

ANXIETY INDUCED BY CARDIAC PERCEPTIONS IN PATIENTS WITH PANIC ATTACKS: A FIELD STUDY

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Summary—In panic disorder bodily sensations appear to play an important role as a trigger for anxiety. In our psychophysiological model of panic attacks we postulate the following vicious circle: individuals with panic attacks perceive even quite small increases in heart rate and interpret these changes as being catastrophic. This elicits anxiety and a further increase in heart rate. To evaluate this model we conducted a field study of 28 subjects with panic attacks and 20 healthy controls. A 24 hr ambulatory ECG was recorded and the subjects were instructed to report any cardiac perceptions during this period and to rate the anxiety elicited by these perceptions. The incidence of cardiac perceptions was about the same in both groups, but only subjects with panic attacks reported anxiety associated with such perceptions. Analysis of the ECGs revealed that in both groups heart rate accelerations preceded cardiac perceptions. Following cardiac perceptions, the healthy controls showed a heart rate deceleration, whereas the subjects with panic attacks had a further acceleration. This heart rate increase after cardiac perceptions was positively related to the level of anxiety elicited by the perceptions. These results provide clear evidence in support of the vicious circle model of panic attacks.

INTRODUCTION

The perception of bodily symptoms plays an important role in the development and maintenance of many psychiatric and psychosomatic disorders. In panic disorder bodily perceptions are thought to trigger panic attacks (Barlow, 1986; Clark, 1986; Ehlers, Margraf & Roth, 1988a; van den Hout, 1988; Rachman, Levitt & Lopatka, 1987). The 'catastrophic' interpretation of bodily symptoms elicits anxiety, and this in turn leads to an increase in physiological activity. The result is an amplification of the bodily symptoms. This positive feedback loop between internal perceptions and physiological activity can cumulate in a panic attack (Clark, 1986; Ehlers, Margraf, Roth, Taylor & Birbaumer, 1988b; Margraf, Ehlers & Roth, 1987). Additionally, the view is generally held that panic patients focus their attention selectively on internal sensations and are therefore prone to perceiving bodily symptoms (Ehlers *et al.*, 1988a; King, Margraf, Ehlers & Maddock, 1986; Tyrer, Lee & Alexander, 1980). Very similar hypotheses have been formulated to explain hypochondriasis (Barsky & Klerman, 1983; Kellner, 1986; Warwick and Salkovskis, 1990) or cardiac neurosis (Liebhart, 1974; Sachse, 1984).

Within the group of patients with panic attacks there seems to exist a subgroup of patients with cardiac-related anxiety (Kellner, 1986; Maier, Buller, Rieger & Benkert, 1985; Nutzinger, 1987; Nutzinger, Zapotoczky, Cayiroglu & Gatterer, 1987). These individuals usually experience panic attacks and as a result develop intense anxiety about having a heart attack or about developing some other cardiac disease. We postulate that in these patients the development and maintenance of panic attacks can be explained in terms of a vicious circle (Fig. 1). Following an initial panic attack the patients selectively monitored their cardiac activity. Subsequently, if they perceive a heart rate acceleration, they interpret it catastrophically as a sign of a cardiac disease or a heart attack. These cognitions elicit anxiety, which leads to a further heart rate acceleration. This positive feedback loop can culminate in a panic attack.

Ehlers *et al.* (1988b; cf. Sachse, 1984) tried to validate such a positive feedback loop empirically in panic patients using the 'false heart rate feedback' paradigm developed by Valins (1966). Their Ss were given heartbeat feedback suggesting a sudden and sharp increase in heart rate. As a

