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A COGNITIVE APPROACH TO PANIC

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Summary—A cognitive model of panic is described. Within this model panic attacks are said to result from the catastrophic misinterpretation of certain bodily sensations. The sensations which are misinterpreted are mainly those involved in normal anxiety responses (e.g. palpitations, breathlessness, dizziness etc.) but also include some other sensations. The catastrophic misinterpretation involves perceiving these sensations as much more dangerous than they really are (e.g. perceiving palpitations as evidence of an impending heart attack). A review of the literature indicates that the proposed model is consistent with the major features of panic. In particular, it is consistent with the nature of the cognitive disturbance in panic patients, the perceived sequence of events in an attack, the occurrence of 'spontaneous' attacks, the role of hyperventilation in attacks, the effects of sodium lactate and the literature on psychological and pharmacological treatments. Finally, a series of direct tests of the model are proposed.

INTRODUCTION

Ever since Freud's (1894) classic essay on anxiety neurosis, it has been accepted that panic attacks are a frequent accompaniment of certain types of anxiety disorder. However, it is only relatively recently that panic attacks have become a focus of research interest in their own right. This shift in emphasis is largely a result of the work of Donald Klein. In a series of studies which started in the 1960s, Klein and his colleagues (Klein, 1964; Zitrin, Klein and Woerner, 1980; Zitrin, Woerner and Klein, 1981; Zitrin, Klein, Woerner and Ross, 1983) appeared to demonstrate that anxiety disorders which are characterized by panic attacks respond to imipramine while anxiety disorders which are not characterized by panic attacks fail to respond to imipramine. This 'pharmacological dissociation' led Klein (1981) to propose that panic anxiety is *qualitatively* different from non-panic anxiety. A view which was subsequently endorsed by the writers of DSM-III (APA, 1980) when they created the two diagnostic categories of 'panic disorder' and 'agoraphobia with panic' and used the presence or absence of panic attacks as a major criteria for distinguishing between different types of anxiety disorder. Following the publication of DSM-III, there has been an enormous increase in research on panic attacks. Perhaps because drug studies were the major stimulus for the creation of the diagnostic category of panic disorder, most recent research has concentrated on biological approaches to the understanding of panic. However, there are a number of reasons for supposing that panic attacks might be best understood from a cognitive perspective. After a brief description of the phenomenology of panic attacks, the present article presents a cognitive approach to the understanding of panic. A literature review indicates that the proposed cognitive model is consistent with existing information on the nature of panic and the article concludes with a set of specific predictions which could be used to test the model.

THE PHENOMENOLOGY OF PANIC ATTACKS

A panic attack consists of an intense feeling of apprehension or impending doom which is of sudden onset and which is associated with a wide range of distressing physical sensations. These sensations include breathlessness, palpitations, chest pain, choking, dizziness, tingling in the hands and feet, hot and cold flushes, sweating, faintness, trembling and feelings of unreality. Panic attacks occur in both phobic and non-phobic anxiety disorders. Within phobics, attacks occur in feared situations (such as a supermarket for an agoraphobic) but some attacks occur in 'safe' situations such as at home. Some attacks follow a clearly identifiable precipitating event or short period of anxious rumination but other attacks are perceived by patients as occurring 'out of the blue'. The latter are commonly termed 'spontaneous' panic attacks. The majority of people who suffer

frequent panic attacks fall into the DSM-III categories of panic disorder or agoraphobia with panic. In order to be diagnosed as suffering from panic disorder an individual must have had at least three panic attacks in the last 3 weeks and these attacks must not be restricted to circumscribed phobic situations. In order to be diagnosed as suffering from agoraphobia with panic, an individual must show marked fear and avoidance of the agoraphobic cluster of situations and also have a history of panic attacks.

