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ANXIETY INDUCED BY FALSE HEART RATE FEEDBACK IN PATIENTS WITH PANIC DISORDER

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Summary—The psychophysiological model of panic attacks postulates a positive feedback loop between anxiety symptoms and the patient's anxious reaction to these symptoms. We tested the underlying assumption that the appraisal of bodily change can induce anxiety in this patient group. Twenty-five patients with panic disorder or agoraphobia with panic attacks (DSM-III) and 25 matched normal controls were given false feedback of an abrupt heart rate increase. Self-ratings of anxiety and excitement, heart rate, skin conductance level, and systolic and diastolic blood pressure were taken. On all measures, patients who believed that the feedback was accurate ($N = 19$) responded differently to the false feedback than controls ($N = 16$). Patients showed increases in anxiety and physiological arousal. The preceding true heart rate feedback did not induce changes in anxiety. Patients and controls did not differ in their accuracy of heart rate perception. The results underline the role of appraisal processes and the fear of anxiety symptoms in panic disorder.

INTRODUCTION

Panic attacks are sudden surges of anxiety accompanied by somatic symptoms such as palpitations, dyspnea, or sweating (Freud, 1895; DSM-III, American Psychiatric Association, 1980). Patients suffering from panic disorder report that panic attacks often occur unexpectedly and unpredictably, that is, in the absence of any perceived situational triggers. It is unknown what causes panic attacks. Their apparent 'spontaneity' has led researchers to believe that they result from an unknown metabolic dysfunction or disease [Klein (1980), Carr and Sheehan (1984); for a review see Margraf, Ehlers and Roth (1986a)]. Other authors have proposed that panic patients suffer from exaggerated fear responses to their anxiety symptoms. They assume that panic attacks are often triggered by internal events such as body sensations and cognitions. Panic attacks are explained by a positive feedback loop between bodily symptoms of anxiety and the person's reaction to these symptoms (Barlow, 1986; Clark, 1986; Griez and van den Hout, 1983; Lader, 1975; Margraf, Ehlers and Roth, 1986b; Mathews, Gelder and Johnston, 1981). Cognitive processes such as the appraisal of bodily changes or environmental cues as dangerous, or as indicating loss of control, are considered to be involved in the exacerbation of anxiety. This psychophysiological model of panic attacks is related to the concept of 'fear of fear' often used to explain the etiology and maintenance of severe anxiety disorders such as agoraphobia and anxiety neurosis (Beck and Emery, 1979; Evans, 1972; Frankl, 1975; Freud, 1895; Fenichel, 1945; Goldstein and Chambless, 1978; Reiss and McNally, 1985; Shands and Schor, 1982; Westphal, 1871).

Empirical evidence supporting the psychophysiological model of panic is still largely lacking [for a review see Margraf *et al.* (1986a)], although some recent data indicate that unpleasant body sensations are present before the patient experiences panic. In an interview study of anxiety patients, Hibbert (1984) found that the reaction to somatic symptoms such as palpitations and breathlessness and their interpretation as dangerous is an essential component of naturally occurring panic attacks. The most frequent sequence of events during panic attacks was the perception of an unpleasant bodily event followed by anxious catastrophizing cognitions and finally the full-blown panic attack. Beck (1985) reports similar findings in his patients. In the same vein, Ley (1985) found that somatic symptoms preceded fear in the majority of patients interviewed. Questionnaire studies showed that agoraphobic patients, the majority of whom suffer from panic attacks, report high 'fear of fear' in the sense of fear of body sensations associated with anxiety

(Chambless, Caputo, Bright and Gallagher, 1984; Reiss, Peterson, Gursky and McNally, 1986). However, so far there is no direct experimental test of the positive feedback hypothesis.

The goal of the present study was to test the hypothesis that the appraisal of physiological arousal will induce anxiety in patients suffering from panic attacks, which is one of the underlying assumptions of the psychophysiological model. Furthermore, we predicted on the basis of the 'fear of fear' concept that panic patients are more prone to respond in this way than are normal controls. We gave *S*s false feedback of an abrupt heart rate increase to manipulate their perception of their physiological state (cf. Ackerman and Sachar, 1974). We chose this manipulation since palpitations are the most commonly reported symptom of naturally occurring panic attacks (Barlow, Vermilyea, Blanchard, Vermilyea, DiNardo and Cerny, 1985; Margraf, Taylor, Ehlers, Roth and Agras, 1987b). The false heart rate feedback paradigm was introduced by Valins (1966, 1967) into the study of emotion and has been shown in many experiments to influence *S*s' affective appraisal of various stimuli given at the same time as the feedback [for reviews see Harris and Katkin (1975), Hirschman and Clark (1983), Liebhart (1980) and Parkinson (1985)]. We modified Valins' paradigm in that we tried to create an experimental analogue of sudden, unexplained heart rate changes and in that we assessed affective changes by direct reports of emotional state.