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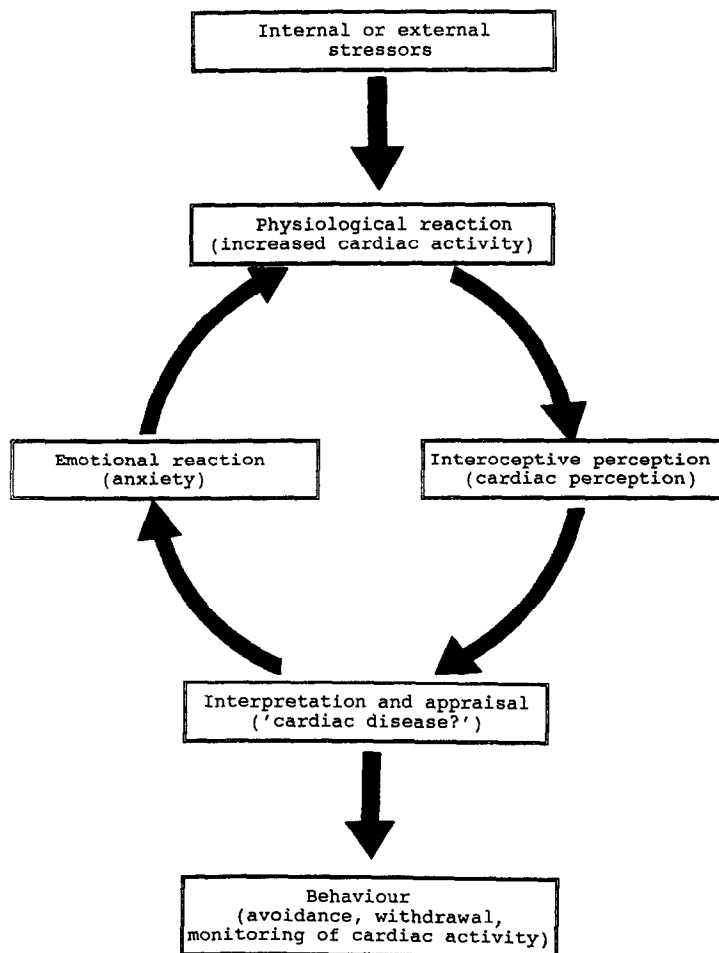


Fig. 1. A vicious circle model of panic attacks.

consequence of this manipulation, the patients showed higher anxiety ratings and greater physiological arousal (heart rate, skin conductance) than the healthy controls.

There are two problems with the false heart rate feedback paradigm, as Ehlers *et al.* themselves point out. First, it is unclear whether the results can be generalized to real interoceptive cardiac perceptions. Second, the results may be confounded with baseline effects: the panic patients had higher baseline anxiety scores than the healthy controls.

In the present study we avoided these problems by using a field approach with ambulatory ECG monitoring. Our method allows the registration of subjective and physiological arousal caused by spontaneously occurring cardiac perceptions. In patients with panic attacks and subsequent cardiac-related anxiety we expected to find clear associations among cardiac perceptions, anxiety, and cardiac activity.

The aim of the present study was to show that the perception of changes in cardiac activity can elicit anxiety in patients with panic attacks. The following hypotheses were tested:

- (1) Patients with panic attacks report cardiac perceptions more frequently than healthy controls.
- (2) Cardiac perceptions elicit more intense anxiety in patients with panic attacks than in healthy controls.
- (3) After cardiac perceptions patients with panic attacks have a faster heart rate than healthy controls.
- (4) In patients with panic attacks there is a positive association between the level of anxiety and the heart rate increase after cardiac perceptions.

METHODS

Subjects

The *Ss* were 28 patients with panic attacks and 20 healthy controls. The criteria for inclusion in the group of patients with panic attacks were:

(1) An initial panic attack fulfilling the DSM-III-R (American Psychiatric Association, 1987) criteria for a panic attack.

(2) Cardiac-related anxiety (e.g. anxiety about a cardiac disorder or fear of having a heart attack) that developed as a result of an anxiety attack and lasted for at least 2 months. The cardiac-related anxiety persisted even through a thorough examination yielded no organic cause and the patient was informed of this. The healthy controls were recruited through an advertisement and were paid for participating.

All *Ss* underwent a psychiatric evaluation, a physical examination, an echocardiogram and blood sampling for endocrine tests. They were included in the study only if they were between 20 and 55 yr of age and weighted between 50 and 100 kg. Other exclusion criteria were mitral valve prolapse, thyroid disease, diabetes mellitus, hypertension or other cardiovascular disorders, and other psychiatric disorders.

The differences between the two groups with respect to gender, age, height and weight are not sufficient. As expected, the groups differed in their state and trait anxiety scores (STAI, German version, Laux, Glanzmann, Schaffner & Spielberger, 1981), in physical complaints (B-L, von Zerssen, 1976a) and in subjective well-being (Bf-S, von Zerssen, 1976b) (Table 1). The questionnaire scores for the healthy controls were all in the normal range, whereas the scores for the patients with panic attacks were comparable to those found for such patients in previous studies (von Zerssen, 1976a; Egle, Hoffmann & Wenzel, 1988).