A COGNITIVE MODEL OF PANIC ATTACKS

Paradoxically, the cognitive model of panic attacks is perhaps most easily introduced by discussing work which has focused on neurochemical and pharmacological approaches to the understanding of panic. This work has established that in patients, panic attacks can be provoked by a wide range of pharmacological and physiological agents including: infusions of lactate (Appleby, Klein, Sachar and Levitt, 1981; Leibowitz, Fyer, Gorman, Dillon, Appleby, Levy, Anderson, Levitt, Palij, Davies and Klein, 1984), yohimbine (Charney, Beninger and Breier, 1984) and isoproterenol (Rainey, Pohl, Williams, Knitter, Freedman and Ettedgui, 1984); oral administration of caffeine (Charney, Beninger and Jatlow, 1985); voluntary hyperventilation (Clark, Salkovskis and Chalkley, 1985) and inhalation of carbon dioxide (van den Hout and Griez, 1984). These agents rarely provoke panic attacks in individuals without a history of panic. However, they produce some of the bodily sensations which are associated with panic attacks in most individuals. The success of the agents at producing panic attacks in panic patients and their less marked effects on normals have been taken to indicate that certain biochemical changes have a direct panic-inducing effect, and that individuals who are vulnerable to the agents have a biochemical disorder. These conclusions have provided a rationale for the further exploration of drug treatments for panic (Chouinard, Annabie, Fontaine and Solyom, 1982; Zitrin, 1983) and also for studies which attempt to identify neurochemical abnormalities in panic patients (Charney *et al.*, 1984; Nesse, Cameron, Curtis, McCann and Huber-Smith, 1984).

However, two recent studies (Clark and Hemsley, 1982; van den Hout and Griez, 1982) suggest an alternative, psychological, explanation for the panic-inducing effects of these diverse agents. These studies investigated the effects of two panic-inducing agents—hyperventilation (Clark and Hemsley, 1982) and CO_2 inhalation (van den Hout and Griez, 1982)—in normal *S*s. It was found that individuals varied considerably in their affective response to the procedures and there was tentative evidence that the extent to which individuals experienced the procedures as pleasurable or aversive was determined by cognitive factors such as expectation and the recall of previous experiences with the induced sensations. This suggests that the various pharmacological and physiological agents which have been shown to promote panic in patients may not have direct panic-inducing effects but instead may provoke panic only if the bodily sensations which they induce are interpreted in a particular fashion. This is the central notion behind the cognitive theory of panic which is described below.

It is proposed that panic attacks result from the catastrophic misinterpretation of certain bodily sensations. The sensations which are misinterpreted are mainly those which are involved in normal anxiety responses (e.g. palpitations, breathlessness, dizziness etc.) but also include some other bodily sensations. The catastrophic misinterpretation involves perceiving these sensations as much more dangerous than they really are. Examples of catastrophic misinterpretations would be a healthy individual perceiving palpitations as evidence of impending heart attack; perceiving a slight feeling of breathlessness as evidence of impending cessation of breathing and consequent death; or perceiving a shaky feeling as evidence of impending loss of control and insanity.

Figure 1 illustrates the sequence of events that it is suggested occurs in a panic attack.* A wide range of stimuli appear to provoke attacks. These stimuli can be external (such as a supermarket for an agoraphobic who has previously had an attack in a supermarket) but more often are internal (body sensation, thought or image). If these stimuli are perceived as a threat, a state of mild

*Although derived independently, the present model has similarities with the models of panic which have recently been proposed by Beck, Emery and Greenberg (1985) and by Griez and van den Hout (1984).

