Psychological Medicine, 1991, **21**, 413–421
Printed in Great Britain

Heartbeat and arrhythmia perception in diabetic autonomic neuropathy

PAUL PAULI,[1] LYDIA HARTL, CHRISTIAN MARQUARDT, HENRICA STALMANN AND FRIEDRICH STRIAN

From the Clinical Department, Max Planck Institute for Psychiatry, Munich, Germany

SYNOPSIS A comparative study of diabetics with autonomic neuropathy ($N = 13$) as against non-neuropathic diabetics ($N = 16$) and healthy control persons ($N = 20$) was carried out with respect to heart rate both at rest and under stress, frequency of cardiac arrhythmias in a 24-h ECG and accuracy of heartbeat and arrhythmia perception. In the subjects with diabetic autonomic neuropathy, the spontaneous variability and stress-induced reactivity of the heart rate as well as the number of tachycardic episodes were reduced, whereas the frequency of ventricular extrasystoles was somewhat increased. Impaired heartbeat perception and a complete loss of perception of arrhythmias as a consequence of neuropathic deafferentation could be demonstrated. Cardiac perception disorders also play a vital role in other clinical problems, e.g. silent myocardial infarction and lack of awareness of hypoglycaemia in diabetes mellitus.

INTRODUCTION

Unpleasant bodily sensations play an important role in internal, psychosomatic and most psychiatric disorders. Distorted visceral perception in patients with panic disorders may trigger anxiety attacks (Ehlers *et al.* 1988*a*). Bodily complaints are an inevitable syndrome in depressive patients (DSM-III, APA, 1980; Kanfer & Hagerman, 1981). The diagnosis of internal diseases is likewise based on the subjective perception of physical symptoms.

For cardiac interoception, it has been demonstrated in laboratory studies that heartbeat perception in healthy subjects is influenced by gender (Whitehead *et al.* 1977; Jones & Hollandsworth, 1981), weight or body fat (Montgomery & Jones, 1984; Rouse *et al.* 1988), body position (Jones *et al.* 1987), physical activity (Jones & Hollandsworth, 1981), emotional arousal (Katkin *et al.* 1982; Katkin, 1985) and emotionality (Schandry, 1981; Montgomery & Jones, 1984). However, it has also been found that less than 50% of the healthy population are reasonably good heartbeat perceivers (Schandry, 1981; Davis *et al.* 1986; Jones *et al.* 1987). Considering that under most circumstances awareness of visceral sensations is of little relevance for healthy subjects, this appears plausible (cf. Jones *et al.* 1985). Nevertheless, most subjects can learn to discriminate their heartbeats (Brener & Jones, 1974; Davis *et al.* 1986; Jones *et al.* 1987).

Several clinical studies have endeavoured to determine whether patients with heart-related complaints or anxieties are more prone to perceiving their cardiac activity correctly. In laboratory studies it was found that patients with cardiac phobia (Stalmann *et al.* 1988), panic disorder (Ehlers *et al.* 1988*b*), hypochondriasis and anxiety neurosis (Tyrer *et al.* 1980) are somewhat more aware of their cardiac activity than healthy controls, whereas patients with mitral valve prolapse (Stalmann *et al.* 1988), specific phobia (Tyrer *et al.* 1980) or myocardial infarction (Jones *et al.* 1985) are not. But even the patients with increased cardiac awareness are not perfect in the perception of heartbeats, and only Ehlers *et al.* (1988*b*) reported a trend towards a higher percentage of panic patients classified as good cardiac perceivers.

Another approach to evaluating cardiac perceptivity has evolved from cardiology. The question of interest is whether there is a

[1] Address for correspondence: Dr Paul Pauli, Max-Planck-Institut für Psychiatrie, Neurologische Poliklinik, Kraepelinstr. 10, 8000 München 40, Germany.

relationship between cardiac perceptions and occurrences of arrhythmias. Studies have been carried out on patients with cardiac symptoms by means of the 24-h (Holter) ECG and time-synchronous recordings of cardiac sensations (event marker on a second Holter channel) (Kunz *et al.* 1977; Krasnow & Bloomfield, 1978; Levine *et al.* 1978; Völker *et al.* 1986). Whereas more than 50% of such patients have arrhythmia, mainly multiple ventricular extrasystoles and tachycardias (Völker *et al.* 1986), they only perceive about 10% of the arrhythmias confirmed by ECG (Kunz *et al.* 1977; Levine *et al.* 1978). Since these studies did not include control groups, there is still no clear conception as to how this weak relationship between occurrence and perception of arrhythmias should be interpreted. However, this approach seems very useful because naturally occurring cardiac perceptions and their physiological triggers can be evaluated.