Procedure

On arrival in the examination room the *Ss* were informed about the experimental procedure. The ECG electrodes were then attached. The *Ss* spent the next 20 min filling out the aforementioned questionnaires and afterwards proceeded with a heartbeat perception test, described elsewhere (Pauli, Hartl, Marquardt, Stalman & Strian, 1990). For the 24 hr ECG recording the *Ss* were instructed to mark any cardiac perceptions on the event track of the recorder by pressing the event button and to rate the anxiety elicited by the cardiac perceptions on a 4-point rating scale (1 = no anxiety, 4 = intense anxiety). In addition they were asked to record the current activities every hour. Except for strenuous physical activity (e.g. sports), they were allowed to carry out their normal daily routine. The ECG apparatus was removed 24 hr later.

Apparatus

The electrocardiogram was recorded by means of two precordial leads on C-120 tapes in a portable ECG-recorder (FM-Recorder MR-20, Oxford Medical System Ltd, U.K.) with a maximum run of 24 hr. The channel with fewer artifacts was selected for evaluation. The event track of the recorder was used by the *Ss* to mark cardiac perceptions.

Table 1. Subjects

	Controls	Patients	Tests
Sex			
male	13	17	Chisq(1) = 0.00
female	7	11	NS
Age (yr)	31.8 ± 7.8	35.3 ± 7.0	<i>t</i> = 1.6 NS
Height (m)	1.7 ± 0.1	1.7 ± 0.1	<i>t</i> = 0.01 NS
Weight (kg)	67.5 ± 11.2	73.8 ± 12.0	<i>t</i> = 1.8 NS
State anxiety	34.8 ± 9.5	49.4 ± 14.4	<i>U</i> = 3.8 <i>P</i> < 0.001
Trait anxiety	33.7 ± 9.6	55.3 ± 11.2	<i>U</i> = 5.7 <i>P</i> < 0.001
Physical complaints	8.8 ± 6.4	39.6 ± 10.3	<i>U</i> = 3.6 <i>P</i> < 0.001
Well-being	10.9 ± 8.4	25.1 ± 11.8	<i>U</i> = 4.9 <i>P</i> < 0.001

Means ± standard deviations are presented.

Chisq = χ^2 test.

t = *t*-test.

U = Mann-Whitney *U*-test.

Digitalization of the ECG

The ECG channel with fewer artifacts was selected for evaluation on the analyser (Oxford Medical System Ltd, U.K., Analyser MA-20). An interface developed by us corrected time distortions caused by the recording technique and digitized the distance between consecutive R-waves (R-R interval) at 256 samples/sec. Then the resting temporal course of R-R intervals and parallel signals providing information about real time, arrhythmias, and artifacts was transferred to a laboratory computer (LSI-11/73) at 60 times real speed (technical details are available from the authors).

Artifact exclusion

A semiautomatic interactive procedure was developed for the detection and exclusion of artifacts. In a first step, definite singular artifacts were corrected and tested by defined jump criteria (Cheung, 1981). In a second step, for each *S* the minimum and maximum R-R interval and a relative jump criterion from beat to beat were preadjusted and the artifact exclusion was executed under statistical and visual control. Because there are no fixed limits for this jump criterion in the ECG, a multistage, conditional rating of the jumps was developed. Any regular jumps from beat to beat are still included, but artifacts are excluded reliably.

Evaluation of the ECG

The cardiac activity was analysed in five 1-min segments preceding and five 1-min segments following each event mark (cardiac perception). The mean heart period for each 1-min segment was calculated with a computer program. Hence each cardiac perception was described by 10 successive heart period values (ranging from minute -5 to +5). Minute segments with artifacts lasting more than 30 sec were excluded.

Statistical design

Group differences on questionnaires and rating scales were tested with the Mann-Whitney *U*-test and the χ^2 test. For all other dependent variables, parametric tests were used. The heart period data were treated as a repeated measure design with the variables *group* (patients, controls) and *time* (10 min repeated measure) and analysed with multivariate analysis of variance methods (MANOVA) following the recommendations of O'Brien and Kaiser (1985). Pillai's test statistic was used for the multivariate tests. Significant main effects and interactions were further evaluated using *t*-tests, paired *t*-tests and analyses of variance (ANOVA). If not otherwise specified, the mean \pm standard deviation is given.