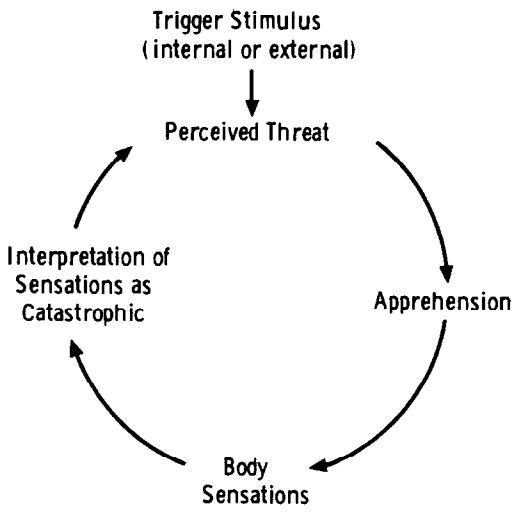

Fig. 1. A cognitive model of panic attacks.

apprehension results. This state is accompanied by a wide range of body sensations. If these anxiety-produced sensations are interpreted in a catastrophic fashion, a further increase in apprehension occurs. This produces a further increase in body sensations and so on round in a vicious circle which culminates in a panic attack.

The model shown in Fig. 1 can deal both with panic attacks which are preceded by a period of heightened anxiety and also with panic attacks which are not preceded by a period of heightened anxiety but instead appear to come 'out of the blue'. In the case of attacks which are preceded by heightened anxiety two distinct types of attack can be distinguished. In the first the heightened anxiety which precedes the attack is concerned with the anticipation of an attack. This is often the case when agoraphobics experience an attack in a situation (such as a supermarket) where they have previously panicked. On entering such a situation they tend to become anxious in anticipation of a further attack, then selectively focus on their body, notice an unpleasant body sensation, interpret this as evidence of an impending attack and consequently activate the vicious circle which produces an attack. In other cases the heightened anxiety which precedes an attack may be quite unconnected with anticipation of an attack. For example, an individual may become nervous as a result of the particular topics which are being discussed in a dispute with a spouse, notice their bodily reaction to the argument, catastrophically interpret these sensations and then panic.

In the case of panic attacks which are not preceded by a period of heightened anxiety, the trigger for an attack often seems to be the perception of a bodily sensation which itself is caused by a different emotional state (excitement, anger) or by some quite innocuous event such as suddenly getting up from the sitting position (dizziness), exercise (breathlessness, palpitations) or drinking coffee (palpitations). Once perceived the body sensation is interpreted in a catastrophic fashion and then a panic attack results. In such attacks patients often fail to distinguish between the triggering body sensation and the subsequent panic attack and so perceive the attacks as having no cause and coming 'out of the blue'. This is understandable given the patients' general beliefs about the meaning of an attack. For example, if an individual believes that there is something wrong with his heart, he is unlikely to view the palpitation which triggers an attack as different from the attack itself. Instead he is likely to view both as aspects of the same thing—a heart attack or near miss.

In Fig. 1 it is hypothesized that the misinterpretation of bodily symptoms of anxiety is always involved in the vicious circle which culminates in a panic attack. However, other sensations can also play a role in panic, particularly as triggering stimuli. We have already mentioned sensations such as breathlessness and palpitations which sometimes are produced by anxiety but other times can initially be produced by innocuous events or positive emotions. In addition, occasionally panic attacks are triggered by sensations which are never part of an anxiety response. For example, floaters in the visual field are not symptoms of anxiety. However, if an individual was concerned about the possibility of a deterioration in sight then perception of a floater could trigger a panic.

The individual might interpret the floater as a sign of impending visual deterioration, become anxious, as a product of this anxiety experience blurred vision, this would further reinforce the belief that there was something seriously wrong with his or her vision and so activate a vicious circle of misinterpretation and increasing blurred vision which culminates in a panic attack.

So far our discussion of the sensations whose misinterpretation results in a panic attack has mainly concentrated on sensations which arise from the perception of internal physical processes (e.g. palpitations). These are the most common sensations involved in the production of panic attacks. However, sensations which arise from the perception of mental processes can also contribute to the vicious circle which culminates in a panic attack. For example, for some patients the belief that they are about to go mad is partly based on moments when their mind suddenly goes blank. These moments are interpreted as evidence of impending loss of control over thinking and consequent insanity.