Two control strategies were used in this experiment. First, true heart rate feedback was given prior to the false feedback of a heart rate acceleration to check for possible differences in the responses of patients and controls to hearing feedback of their heart beat. Second, the accuracy of the *S*s' heart rate perception was assessed since it might represent an important mediating factor in the credibility of the false feedback procedure. Previous research has shown that there are large interindividual differences in heart beat perception (Carroll and Whellock, 1980; Schandry, 1981; Katkin, 1985). The patients' ability to perceive their heart rate is of interest since there is some reason to assume that patients with panic attacks could be more aware of their physiological functions than normal controls. In self-reports, panic patients describe high awareness of their heart beat and other visceral functions (King, Margraf, Ehlers and Maddock, 1986). Consistent with this, Tyrer, Lee and Alexander (1980) found that patients with anxiety neurosis gave more accurate ratings of their heart rate changes than phobic patients.

METHOD

Subjects

Twenty-five panic attack patients (22 women, 3 men) and 25 control *S*s (24 women, 1 man) were recruited from the community by newspaper advertisements. All subjects were Caucasian. Patients were recruited to participate in a treatment study. Controls were paid for their participation.

Patients met DSM-III criteria for panic disorder or agoraphobia with panic attacks as determined by the Structured Clinical Interview for DSM-III—Upjohn Version (SCID-UP, Spitzer and Williams, 1983). Patients were excluded if they had a history of major depressive episodes that preceded the onset of panic attacks. Five patients were diagnosed as having agoraphobia with panic attacks, 13 as having panic disorder with limited phobic avoidance, and seven as having uncomplicated panic disorder. The average duration of the disorder was 4.6 ($\pm$4.8) yr. All patients had experienced at least one panic attack for each of the 3 weeks prior to entering the study. For the week prior to testing, patients reported an average of 3.5 ($\pm$2.6) panic attacks and an average of 2.0 ($\pm$1.9) other anxiety episodes with fewer symptoms as determined by a self-report diary. Patients had mean Hamilton Anxiety Rating scores (Hamilton, 1959) of 17.3 ($\pm$7.7) and mean Hamilton Depression Rating scores (Hamilton, 1967) of 10.8 ($\pm$6.8). At the time of testing, patients had completely discontinued taking any psychotropic medication for at least 10 days.

Control *S*s described themselves as 'non-anxious'. They had to be free of any history of psychiatric problems as determined by the SCID-UP and a structured interview based on the SADS-L (Spitzer and Endicott, 1978) for diagnoses not covered by the SCID-UP. Controls were not taking psychoactive medications.

The groups were matched for age, ranging from 21 to 59 in the patient, and from 22 to 59 in the control group. Eleven patients (10 controls) were married, 11 (eight) had never been married, and three (seven) were divorced. Fourteen patients (16 controls) had full-time employment, three (four) worked part time, five (three) were housewives, and three (two) were unemployed. All *S*s

Table 1. Subjects

	Patients	Controls
N	25	25
Age (yr)	34.6 (9.0)	35.8 (10.2)
Height (cm)	165.4 (8.2)	166.6 (7.0)
Weight (kg)	64.2 (11.4)	60.6 (7.8)
Education (yr)	14.0 (2.2)	15.7 (2.5)
Anxiety (STAI/S)	41.5 (13.0)	28.8 (4.5)
Depression (BDI, short form)	6.4 (4.9)	1.2 (1.6)
Avoidance (MI)		
accompanied	1.6 (0.7)	1.0 (0.3)
alone	2.3 (0.9)	1.1 (0.5)

Means (S.D.s) are presented.

were in good physical health as determined by medical history and physical examination. Seven patients and six controls were smokers. None of the *S*s had taken alcoholic beverages or recreational drugs on the day of testing. Table 1 shows mean age, height, weight, Beck Depression Inventory scores (short version, Beck and Beck, 1972; cf. Beck, Ward, Mendelsohn, Mock and Erbaugh, 1961), state anxiety scores in the State–Trait Anxiety Inventory (Spielberger, Gorsuch and Lushene, 1970) and Mobility Inventory scores [a measure of agoraphobic avoidance behavior; Chambless, Caputo, Jasin, Gracely and Williams (1985)] for patients and controls. Informed consent was obtained from all *S*s after the nature of the procedures had been explained.