RESULTS

Frequency of cardiac perceptions

Table 2 shows the number of cardiac perceptions for all patients with panic attacks and all healthy controls and, separately, for the subgroups of *S*s who reported at least one cardiac perception. The patients with panic attacks reported a total of 96 cardiac perceptions and the healthy controls a total of 60 cardiac perceptions. The percentage of *S*s without any cardiac perceptions during the recording period was about the same in both groups (30%). The patients with panic attacks reported somewhat more cardiac perceptions than the healthy controls, but this

Table 2. Frequency of cardiac perceptions

	Controls	Patients	Tests
Total population	20	28	
Frequency	60	96	<i>U</i> = 0.9
Mean (SD)	3.0 \pm 4.7	3.43 \pm 3.6	NS
Subpopulation	14	20	Chisq(2) = 0.0
with cardiac perceptions	(70%)	(71%)	NS
Frequency	60	96	<i>U</i> = 1.3
Mean (SD)	4.3 \pm 5.2	4.8 \pm 3.3	NS

Means \pm standard deviations are presented.

Chisq = χ^2 test.

U = Mann-Whitney *U*-test.

Table 3. Cardiac perceptions and amount of anxiety elicited

	Controls (%)	Patients	Tests
Anxiety			
None	60 (100)	27 (28.4%)	Chisq(3) = 76
Some	0 (0)	29 (30.5%)	$P < 0.001$
Moderate	0 (0)	23 (24.3%)	9 missing
Intense	0 (0)	16 (16.8%)	data

Chisq = χ^2 test.

difference is not significant. If we only look at the *Ss* with cardiac perceptions, the same result is found. The maximum number of cardiac perceptions reported by any one person was 18 in the healthy control group and 13 in the panic attack group.

Anxiety

Table 3 shows the amount of anxiety elicited by the cardiac perceptions. The healthy controls did not experience any anxiety related to cardiac perceptions. The *Ss* with panic attacks, however, reported at least some anxiety associated with 70% of their cardiac perceptions. The difference between the two groups is highly significant.

Heart period

Figure 2 shows the mean heart period for the 5 min preceding and following cardiac perceptions. A two-factorial analysis of variance with the variables *group* (patients, controls) and *time* (10 min repeated measure) revealed a significant group effect [$F(1,150) = 4.33$, $P = 0.04$, univariate], a highly significant time effect [$F(9,142) = 3.81$, $P < 0.001$, multivariate] and a significant group \times time interaction [$F(9,142) = 2.11$, $P = 0.03$, multivariate].

The group effect was further analysed with 10 simultaneous *t*-tests for each minute of registration and the level of significance was corrected to $P = 0.005$ following the Bonferroni method. The group difference in the first minute after cardiac perceptions clearly is significant at this level [min + 1: $t = 3.27$, $P = 0.001$], whereas the second minute difference is not quite significant [min + 2: $t = 2.80$, $P = 0.006$]. Three and 4 min after cardiac perceptions the group differences are only significant at the 5% level [min + 3: $t = 2.32$, $P = 0.022$; min + 4: $t = 2.28$, $P = 0.024$]. There are no group differences in the fifth minute after perceptions or in the 5 min preceding perceptions.

To further analyse the interaction effect, we evaluated the changes in heart period immediately preceding and following cardiac perceptions. In the patients with panic attacks, the heart period