A final aspect of the cognitive model which requires comment concerns the temporal stability of patients' catastrophic interpretations of bodily sensations. For some patients the panic-triggering sensations and their interpretations of those sensations remain fairly constant across time. However, in other patients both the sensations and interpretations change over time. For example, some patients appear to have a rather vague belief that they are going to suffer from some serious illness, this leads them to misinterpret a very wide range of bodily sensations and the particular misinterpretations will vary depending on which bodily sensations are noticed, what illnesses they have information about and what illnesses they have already been able to discount.

A BRIEF REVIEW OF RESEARCH ON PANIC ATTACKS

Having presented a cognitive model of panic, I will now briefly review the literature on panic to determine the extent to which it is consistent with the proposed model.

(1) Ideational components of panic anxiety

If the above model is correct, one would expect that the thinking of patients who suffer from panic attacks would be dominated by thoughts which relate to the catastrophic interpretation of bodily sensations. A recent interview study has provided data which is broadly consistent with this hypothesis. Hibbert (1984a) compared the ideation of non-phobic patients who experienced panic attacks ($n = 17$) and non-phobic patients who did not experience panic attacks ($n = 8$). Panic patients were significantly more likely than non-panic patients to have thoughts concerned with the anticipation of illness, death or loss of control (includes 'going mad'), but did not differ from non-panic patients in the frequency of thoughts concerned with general feelings of being unable to cope or with the anticipation of social embarrassment. In discussing these results Hibbert (1984a, p. 622) concluded that "the ideational content of those experiencing panic attacks can be understood as a reaction to the somatic symptoms, a connection insisted upon by all but 2 of the patients".

(2) Perceived sequence of events in a panic attack

As the cognitive model specifies that panic attacks result from the catastrophic interpretation of bodily sensations, one would expect that a bodily sensation would be one of the first things which individuals notice during an attack. Two studies have asked patients about the perceived sequence of events in an attack and both have provided results consistent with this expectation. Hibbert (1984a) and Ley (1985) both found that panic patients frequently report that the first thing they notice during an episode of anxiety is a physical feeling. In addition, Hibbert (1984a) found that this sequence of events was reported significantly more often by patients with panic attacks than by patients without panic attacks (53 and 0%, respectively).

(3) The role of hyperventilation in panic attacks

The bodily sensations which are produced by voluntary hyperventilation are very similar to those experienced in naturally occurring panic attacks. This observation has led a number of writers (Clark, 1979; Clark and Hemsley, 1982; Gibson, 1978; Hibbert, 1984b; Kerr, Dalton and Gliebe, 1937; Lewis, 1957; Lum, 1976; Wolpe, 1973) to suggest that hyperventilation may play an

important role in the production of panic attacks. Consistent with this suggestion it has been shown that in some panic patients (i) voluntary hyperventilation produces a panic-like state (Clark *et al.*, 1985) and (ii) hyperventilation accompanies naturally occurring panic attacks (Hibbert, 1986; Salkovskis, Warwick, Clark and Wessels, 1986c), panic attacks produced by contrived psychological stress (Salkovskis, Clark and Jones, 1986a) and panic attacks induced by sodium lactate (Liebowitz, Gorman, Abby, Levitt, Dillon, Gail, Appleby, Anderson, Palij, Davies and Klein, 1985b). These observations suggest that hyperventilation plays a role in some panic attacks. However, it is clear that hyperventilation *per se* does not produce panic. As already mentioned, studies of the effects of hyperventilation in normals (Clark and Hemsley, 1982; Svebak and Grossman, 1986) have shown that individuals vary considerably in their affective response to hyperventilation with some individuals actually finding the experience enjoyable. It is therefore suggested that hyperventilation only induces panic if the bodily sensations which it induces are (a) perceived as unpleasant and (b) interpreted in a catastrophic fashion.