Procedure

General procedure. Test sessions took place in the afternoon between one and five o'clock. The experimental setting was a sound-attenuated, electrically shielded chamber. The *S* sat alone and could not see the experimenter during the test periods, but could communicate with her by intercom at any time. Between the different test periods described below, the experimenter came into the chamber to administer self-report scales and to give instructions. The experimenter allowed the *S*s time to familiarize themselves with the laboratory environment. Subjects had spent more than 1 hr in the laboratory before recording was started.

Baseline. Baseline levels of the dependent variables were recorded at the beginning of the laboratory session, that is, prior to the heart rate feedback paradigm. Subjects were instructed to sit quietly with their eyes open. Blood pressure was taken automatically every 4.5 min. The last 5 min of this 15-min recording period were used as baseline levels.

Heart rate feedback. The false heart rate feedback manipulation was introduced after *S*s had received true heart rate feedback for 5 min. There was no break between these conditions, that is, *S*s sat alone in the recording chamber during the entire feedback run. The order of true and false feedback was not balanced because we were concerned that increases in anxiety or even panic attacks induced by false feedback might last so long that they would carry over into the true feedback portion of the paradigm when true feedback came second. The auditory feedback was a 1000-Hz tone pip. During the 5 min of *true* feedback the pip was triggered by the R-wave of the EKG. Thus, *S*s heard a tone with every heart beat. Automatic measurements of blood pressure and self-ratings of anxiety were taken at minutes 1, 3, and 5. At min 5, the experimenter switched to *false* feedback. False feedback was produced by a function generator that generated electrical pulses at regular intervals. The switching was done while the *S* filled out the third anxiety rating scale (AR, see Assessment section). For approximately 1 min, the function generator was set at a frequency equal to the *S*'s heart rate during the last 2 min of true feedback. At min 6:20, the experimenter gradually increased the frequency of the function generator over 30 s the equivalent of 50 beats/min (bpm). Similar heart rate increases were found in natural panic attacks by Lader and Mathews (1970) and Cohen, Barlow and Blanchard (1985). Blood pressure was taken at the end of the 'heart rate increase' (min 7) and *S*s filled out another AR. At min 7:10, the experimenter gradually decreased the frequency of the function generator back to the original level and recording was stopped at min 8. After the test, *S*s were asked to write down comments on the heart rate feedback (pitch, volume, accuracy) and their reaction to it. This provided information on whether *S*s noticed the feedback manipulation.

Heart rate perception test. After the baseline and at the end of the session, a heart rate perception test was given. The purpose of this test was twofold. It provided an assessment of how accurately *S*s were able to perceive their heart rates, and it gave a rationale for the heart rate feedback procedure. Subjects were instructed to match the rate of a train of tone pips to their heart rate without actually taking their pulse [similar to the procedure used by Porges and Raskin (1969)]. They were given 1 min to complete this task using a dial with a range from 1 to 10 which was set at 5 (equalling 60 bpm) before the *S* started.

Instructions. Identical, written instructions were used for patients and controls. Subjects were told that one of the purposes of the session was to find out how accurately people can perceive their heart rate and whether the accuracy can be enhanced by heart rate feedback. Before the heart rate feedback run, *S*s were not explicitly informed about the possibility of false feedback. Instructions and the informed consent form referred to 'more or less accurate feedback'. To enhance the credibility of the feedback paradigm, the experimenter had the *S*s briefly stand up in the break prior to this run while true feedback was on and demonstrated how heart rate increased with changes in posture. For ethical reasons, instructions mentioned the possibility that the *S*s might feel increases in anxiety with any of the tasks.

Assessment

Self-reports. Subjects repeatedly filled out an Anxiety Rating Scale (AR). This scale asks *S*s to rate themselves for 'anxiety' and 'excitement', each on a scale from 0 (labelled *none*) to 10 (labelled *extreme*). The rating of excitement was included because patients and controls might differ in their ways of labelling arousal. Subjects completed an AR each time after blood pressure had been taken. They were given a supply of the scales prior to the test run. All *S*s were able to follow these instructions.