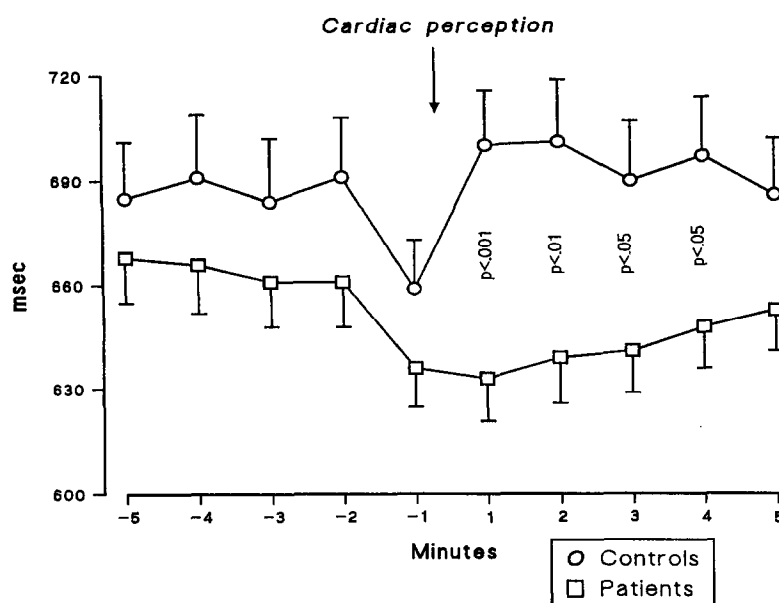


Fig. 2. Heart period and cardiac perceptions (means \pm SEMs are presented).

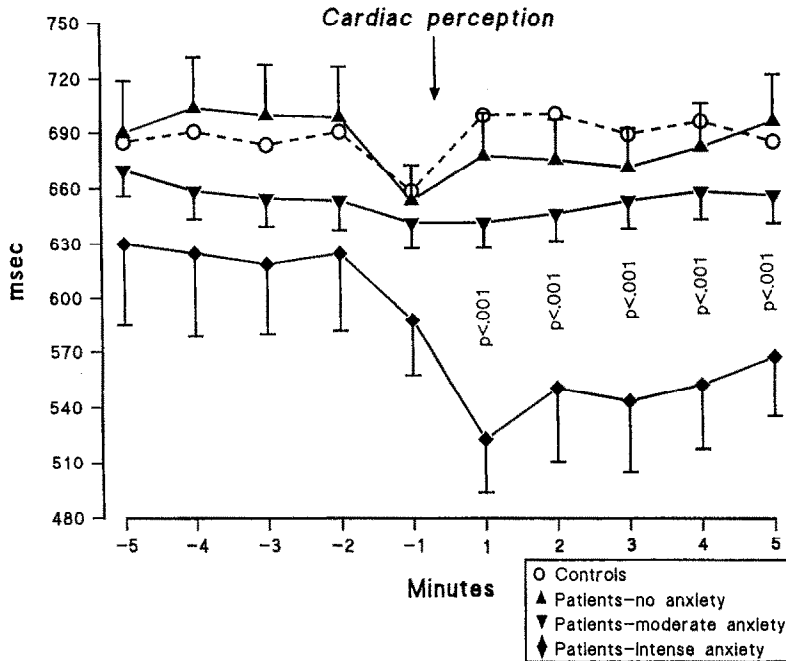


Fig. 3. Heart period and anxiety elicited by cardiac perceptions (means \pm SEMs are presented).

immediately preceding cardiac perceptions showed a shortage of 25.6 msec (± 69.9) and immediately following perceptions another shortage of 3.3 msec (± 59.8). The healthy controls showed a similar reduction in heart period of 27.6 msec (± 91.3) preceding reduction in heart period of 27.6 msec (± 91.3) preceding perceptions, but a substantial increase of 41.9 msec (± 81.8) immediately afterward. The two groups did not differ in the heart period changes preceding cardiac perceptions [*t*-test: $t = -0.02$, NS], but showed a significant difference immediately following perceptions [*t*-test: $t = 3.9$, $P < 0.001$].

The time effect within the groups was further analysed with paired *t*-tests. The healthy controls showed significant heart period changes preceding [$t = 2.3$, $P = 0.024$] and following [$t = -3.9$, $P < 0.001$] cardiac perceptions, whereas the heart period changes in the patients with panic attacks were significant only immediately before perceptions [$t = 3.5$, $P = 0.001$].

Interaction between anxiety and heart period

Figure 3 shows the heart period changes associated with cardiac perceptions in the patients with panic attacks differentiated by amount of anxiety (no anxiety, moderate and intense anxiety; the two categories some and moderate anxiety are combined). It also includes the heart period data for the healthy controls. A clear relationship is evident between the amount of anxiety and the heart period level: the more intense the anxiety elicited by cardiac perceptions, the faster the heartbeat after perceptions. Especially noteworthy are the heart period changes after perceptions with intense anxiety. Here the heart period dropped to about 500 msec, which corresponds to 120 bpm.