(4) Lactate-induced panic

Infusions of sodium lactate are the most frequently used technique for inducing panic attacks in the laboratory. Between 60–90% of panic patients and 0–20% of normals and non-panic anxious patients experience an attack when given an i.v. infusion of 0.5–1.0 M racemic sodium lactate (Appleby *et al.*, 1981; Liebowitz *et al.*, 1984, 1985a; Rainey *et al.*, 1984). Even when *S*s do not panic, lactate infusions are accompanied by a wide range of physiological and biochemical changes. Liebowitz *et al.* (1985b) reported that lactate produces significant increases in heart rate, systolic blood pressure, pyruvate, prolactin and bicarbonate, and significant decreases in cortisol, pCO_2, phosphate and ionized calcium. As some of these changes in bodily function are likely to be perceived, the cognitive model could account for the panic-inducing effects of lactate by proposing that individuals who panic do so because they catastrophically interpret the induced sensations. This is consistent with Liebowitz *et al.*'s (1985b, p. 718) observation that individuals who panic during lactate infusion perceive bodily changes such as tremor and parasthesias well before the onset of panic. The fact that more patients than controls panic on lactate would mainly be explained by supposing that patients have, as a relatively enduring characteristic, a particularly marked tendency to interpret certain bodily sensations in a catastrophic fashion. However, in some studies part of the difference in response between patients and controls may be due to differences in the instructions given to the two groups. For example, in their pre-infusion instructions, Appleby *et al.* (1981) told patients that they "might experience a panic attack" (p. 413) but told controls that they "might experience an attack with symptoms analogous to those of 'public speaking'" (p. 413). As the controls presumably had never experienced a panic attack but probably had been slightly nervous during public speaking these instructions are likely to lead controls to expect a less-frightening experience than patients. Such differences in expectation can have marked effects on the affect produced by biochemical interventions (cf. van den Hout and Griez, 1982).

(5) Effects of psychological treatment

The proposal that panic attacks result from the catastrophic interpretation of certain bodily sensations suggests both a cognitive–behavioural and a behavioural approach to the treatment of panic attacks. The cognitive–behavioural approach would involve identifying patients' negative interpretations of the bodily sensations which they experience in panic attacks, suggesting alternative non-catastrophic interpretations of the sensations and then helping the patient to test the validity of these alternative interpretations through discussion and behavioural experiments. The behavioural approach would capitalize on the observation that fear of specific stimuli can often be treated by repeated, controlled exposure to those stimuli and would consist of graded exposure to the body sensations which accompany panic. Recently both of these approaches have been tried and the initial results are highly encouraging.

Clark *et al.* (1985) adopted the cognitive–behavioural approach and concentrated on one particular alternative interpretation—the view that the bodily sensations which patients experience in a panic attack are the result of stress-induced hyperventilation rather than the more catastrophic

things which patients usually fear (impending heart attack, insanity, loss of control). The treatment had five stages:

(1) Brief voluntary hyperventilation. This was intended to induce a state which patients recognized as similar to their panic attacks.

(2) Explanation and discussion of the way hyperventilation induces panic. On the basis of the results of the brief period of voluntary hyperventilation, it was suggested to patients that during a panic attack they may be overbreathing. This then produces a wide range of bodily sensations which they interpret in a catastrophic fashion leading to greater anxiety, more overbreathing and so on round in a vicious circle which culminates in a panic attack. Patients attempted to fit their own recent experience of panic attacks into this model and where doubts were expressed these were carefully considered. Attacks which initially appeared inconsistent with the model were carefully discussed. After reviewing details of these attacks with the therapist, the patient was often able to see ways in which the model could account for the attacks.

(3) Training in a pattern of slow, controlled breathing to use in a coping technique during attacks.

(4) Training in more appropriate cognitive responses to bodily symptoms. These responses were based on the discussion described above.

(5) Identification and modification of panic triggers. By reviewing panic diaries, it was often possible to identify hitherto unrecognized triggers for panic. This helped some patients to see their panic attacks as more understandable, made them less frightening and suggested control techniques. Examples of triggers identified in this way were high caffeine intake, misinterpretation of the bodily symptoms of a hangover, postural hypotension, phase in the menstrual cycle and fleeting, bizarre images.