Physiological measures. The EKG was continously recorded from electrodes over the 10th left rib on the midclavicular line and the right mastoid. Skin conductance was continously recorded from a pair of Ag–AgCl disc electrodes, 0.8 cm^2 in area, both placed on the thenar eminence of the nondominant hand. The electrode medium was a mixture of creamy ointment and physiological saline as recommended by Fowles, Christie, Edelberg, Grings, Lykken and Venables (1981). Subjects rinsed their hands with water alone before the electrodes were applied. The skin conductance transducer applied a constant 0.5 V across the electrodes. Blood pressure (BP) was measured automatically (Accutorr 2, Datascope Corp.). Averages for heart rate (HR) are based on a beat-to-beat analysis following Graham (1978). Skin conductance level was log transformed for the statistical analysis as recommended by Venables and Christie (1980).

RESULTS

Subjects

One patient had to be excluded from the data analysis because she fell asleep during the true heart rate feedback. Due to a technical error, HR and SCL of one control *S* were not recorded. Five patients and nine controls noticed that the false feedback did not accurately reflect their HR and had to be excluded from the analysis of the false feedback manipulation [chi square (1) = 0.74, nonsignificant]. Thus, the analysis of the false feedback compared 19 patients and 16 controls (15 for HR and SCL) who believed that the feedback reflected their heart rate accurately or who just felt surprised that 'their heart rate' speeded up without them noticing any reasons for it. There were too few *S*s that doubted the feedback accuracy to perform a statistical analysis including both doubters and non-doubters. We chose to use only descriptive statistics on doubters.

Effects of false heart rate feedback

To assess the effects of the *false* heart rate feedback manipulation, we compared the AR and BP responses during the false feedback of a heart rate increase with the average of the three assessments during true feedback. For HR and SCL, 90-sec averages were calculated that represented the 30-sec false heart rate increase and the following minute. Three corresponding 90-sec averages were calculated and averaged that represented the three assessments during true feedback. A hierarchical approach was used for the statistical analysis. The responses of patients

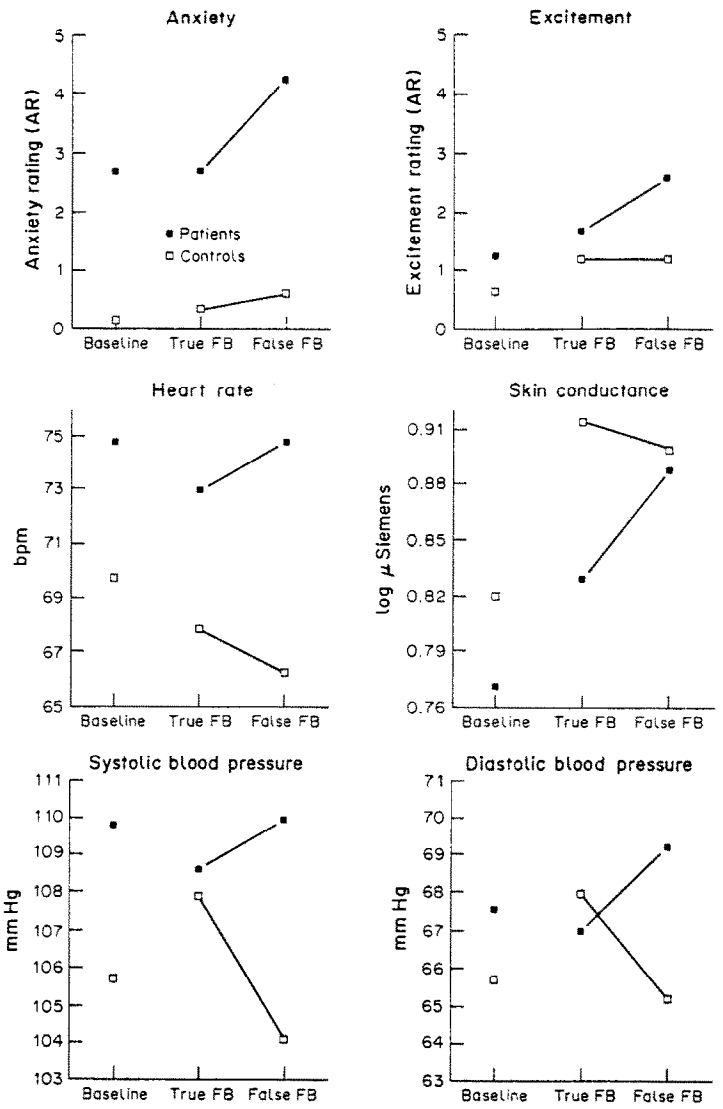

Fig. 1. Responses of panic patients (■) and normal controls (□) to false feedback of heart rate acceleration. Reactions to the false feedback are compared with the average of three assessments during true heart rate feedback. Baseline levels are indicated. Results for self-rated anxiety and excitement (each on a scale from 0 to 10), heart rate (in bpm), skin conductance level (in log microSiemens), and systolic and diastolic blood pressure (in mmHg) are presented.