An analysis of variance with the variables group (no, moderate, intense anxiety) and time (10 min repeated measure) showed significant group differences [$F(2,91) = 5.37$, $P = 0.006$, univariate], significant time changes [$F(9,83) = 3.0$, $P = 0.004$, multivariate] and a significant group \times time interaction [$F(18,168) = 2.1$, $P = 0.006$, multivariate].

To further analyse the group difference, we performed 10 simultaneous analyses of variance (corrected level of significance $P = 0.005$). Preceding cardiac perceptions there are no significant group differences, but following cardiac perceptions the group differences are highly significant [min + 1: $F(2,92) = 10.9$, $P = 0.0001$; min + 2: $F(2,92) = 6.5$, $P = 0.0023$; min + 3: $F(2,92) = 7.6$, $P = 0.001$; min + 4: $F(2,92) = 7.7$, $P = 0.0008$; min + 5: $F(2,92) = 7.4$, $P = 0.001$]. As the Scheffé *a posteriori* tests reveal, these differences can be attributed to the significantly shorter heart period (elevated heart rate) following perceptions with intense anxiety. Perceptions without anxiety or with only moderate anxiety show no difference in the heart period.

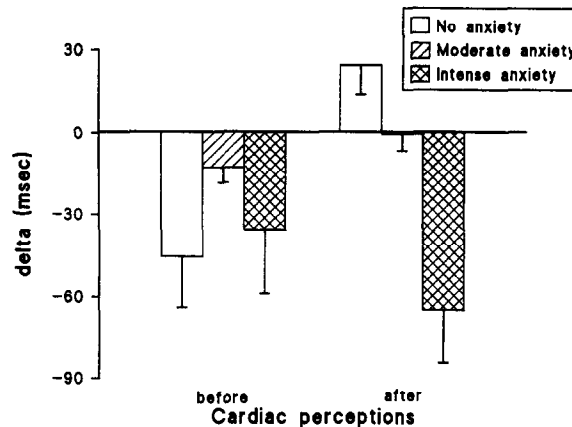


Fig. 4. Heart period changes and amount of anxiety (means \pm SEMs are presented).

Of special importance for this study is the relation between the anxiety elicited by the cardiac perceptions and the perception-related heart period changes (Fig. 4). An analysis of variance revealed that the heart period changes preceding perceptions are not related to the amount of anxiety experienced [$F(2,92) = 2.1$, NS]. But there is significant relation between the anxiety elicited by perceptions and the heart period changes following that perceptions [$F(2,92) = 4.8$, $P = 0.01$]. The more intense the anxiety, the more pronounced is the shortening of the heart period. This association between anxiety and heart period change is linear [linear: $F(1,92) = 7.0$, $P = 0.009$; quadratic: $F(1,92) = 2.5$, NS].

DISCUSSION

Counter to hypothesis 1, the *Ss* with panic attacks did not report any more cardiac perceptions than the healthy controls during the 24-hr registration period. However, in support of hypothesis 2, only the *Ss* with panic attacks experienced any anxiety related to cardiac perceptions. There are no group differences in heart period preceding cardiac perceptions. Furthermore, the two groups show a similar and significant shortening of the heart period immediately before cardiac perceptions. However, the groups differ significantly in the heart period after cardiac perceptions: whereas in the healthy controls the heart period becomes longer again, in the patients with panic attacks it becomes even shorter. The effect is that the patient group has a faster heart rate following cardiac perceptions than the control group, an effect postulated in hypothesis 3. Finally, the results support hypothesis 4. As predicted, there was a significant positive relation between the anxiety elicited by cardiac perceptions and the heart period changes after such perceptions. Cardiac perceptions without anxiety were associated with a heart rate deceleration in the patients with panic attacks as well as in the healthy controls. After cardiac perceptions with moderate anxiety, there was no heart rate deceleration, and after perceptions with intense anxiety the heart rate accelerated even further. One can therefore conclude that the more intense the anxiety the faster the heart rate.

