td>
</tr>
<tr>
<td>Control words</td>
<td>72.1</td>
<td>71.0</td>
</tr>
</tbody>
</table>

Thus, there is preliminary evidence for selective information processing of physical threat cues in panic disorder. Panic disorder patients as well as nonclinical panicers showed an attentional bias towards threat-related material. That physical threat words had a larger impact on the patient's performance than social threat words underlines the role of the association between bodily symptoms and danger in panic disorder postulated by psychophysiological models.

Summary and Conclusions

Evidence supporting a psychophysiological perspective of panic attacks is rapidly accumulating. Questionnaire and interview studies, experimental procedures, and treatment trials have yielded converging results. A challenge for future specification of current models is to predict whether a person will have a panic attack in a given period of time. At present, the psychophysiological model seems most powerful in explaining panic attacks retrospectively.

We have outlined two areas of research that seem promising in specifying psychophysiological models. First, the role of treatment should be studied since the models give bodily sensations a central role in the positive feedback loop assumed to lead to panic. In this chapter, we have discussed the relation of self-reported and objectively assessed cardiac awareness in panicers and controls. We found that while panic patients report more than controls that they are generally very aware of sensations from their heart, they are not generally better cardiac perceivers in objective tests of cardiac awareness. Several open questions need to be answered. First, what do patients mean when they say that they are generally very aware of sensations from their heart? Do they mean that they feel their heart beat usually, often, or sometimes? When they speak of sensations from their heart, do they mean heart beats, changes in their heart rate, or arrhythmias? Or is it rather that their pulse sensations make them anxious? These different aspects of self-reported interior perception should be distinguished (see the concept of anxiety probability and sensitivity, Reiss and McNally 1985).

Future studies are needed to clarify the role of accurate vs inaccurate interior perception in panic disorder. It is possible that only in some patients accurate perceptions of internal cues trigger panic. Our studies have shown that only a subgroup of panic disorder patients seems to show accurate cardiac perception. Nevertheless, this variable could be an important factor in the maintenance of their panic attacks.

This subgroup of patients is likely to perceive changes in their cardiac function like fast or strong heart beats that could trigger panic attacks depending on the different factors described above. The etiology of panic attacks in patients who believe that they perceive their heart beat accurately, but who are objectively poor cardiac perceivers might be quite different. One factor contributing to attacks might be that the majority tends to underestimate their heart rate, while those who might overrespond when their heart beat becomes perceptible, for instance, when lying in bed.
Future studies should also investigate the state-dependence of cardiac perception. It is possible that while most panic patients cannot monitor their cardiac function accurately in the usual laboratory conditions, they may be more likely than controls to become aware of their heart beat under certain conditions such as changes in posture, drug intake, or anxiety. Another possibility is that while panic patients may usually not be more aware of their heart rate they might be more sensitive to changes in other cardiac parameters like stroke volume.

At present, the reasons for why there are higher percentages of good cardiac perceivers among patients with panic disorder compared to infrequent perceivers among patients with panic disorder remains unknown. It is possible that good cardiac perception in patients reflects a consequence of the disorder. For example, panic patients might train their cardiac perception by taking their pulse frequently. At the same time, good cardiac perception may be one of the factors increasing the probability for an infrequent pan­ accident to develop more frequent panic attacks. Only prospective longitudinal studies can decide which of the interpretations is valid. We are currently studying this question in a prospective study of infrequent perceivers.

A second area of great interest is the role of possible attentional and/or memory factors increasing the probability for an infrequent perceiver to develop more frequent panic attacks. Only prospective longitudinal studies can decide which of the interpretations is valid. We are currently studying this question in a prospective study of infrequent perceivers.

Acknowledgements: Preparation of this paper was supported in part by the German Research Foundation (grant Eh 97/1-1 to Anke Ehlers) and by the Medical Research Service of the Veterans Administration. We thank Sylvia Davies, Gerhard Jakuch, Frank Wrobel, and Peter Zesula for their assistance in data collection, and Franziska Schneider and Peter Zesula for technical support.

References


Ley R (1985) Agoraphobia, the panic attack and the hyperventilation syndrome. Behav Res Ther 23:79-81
Margraf J, Ehlers A, Roth WT (1986a) Biological models of panic disorder and agoraphobia. a review. Behav Res Ther 24:553-567
Sanderson WC, Rapee RM, Barlow DH (submitted) The influence of an illusion of control on panic attacks in response to inhalation of 5.2% carbon dioxide. Arch Gen Psychiatry
Sartory G (1985) Vagal innervation techniques in the treatment of panic attacks. 15th Annual meeting of the European Association for Behaviour Therapy, Munich
Wampa, Hillsdale