and controls to the false feedback manipulation first subjected to an overall comparison using multivariate analyses of variance (MANOVA) (cf. O'Brien and Kaiser, 1985). Different reactions of patients and controls would be seen in significant interactions between the *condition* (true vs false feedback) and *group* (patients vs controls) variables. The six dependent measures (self-reported anxiety and excitement, heart rate (HR), skin conductance level (SCL), systolic (SBP) and diastolic blood pressure (DBP)) were used as a within-variable in the MANOVA. Different changes in the different measures would be seen in significant interactions with the *measure* variable. Significant main effects and interactions were further evaluated using repeated measures analyses of variance (ANOVA) and paired *t*-tests.

Figure 1 shows the results of the false heart rate feedback condition. Responses after the false feedback of a heart rate increase are compared with the average responses during true feedback. In addition, baseline levels are indicated. Note that the baseline did not directly precede the true feedback, whereas true and false feedback were presented in the same run. The MANOVA showed a highly significant Group × Condition interaction [$F(1,32) = 11.67$, $P < 0.005$]. The group

Table 2. Results of false feedback

Variable	Group (G) (Patients vs controls)	Condition (C) (True vs false feedback)	G × C
Anxiety	$P < 0.0001$ $F(1,33) = 40.60$	$P < 0.0005$ $F(1,33) = 15.07$	$P < 0.01$ $F(1,33) = 7.48$
Excitement	$P < 0.05$ $F(1,33) = 4.18$	$P < 0.05$ $F(1,33) = 4.88$	$P < 0.05$ $F(1,33) = 4.88$
Heart rate	$P < 0.05$ $F(1,32) = 4.41$	n.s.	$P < 0.05$ $F(1,32) = 4.31$
Skin conductance level	n.s.	$P < 0.11$ $[F(1,32) = 2.78]$	$P < 0.01$ $F(1,32) = 7.99$
Systolic blood pressure	n.s.	n.s.	$P < 0.05$ $F(1,33) = 6.73$
Diastolic blood pressure	n.s.	n.s.	$P < 0.01$ $F(1,33) = 7.78$

Results of univariate repeated measures ANOVAs are presented for *S*s who believed the false feedback was accurate.

effect was marginally significant [$F(1,32) = 3.69$, $P < 0.07$]. There were highly significant interaction effects with the measure variable [Group × Measure interaction $F(5,28) = 8.63$, $P < 0.0001$; Condition × Measure interaction $F(5,28) = 4.38$, $P < 0.005$; Group × Condition × Measure interaction $F(5,28) = 2.70$, $P < 0.05$].

The results of the subsequent univariate ANOVAs are shown in Table 2. On all of the measures, patients and controls differed in their responses to the false feedback. For self-rated anxiety and excitement, and SCL, the interactions resulted from increases in the patient group only ($t = 3.97$, $P < 0.001$; $t = 2.51$, $P < 0.05$; $t = 4.13$, $P < 0.001$, respectively). On the cardiovascular measures (HR, SBP and DBP), controls showed decreases in response to the false feedback ($t = -4.28$, $P < 0.001$; $t = -3.34$, $P < 0.005$; $t = -2.19$, $P < 0.05$, respectively). The increases in patients seen in Fig. 1 were not statistically significant.

Examples of patients' written comments on the feedback were: "The faster the beat the more anxious I became", "I got a little nervous in the end when my heart rate increased for a period of time", "My heart rate speeded up with some thoughts and it frightened me somewhat and made me confused and scared", "It made me remember the death of my mother and brother", "I wondered why the heart rate speeded up after a while. It reminded me of being in a hospital", and "At one point the rate picked up and I began to feel very nervous about it . . . I thought: "Oh no, I'm going to have a heart attack". These statements are in contrast to the comments of control *S*s like "I was quite surprised when the rate suddenly became faster towards the end of the test", "Some of the fluctuations surprised me", "I was surprised when the feedback speeded up in the second part of the run because I didn't understand why my heartbeat was faster", or "It didn't make me feel anxious when it sped up. It seemed to have a slight hypnotic effect on me."