The present study examines whether the innervation disorder in diabetic autonomic neuropathy impairs the perception of normal and arrhythmic cardiac activity (cf. Lautenbacher *et al.* 1987). The autonomic deafferentation in diabetic neuropathy serves as a paradigm for the visceral afferent blockade. At the same time, the cardiovascular innervation disorder appears to be an early symptom of diabetic autonomic neuropathy (Lehmann *et al.* 1985). Cardiac signs of autonomic neuropathy are reductions in spontaneous variability, in nocturnal decrease and in stress adaptation of the heart rate (Ewing *et al.* 1984; Masaoka *et al.* 1985), as well as an increased basal heart rate (Clarke & Ewing, 1982) and an increased number of arrhythmias (Runge & Kühnau, 1983). The impaired cardiac perception in diabetic autonomic neuropathy is one of the possible causes of asymptomatic angina pectoris and silent myocardial infarction (Faerman *et al.* 1977; Clarke *et al.* 1979; Runge & Kühnau, 1983; Stalmann *et al.* 1987). Furthermore, the reduction in the number and strength of visceral signals seems to lead to a flattened emotional state in patients with this disorder (Pauli *et al.* 1989). On the other hand, the correct perception of cardiac symptoms (tachycardia) can contribute to early detection of hypoglycaemia (Pennebaker *et al.* 1981; Klosiewski, 1984).

Cardiac perception in diabetic autonomic neuropathy was assessed with a heartbeat (mental) tracking task and an arrhythmia perception task. Whereas the latter is a naturalistic and established procedure for studying the perceptivity of arrhythmias, the validity and reliability of heartbeat (motor) tracking techniques have been questioned (Pennebaker & Hoover, 1984; Flynn & Clemens, 1988). We have included our findings with the heartbeat tracking task for several reasons. First, the validity and reliability of *motor tracking* techniques are presumed to be low because the required motor responses interfere with the perceptual process (Pennebaker & Hoover, 1984). However, this is not the case for *mental tracking*. Secondly, the relationship among findings on all commonly used heartbeat perception tasks is weak (Jones *et al.* 1984; Pennebaker & Hoover, 1984; Davis *et al.* 1986), and so far there is no generally accepted method. Thirdly and most importantly, the results of the mental tracking task employed are consistent with the findings on the arrhythmia perception task and, therefore, are very interesting in the context of the study as a whole.

The following three hypotheses were tested. (1) In patients with diabetic autonomic neuropathy, the spontaneous variability and the orthostatic reactivity of the heart rate are diminished in comparison with patients with non-neuropathic diabetes mellitus and healthy subjects; on the other hand, in patients with diabetic autonomic neuropathy there is an increase in heart rate at rest and in the incidence of arrhythmias. (2) The perception of heartbeat is less accurate in patients with diabetic autonomic neuropathy than in the control groups. Within each group, heartbeat perception is best after physically or psychologically induced arousal and worst in a standing or sitting position. (3) The perception of tachycardias and ventricular extrasystoles is heavily impaired in patients with diabetic autonomic neuropathy.

METHOD

Subjects

Thirteen diabetics with autonomic neuropathy (DAN), 16 diabetics without neuropathy (DWN) and 20 healthy controls (HC) between 20 and 60 years of age were examined (Table 1). The control group contained more men than

Table 1. *Subjects*

		Healthy controls	Diabetics without AN	Diabetics with AN	Test of significance
Sex	Male	13	6	5	Chi-square
	Female	7	10	8	*X* = 3·5 NS
BMI	M	22·6	21·9	21·2	*H* test
(kg/m²)	S.D.	2·1	2·2	3·5	*X* = 4·1 NS
Age (yr)	M	31·8	28·4	37·6	*H* test
	S.D.	7·9	7·1	10·5	*X* = 3·5 NS
Height	M	172·4	170·4	167·3	*H* test
(cm)	S.D.	8·8	8·4	7·8	*X* = 2·0 NS
Weight	M	67·5	63·1	59·5	*H* test
(kg)	S.D.	11·2	8·7	12·4	*X* = 4·4 NS
Duration of	M		4313	6089	*U* test
illness (day)	S.D.		738	3019	*Z* = 1·5 NS

AN = autonomic neuropathy; NS = not significant; M = mean, S.D. = standard deviation; BMI = body mass index.

either patient group, but the differences are not statistically significant. Furthermore, as Rouse *et al.* (1988) have shown, the frequently reported superior cardiac perception in men is caused by differences in body fat. All three groups examined had very similar body mass indexes (BMI = kg/m²) (cf. Montgomery & Jones, 1984) and therefore gender effects on cardiac perception are not likely. The differences between the three groups with respect to age, height, weight and duration of illness are not significant.