To date, two evaluations of this treatment approach have been reported. In the first evaluation (Clark *et al.*, 1985) patients were selected who perceived a similarity between the effects of overbreathing and naturally occurring panic attacks. Substantial reductions in panic attack frequency were observed during the first few weeks of treatment. These initial gains, which occurred in the absence of exposure to feared external situations, were improved upon with further treatment and maintained at 2-yr follow-up. In the second evaluation (Salkovskis, Jones and Clark, 1986b), an unselected group of panic patients were studied. Again a substantial reduction in attack frequency was observed. In addition, there was some evidence that outcome was positively correlated with the extent to which patients perceived a marked similarity between the effects of voluntary overbreathing and naturally occurring attacks. Neither study employed a waiting-list control group. However, it is unlikely that the observed improvements are due to spontaneous remission as, in both studies, a stable baseline was established before treatment, and significant improvements from baseline took place in a treatment period shorter than the baseline. It therefore appears that the cognitive–behavioural package is an effective treatment for panic, especially in patients who perceive a marked similarity between hyperventilation and naturally occurring panic. Patients who fail to perceive a marked similarity between the effects of hyperventilation and naturally occurring panic would probably benefit from the inclusion of additional procedures which concentrate on other, non-catastrophic explanations of bodily sensations (cf. Clark, 1986).

Griez and van den Hout (1983, 1986) adopted the behavioural approach to treatment and used inhalations of 35% CO_2/65% O_2 as a way of repeatedly exposing patients to the bodily sensations which accompany panic attacks. Inhalation of 35% CO_2/65% O_2 is a highly effective technique for inducing the bodily sensations of panic (van den Hout and Griez, 1984). Its effects appear to result from the sudden drop in pCO_2 (hyperventilation) which follows exhalation of the gas rather than from the increase in pCO_2 (hypercapnia) which accompanies inhalation (van den Hout and Griez, 1984). When used as a treatment, inhalations are introduced in a graded fashion. Initially, *S*s take small inhalations, as their anxiety drops they are encouraged to take a full-depth inhalation and eventually take several full-depth inhalations each session. Griez and van den Hout (1986) evaluated the short-term effectiveness of this treatment using a cross-over design in which 2 weeks

of CO_2 inhalation therapy was compared with 2 weeks of propranolol. CO_2 inhalation therapy was associated with significant reductions in panic attack frequency and fear of autonomic sensations. Propranolol failed to have significant effects on either of these measures. However, the difference in change scores between treatments only reached significance on the measure of fear of autonomic sensations. In view of the unusually brief duration of therapy, it is possible that Griez and van den Hout's (1986) results are an underestimate of the effectiveness of CO_2 inhalation therapy. Although the therapy was associated with substantial drops in panic attack frequency, most patients were not panic-free at the end of 2 weeks and it is possible that further improvements would have been observed if the therapy had been extended over a longer and more normal period of time.

At this stage, neither Clark *et al.*'s (1985) cognitive–behavioural treatment nor Griez and van den Hout's (1984) behavioural treatment have been compared against an alternative psychological treatment in order to control for non-specific therapy ingredients. Until this is done it is not possible to say whether the apparent effectiveness of the treatments is due to their specific emphasis on fear of internal sensations. However, it is encouraging to note that both treatments appear to be effective with panic disorder patients as these patients form a group for whom there is no generally accepted psychological treatment. In Clark *et al.*'s (1985) study, these patients (termed 'non-situationals') did extremely well. Indeed most were panic-free by the end of treatment.