One patient volunteered to the experimenter that she had a panic attack after hearing the false feedback. This case has been described in more detail elsewhere (Margraf, Ehlers, and Roth, 1987a). We did not expect that false heart rate feedback would trigger full-blown panic attacks because feedback through an external speaker is only a weak analogue of interoception. Following the psychophysiological model, we expected anxiety increases in our panic disorder *S*s, but we did not expect that these anxiety increases would be large enough to escalate into a full-blown panic attack. The false feedback was only one of many pieces of external and internal information, many of which were reassuring (for instance, that the physiological changes were monitored, being in a hospital). Therefore, we did not expect that our manipulation would be powerful enough to trigger panic. For these reasons we did not ask our *S*s routinely whether they had experienced panic attacks. Instead, we measured self-reported anxiety and physiological changes. It is possible that our experience with lactate infusions (Ehlers *et al.*, 1986) and CO_2 inhalations (Ehlers *et al.*, 1987a) made us careful in our predictions. These agents are very powerful in producing bodily symptoms, and even in these conditions many patients say that they did not panic. The anxiety levels they reach with these manipulations are also not extreme (6–7 on a scale from 0 to 10).

Effects of true heart rate feedback

To check whether heart rate feedback in itself has any effects on anxiety or the physiological variables measured, we compared *S*s' responses during *true* heart rate feedback with their baseline

Table 3. Results of true feedback

Variable	Group (G) (Patients vs controls)	Condition (C) (Baseline vs true feedback)	G × C
Anxiety	$P < 0.0001$ $F(1,47) = 28.84$	n.s.	n.s.
Excitement	$P < 0.05$ $F(1,47) = 4.57$	$P < 0.0005$ $F(1,47) = 16.38$	n.s.
Heart rate	$P < 0.05$ $F(1,46) = 4.53$	$P < 0.0001$ $F(1,46) = 27.49$	n.s.
Skin conductance level	n.s.	$P < 0.0001$ $F(1,46) = 20.31$	n.s.
Systolic blood pressure	n.s.	n.s.	n.s.
Diastolic blood pressure	n.s.	n.s.	n.s.

Results of univariate repeated measures ANOVAs for the entire patient and control groups are presented.

Table 4. Heart rate perception tests

	Test 1 (Before feedback)	Test 2 (After feedback)
Patients	12.5 (9.2)	13.2 (9.4)
Controls	12.7 (10.6)	6.2 (5.6)

Means (S.D.s) of the absolute difference between estimated and actual heart rate (error scores) are presented.

levels. Averages were calculated for each of the dependent measures representing the baseline and the 5 min of true heart rate feedback, respectively. A repeated measures MANOVA was calculated comparing the responses of patients and controls (variable group) to true feedback compared to baseline (variable condition). The six different measures represented the measure variable. Subsequent analyses included repeated measures ANOVAs when appropriate. This analysis was performed in two ways. First, we compared the entire patient group with the entire control group. Second, we analysed only the responses of *S*s who subsequently believed in the accuracy of the false feedback manipulation to check whether this group had a typical response pattern to true feedback.

The MANOVA showed a marginally significant group effect [$F(1,46) = 3.29$, $P < 0.08$]. There was no condition effect or Group × Condition interaction. In addition, there were significant interaction effects due to the different dependent measures used [Group × Measure interaction $F(5,42) = 7.61$, $P < 0.0001$; Condition × Measure interaction $F(5,42) = 8.19$, $P < 0.0001$].

Results of the subsequent univariate ANOVAs are shown in Table 3. Patients had higher HR and gave higher anxiety and excitement ratings than controls during both conditions. Patients and controls responded similarly to the true heart rate feedback. During true feedback, both groups had higher SCL and gave higher excitement ratings, but had lower HR than at the end of the baseline. Blood pressure and self-reported anxiety did not change.

The results were parallel when only the subgroup of *S*s was analysed that subsequently did not notice that the feedback of a heart rate increase was false. The exception was a marginally significant Group × Condition interaction [$F(1,32) = 3.32$, $P < 0.08$]. Univariate analyses showed a marginally significant interaction for systolic blood pressure [$F(1,33) = 3.67$, $P < 0.07$] resulting from increases in the control group only (see Fig. 1).

Results of the heart rate perception tests

The accuracy of heart rate perception was assessed by the absolute difference of estimated and actual heart rate for each of the two heart rate perception tests, respectively (error score). The accuracy of patients and controls in the two tasks (variable group) and possible changes that occurred during the session (variable time) were compared using repeated measures ANOVA. The analysis was done in two ways, using all *S*s or using only those *S*s who had not doubted the accuracy of the false feedback. Pearson correlation coefficients were calculated between the error score and the actual HR.