About 30% of both the patients with panic attacks and the healthy controls did not report any cardiac perceptions. To evaluate whether these were special subgroups of *Ss*, we compared *Ss* with and without cardiac perceptions within the groups with regard to the personal and questionnaire data. No significant differences were found either in the patients with panic attacks or the healthy controls. We think that the relatively large percentage of *Ss* without cardiac perceptions was a result of the relatively short registration period of 24 hr. Other authors who have tried to register panic attacks with comparable methods have used registration periods of between 2 and 6 days (Freedman, Ianni, Ettedgui & Puthezath, 1985; Gaffney, Fenton, Lane & Lake, 1988; Margraf *et al.*, 1987). Presumably a longer registration period would have enhanced the likelihood of cardiac perceptions and we would have been able to clarify the relation between anxiety and cardiac perceptions even in our subgroup of patients without cardiac perceptions.

Taken together, our results support the psychophysiological model of panic attacks outlined earlier, namely that cardiac perceptions are triggered mainly by heart rate accelerations, and

perception of these accelerations elicits anxiety in patients with panic attacks. The anxiety then leads directly to a further heart rate acceleration, and the positive feedback loop is closed. However, a negative, homeostatic feedback mechanism seems to work against this positive feedback loop (presumably the baroreceptor reflex or an emergency inhibition mechanism, postulated by Lader & Mathews, 1977). In patients with panic attacks, the heart rate slows down to the pre-perception level within 5 min. In healthy controls, who do not experience any anxiety related to cardiac perceptions, only the negative, homeostatic feedback loop is active. Therefore, the heart rate acceleration preceding cardiac perceptions is immediately compensated for, and the heart rate in the minute after the perception is actually slower than in the minute before. From then on, the heart rate slowly approaches the pre-perception level. This process resembles a biphasic process of achieving equilibrium, which often occurs after heart rate fluctuations. The interaction of the two feedback loops—the psychophysiological vicious circle and the physiological, homeostatic regulatory mechanism—can explain the observed heart rate changes after cardiac perceptions in both patients with panic attacks and healthy controls.

A central assumption of the postulated psychophysiological model of panic attacks is that the physiological changes occurring after cardiac perceptions are directly related to the anxiety these perceptions elicit. Our results unequivocally support this assumption. The heart rate changes after the cardiac perceptions are the physiological component of the anxiety reaction elicited by the perception. The positive feedback loop is closed only when there is anxiety, and according to this model, anxiety attacks are elicited by cardiac perceptions and are the result of an escalation of the positive feedback loop caused by too intense anxiety. This does in fact seem to be the case for the cardiac perceptions with intense anxiety we recorded. Here the heart rate increases to a maximum of 120 bpm (510 msec) within 2 min and then decreases within the next 4 min. Panic attacks registered so far in the laboratory and in the field show very similar heart rate changes, also reaching a maximum of about 120 bpm within 1 or 2 min and then decreasing to the pre-panic level within 5 min (Freedman *et al.*, 1985; Lader & Mathews, 1970; Margraf *et al.*, 1987).

Our findings are not compatible with hypothesis 1: the *Ss* with panic attacks did not report more cardiac perceptions than the healthy controls. We think that our experimental conditions led to the similarity in the frequency of perceptions in the two groups. In particular, the instruction to monitor the heartbeat and to note any cardiac perceptions led the healthy controls to focus their attention on their cardiac activity more than they usually would. This presumably increased the likelihood of cardiac perceptions in the healthy controls. On the other hand, patients with panic attacks are likely to try to avoid interoceptive, anxiety-eliciting cardiac perceptions. Both factors could have led to the observed similarity in the number of cardiac perceptions. Furthermore, the groups did not differ in the heart rate changes preceding cardiac perceptions, i.e. in the physiological triggers of the perceptions. If *Ss* with panic attacks are better at perceiving cardiac activity, then they should be able to detect smaller heart rate changes than healthy controls. But this was not the case, which is consistent with the results of Ehlers *et al.* (1988b). In neither study was there any evidence that patients with panic attacks are more accurate heartbeat perceivers than healthy controls. These results are not in conflict with a psychophysiological model of panic attacks. Improved cardiac awareness is not an essential part of the vicious circle model postulated. Better cardiac perception would only enhance the likelihood of the vicious circle being triggered (Ehlers *et al.*, 1988b; Sachse, 1984).

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