In contrast to panic disorder, there is a generally accepted psychological treatment for agoraphobia with panic. Numerous studies (cf. Mathews, Gelder and Johnston, 1981) have shown that graded, *in vivo* exposure to feared external situations is an effective treatment for agoraphobic avoidance and situational fear. Early studies did not include direct measures of panic but it was assumed that panic attacks would decline as situational fear declined and recent studies have confirmed this assumption (Marks, Grey, Cohen, Hill, Mawson, Ramm and Stern, 1983; Michelson, Marchione and Mavissakalian, 1985; Mavissakalian and Michelson, 1986). The question therefore arises of whether the cognitive–behavioural and behavioural treatments described above have anything to add to graded, *in vivo* exposure. Several authors (Freud, 1895; Goldstein and Chambless, 1978; Hallam, 1978; Klein, 1981; Westphal, 1872) have argued that in many cases agoraphobia is best viewed as a fear of panic rather than a fear of specific situations. This suggests that treatments which directly tackle panic may produce more generalized change. In particular, they may be more effective than graded exposure alone in reducing the frequency of 'spontaneous' panic attacks and panic attacks which occur in patient's homes. Certainly there is room for further improvement in these areas. In a recent study, Michelson *et al.* (1985) found that 45% of patients given the DSM-III diagnosis of agoraphobia with panic were still experiencing panic attacks at home without obvious environmental provocation after 3 months of *in vivo*, therapist-assisted exposure to feared situations.

(6) The role of biological factors in panic

By specifying that the catastrophic interpretation of certain bodily sensations is a necessary condition for the production of a panic attack, the cognitive model provides a different perspective to that offered by biological models of panic and also provides a rationale for psychological approaches to treatment. However, it would be wrong to assume that biological factors have no role to play in panic attacks. *In principle*, there are, at least three ways in which biological factors might increase an individuals' vulnerability to the vicious circle shown in Fig. 1.

First, biological factors may contribute to the triggering of an attack. As already mentioned, panic attacks are often triggered by a perceived body sensation such as breathlessness or palipitations, and such sensations appear to be reported more frequently by panic patients than by other patients or normals. Body sensations are particularly likely to be noticed when there is a change in bodily processes (Pennebaker, 1982). It is therefore possible that the increase in perceived body sensations observed in panic patients occurs because such patients experience more, or more intense, benign fluctuations in body state than others.

Second, biological factors are likely to influence the extent to which a perceived threat produces an increase in bodily sensations, as shown in Fig. 1. The reduced efficiency of central adrenergic α_2-autoreceptors, which it has been suggested is characteristic of panic patients (Charney *et al.*, 1984), would be an example of such an effect. Noradrenergic neurons in the locus coerulus and

other brain-stem areas play an important role in the control of the autonomic nervous system. The α_2-adrenergic autoreceptor has an inhibitory influence on presynaptic noradrenergic neurons. A deficiency in this autoreceptor would mean that release of noradrenaline would not be damped down by presynaptic inhibition and individuals with such a deficiency would experience larger than normal surges in noradrenaline and sympathetic nervous system activation in response to a perceived threat. A further example of a biological influence on the extent to which a perceived threat produces an increase in body sensations comes from the literature on hyperventilation. As already mentioned, in some patients the bodily sensations which occur in a panic attack are partly a result of hyperventilation and the effects of hyperventilation vary with resting levels of pCO_2 which in turn vary with phase in the menstrual cycle (Damos-Mora, Davies, Taylor and Jenner, 1980).

Finally, the extent to which bodily sensations which accompany anxiety are interpreted in a catastrophic fashion will largely be determined by psychological factors. However, biological factors may also have a role to play in this aspect of the vicious circle. For example, the hypothesized deficiency in central α_2-adrenergic autoreceptors would mean that individuals would be more likely to experience sudden surges in sympathetic activity and surges in activity may be more likely to be interpreted in a catastrophic fashion than gradual build-ups.

(7) Effects of pharmacological treatment

Within the model shown in Fig. 1, there are several ways in which drugs could be effective in reducing the frequency of panic attacks. Blockade of, or exposure to the bodily sensations which accompany anxiety, and a reduction in the frequency of bodily fluctuations which can trigger panic could all have short term effects on panic. However, if patients' tendency to interpret bodily sensations in a catastrophic fashion is not changed, discontinuation of drug treatment should be associated with a high rate of relapse.