The results of the heart rate perception tests are shown in Table 4. Note that larger numbers reflect greater inaccuracy. There was no difference between the groups in the accuracy of their estimation at the beginning of the session (no group effect in the ANOVA). During the second assessment at the end of the session, after *S*s had received heart rate feedback, controls, but not

patients gave more correct estimates than at the beginning of the session [time effect $F(1,46) = 4.01$, $P < 0.06$; Group × Time interaction $F(1,46) = 6.36$, $P < 0.05$]. The results were the same when only those *S*s were analysed who subsequently did not notice that the feedback was false [time effect $F(1,32) = 3.93$, $P < 0.06$; Group × Time interaction $F(1,32) = 5.06$, $P < 0.05$]. The majority of *S*s underestimated their heart rates (18 of 24 *S*s in each group, Test 1).

Error scores during both heart rate perception tests were correlated with actual HR in both groups ($r = 0.60$ and $r = 0.48$ for patients; $r = 0.41$ and $r = 0.70$ for controls, all Ps < 0.05). Therefore, additional ANCOVAs of the error scores were performed, using the actual HR as the covariate. In these analyses, Group × Time interactions were only marginally significant [$F(1,45) = 4.00$, $P < 0.06$ for the entire sample; $F(1,31) = 3.70$, $P < 0.07$ for non-doubters].

Subjects doubting the accuracy of false feedback

Descriptive statistics indicate that *S*s who noticed that the feedback was false had a qualitatively different response pattern from those who believed it was accurate. The five patients showed average decreases of 6.9 mmHg in SBP and of 0.8 units in self-rated excitement, and an increase in SCL of 0.045 log microSiemens. The nine controls had a decrease of 1.6 mm Hg in SBP, and increases of 1.1 units in self-rated anxiety and of 0.022 log microSiemens in SCL. There were no changes in the other variables. Two controls wrote that their anxiety had increased for a short time while they were wondering whether the equipment was working properly.

The *S*s who noticed that the feedback was false were not more accurate in the heart rate perception test. In fact, in none of the variables that we assessed during baseline or true feedback did these *S*s appear different from those who believed the feedback was accurate.

DISCUSSION

Our results show that false feedback of a heart rate acceleration can induce anxiety and physiological reactions in patients suffering from panic attacks. Patients showed subjectively and physiologically different responses to the false feedback from matched control *S*s. The response pattern of patients was in the direction of increased subjective and physiological arousal on all measures, although changes in the cardiovascular measures were not statistically significant. In contrast, controls showed decreases in the cardiovascular measures and did not report increases in subjective anxiety and excitement.

These differential reactions of patients and controls were specific to the false feedback condition since they did not occur during true heart rate feedback. Furthermore, they are not likely to be differential time effects, since for most variables, the false feedback values did not appear to be an extrapolation of the time trend between baseline and true feedback. During true feedback, both groups reported higher excitement and had higher skin conductance levels than at baseline. This response pattern probably reflects the novelty of being presented with another paradigm.

Overall, our results are consistent with one of the basic assumptions of the psychophysiological model of panic disorder (Beck, 1985; Clark, 1986; Margraf *et al.*, 1986a): the belief that bodily changes have occurred can lead to increases in anxiety and physiological arousal in this patient group. The comments given by our *S*s are in line with this interpretation. Our results are in line with the interview data of Hibbert (1984) and Ley (1985) and the treatment effects of cognitive behavioral therapy reported by Griez and van den Hout (1983, 1986) and Clark, Salkovskis and Chalkley (1985).

It is unlikely that the larger responses of panic patients to the false heart rate feedback reflects a general tendency to over-react to aversive stimulation. Research from our own group and other research centers did not find patients with panic attacks to be generally over-responsive to neutral, midly aversive and aversive stimulation (Grunhaus, Gloger, Birmacher, Palmer and Ben-David, 1983; Ehlers, Margraf, Roth, Taylor, Maddock, Sheikh, Kopell, McClenahan, Gossard, Blowers, Agras and Kopell, 1986; Ehlers, Margraf and Roth, 1987a; Roth, Telch, Taylor, Sachitano, Gallen, Kopell, McClenahan, Agras and Pfefferbaum, 1986). In interpreting the differential reactions of patients and controls to the false feedback, we have to bear in mind, however, that patients had higher anxiety and heart rate levels throughout the session. It is conceivable that controls would have shown similar reactions if they had started at similar levels. These baseline differences are a

basic problem in anxiety induction studies comparing patient and control populations (Ehlers *et al.*, 1986; Margraf *et al.*, 1986b). One way of studying the role of baseline differences would be to elevate controls to patient levels of anxiety and cardiovascular arousal prior to giving the feedback manipulation [for instance, by caffeine ingestion; Uhde, Boulenger, Jimersonn and Post (1984)]. Whatever the effect of different baselines might be, these differences are the ordinary state of panic patients outside the laboratory as well as inside (Ehlers *et al.*, 1987b; Hoehn-Saric, 1981, 1982; Roth *et al.*, 1986).

We cannot rule out that demand characteristics of the experimental situation were partly responsible for our results. Although we limited interactions of *S*s with the experimenter and tried to create the same expectancies in patients and controls, it is possible that patients might have interpreted our instructions and might have responded to the experimental setting in a different way. One approach to studying the impact of demand characteristics would be to systematically vary the expectancies by various instructions. However, even if differential expectancies account for some of the differences we found between patients and controls, our results would still reflect the natural reaction of patients, since anxious patients are more prone to anxious expectations than normal controls and tend more to focus their attention on danger cues (Butler and Mathews, 1983; MacLeod, Mathews and Tata, 1986; Mathews and MacLeod, 1985, 1986).

Our study showed that panic patients become anxious when they believe their heart rate has accelerated. Can we generalize this result to real heart rate acceleration or other cardiac sensations these patients might perceive before they go on to panic? There are several problems with this generalization. First, HR increases of this magnitude are only found in a subgroup of naturally occurring panic attacks (Taylor, Sheikh, Agras, Roth, Margraf, Ehlers, Maddock and Gossard, 1986; Margraf *et al.*, 1986b). In pilot *S*s we had experimented with false feedback of smaller and less abrupt heart rate increases and had found that *S*s often did not notice these changes in the feedback tone. Second, even with a 50 bpm increase, the majority of patients did not notice that the feedback was false. Furthermore, the results of our heart rate perception tests indicate that both patients and controls had poor heart rate perception before feedback was given. Untrained *S*s in general and women in particular have poor heart beat perception (Katkin, 1985).

However, there are counter-arguments that suggest the generalization may be valid. First, as for the magnitude of heart rate changes, false feedback is only a rather weak experimental analogue of internal perceptions. Subjects could probably ignore the feedback tone much more easily than cardiac sensations in their own bodies. One patient wrote: "I was successful in detaching myself from the sound, i.e. did not associate it with me". Furthermore, the palpitations reported in descriptions of panic attacks might correspond to the perception of stroke volume or changes in pulse pressure more than simple heart rate changes. Thus, our large artificial heart rate change may be equivalent to a much smaller true heart rate change accompanied with other cardiac sensations.

Second, as for the accuracy of heart rate perception, the presence of external stimuli could have distracted *S*s from their own cardiac sensations during our paradigms and made them unaware of their true HR. Furthermore, accurate heart rate perception is not an essential assumption of the psychophysiological model. Panic patients may haphazardly overinterpret and over-respond to some change in cardiac function that suddenly becomes perceptible to them, for instance after exercise, caffeine consumption, or changes in posture. Thus, the sharpened cardiac awareness reported by panic patients (King *et al.*, 1986) may reflect patients' beliefs more than better cardiac perception. This interpretation is in line with studies using the Autonomic Perception Questionnaire (APQ; Mandler, Mandler and Uviller, 1958) that found no consistent relationship between self-reported and objectively measured cardiac perception (McFarland, 1975; Whitehead, Drescher, Heiman and Blackwell, 1977).

In conclusion, our results support the hypothesis that the appraisal of physiological change can play a role in the subjective experience of anxiety in patients with panic disorder. Our finding of different responses of panic attack patients and normal controls to false feedback of heart rate acceleration has implications for the maintenance and perhaps development of panic attacks. It is consistent with the idea that panic patients have more fear of certain body sensations associated with anxiety than controls. Thus, although we did not directly show that physiological changes can trigger anxiety responses in this patient group we showed that only patients become anxious when they believe these changes have occurred. It would be desirable to test other patient groups with

this paradigm. We would expect that some groups, like hypochondriacal patients, would show responses similar to those of panic patients whereas others, like simple phobics, should not differ from normal controls. Although the physiological changes induced by our experimental manipulation in panic disorder patients were rather small, their direction is consistent with the hypothesis of a positive feedback cycle between anxiety and physiological symptoms. That the perception of bodily events need not be accurate further emphasizes the relevance of cognitive factors in panic disorder.

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