So far, three drugs (propranolol, diazepam and imipramine) have been investigated in controlled trials which include measures of panic. Propranolol appears to be ineffective, even when given in doses which are sufficient to effect β-blockade (Noyes, Anderson, Clancy, Crowe, Slymen, Ghoneim and Hinrichs, 1984; Griez and van den Hout, 1986). This is perhaps because β-blockade reduces the cardiovascular aspects of panic but appears to leave some of the other bodily sensations unaffected (Gorman, Levy, Liebowitz, McGrath, Appleby, Dillon, Davies and Klein, 1983). Noyes *et al.* (1984) found that high doses of diazepam (up to 30 mg) were effective in reducing panic frequency over a period of 2 weeks but they failed to provide data on the long-term effectiveness of diazepam. However, other studies (Catalan and Gath, 1985) have raised serious doubts about the long-term effectiveness of diazepam as a treatment for anxiety. In contrast to propranolol and diazepam, more positive results have been obtained with imipramine. Three controlled trials (Zitrin *et al.*, 1980, 1983; McNair and Kahn, 1981) have found that imipramine is more effective than an inert placebo in reducing the frequency of panic attacks in agoraphobics with panic and a further trial (Telch, Agras, Taylor, Roth and Gallen, 1985) obtained a trend towards a significant difference between imipramine and placebo ($P < 0.1$). However, two further studies with agoraphobics (Marks *et al.*, 1983; Mavissakalian and Michelson, 1986) failed to find differences between imipramine and placebo on measures of panic. In those studies in which imipramine has been more effective than placebo it has always been combined with graded exposure to feared situations. This raises the possibility that imipramine may not have direct anti-panic effects, but instead simply potentiates the effects of self-initiated and/or therapist-initiated graded exposure. Consistent with this suggestion, Telch *et al.* (1985) found that imipramine had no effect on panic when given in conjunction with counter-exposure instructions. However, in the only study to investigate the effects of imipramine in panic disorder (as opposed to agoraphobia with panic), Garakani, Zitrin and Klein (1984) found that imipramine without the addition of psychological treatment was associated with a marked reduction in panic attacks. This study was a case series and so did not include a placebo control group. Until a study is reported which includes such a group, it will remain unclear whether imipramine has a specific anti-panic effect in panic disorder.

SUMMARY AND PREDICTIONS

It has been suggested that panic attacks result from the catastrophic interpretation of certain bodily sensations. The sensations which are misinterpreted are mainly those which are involved in normal anxiety responses (e.g. palpitations, breathlessness, dizziness etc.) but also include some other sensations. The catastrophic misinterpretation involves perceiving these sensations as much more dangerous than they really are (e.g. perceiving palpitations as evidence of an impending heart attack). Encouragingly, a review of the literature indicates that the proposed model is consistent with the nature of the cognitive disturbance in panic patients, the perceived sequence of events in an attack, the occurrence of 'spontaneous' attacks, the role of hyperventilation in attacks, the effects of sodium lactate and the literature on psychological and pharmacological treatments. However, at this stage, no studies have been reported which provide a direct test of the cognitive model. Final evaluation of the model must therefore await studies which test its central predictions. These predictions are:

(1) Compared to other anxious patients and normal controls, patients who suffer from panic attacks will be more likely to interpret certain bodily sensations in a catastrophic fashion.*

(2) Pharmacological agents which provoke panic (such as sodium lactate) do so only when the somatic sensations produced by the agent are interpreted in a catastrophic fashion, and the panic-inducing effects of these agents can be blocked by instructional manipulations.

(3) Treatments which fail to change patients tendency to interpret bodily sensations in a catastrophic fashion will have higher rates of relapse than treatments which succeed in changing interpretations.

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*In testing this prediction it may be important to distinguish between immediate and long-term threat. It is likely that interpretations of sensations which lead patients to believe they are in immediate danger of a catastrophy such as dying or going mad will be particularly characteristic of panic while interpretations which imply some distant danger may be more characteristic of hypochondriasis.

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