All of the patients had insulin-dependent Type I diabetes with age of manifestation under 40 and HbA1 or HbA1c values of at least 8 and 6%, respectively. The diabetics with autonomic neuropathy had to have an additional clearly pathological finding in at least one autonomic functional system. Tests were performed for orthostatic hypotonia, bladder atonia, gastroparesis and erectile impotence. Cardiac disease was excluded in all participants by clinical investigations such as echo- and electrocardiography. All medications affecting the heart and CNS (e.g. beta-blockers and sedatives) were withdrawn for the duration of the study, whereas insulin therapy for the diabetics was continued.

Due to technical problems, data errors were detected for two control subjects and two non-neuropathic diabetics with respect to heartbeat perception and for one non-neuropathic diabetic regarding perception of arrhythmias.

Apparatus

The electrocardiogram was recorded by means of two pre-cordial leads on C-120 tapes in a portable ECG-recorder (FM-Recorder MR-20, Oxford Medical System Ltd/UK) with a maximum run of 24 h. The channel with fewer artefacts was selected for evaluation. The event track of the recorder was used by the experimenter to mark the time intervals for the heartbeat perception task and by the subjects to mark arrhythmic sensations. The ECG-analysis instrument (Analyser MA-20, Oxford Medical System Ltd/UK) yielded the heart rate for the marked intervals and enabled the semi-automatic detection of arrhythmia under visual control.

Procedure

The subjects were informed about the experimental procedure on arrival in the examination room. Then the ECG electrodes were attached. The subjects spent the next 20 min filling out several questionnaires and afterwards proceeded with the heartbeat perception test (described later). Instructions for long-term ambulatory ECG recording followed, including instructions to record current activities every hour. Except for strenuous physical work (e.g. sports), the subjects were allowed to carry out their normal daily activities. The ECG apparatus was removed 24 h later.

Heartbeat perception

A modification of the mental tracking test described by Schandry (1981) was used. The subjects were requested to concentrate on their heart at defined time intervals (onset and offset indicated by a tone) and to count the heartbeats perceived. They were instructed not to take their pulse or to try any other physical manipulation

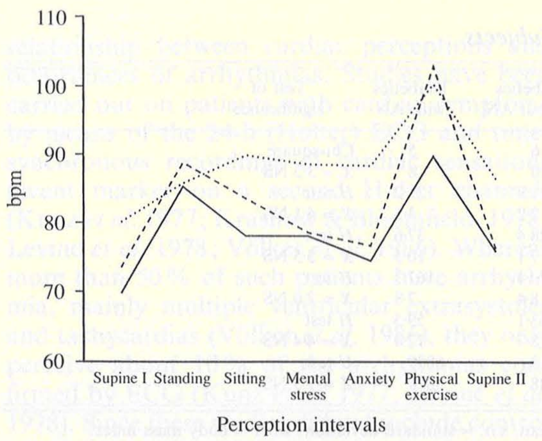

FIG. 1. Mean heart rate (bpm) under the seven conditions of the heartbeat perception task (for significance levels see text). ——, Healthy controls; ---, diabetics without neuropathy; ····, diabetics with autonomic neuropathy.

that might facilitate the detection of heartbeats. The task was performed seven times to measure heartbeat perception and heart rate under various physical and psychological stress manipulations. The order of manipulation was as follows.

(1) Supine I – after 10 min of relaxation in a supine position a perception interval of 30 s.

(2) Standing – immediately after getting up a perception interval of 25 s.

(3) Sitting – after 5 min of relaxed sitting a perception interval of 25 s.

(4) Mental stress – immediately after a 2-min subtraction task (starting with 3000, repeatedly subtracting 13) a perception interval of 30 s.

(5) Anxiety – after imagining an anxiety-provoking situation for 2 min a perception interval of 30 s.

(6) Physical exercise – immediately after deep knee bends for one min a perception interval of 15 s.

(7) Supine II – after 10 min of relaxation in a supine position a perception interval of 20 s.

At the end of each interval the subjects were requested to report how many heartbeats they had counted. Note that Schandry (1981) asked his subjects to report the number of heartbeats counted or estimated. We changed this instruction because we wanted the subjects to register only the number of heartbeats actually perceived. The number of heartbeats perceived (SB = subjective beats) was then compared with the actual number of heartbeats (OB = objective beats) for the same interval. A perception score (PS) was calculated as follows (cf. Bestler *et al.* 1990):

$$PS = 1-(|SB-OB|)/OB.$$

Additionally, the responses were classified into three perception categories:

exact perception: $|SB-OB| \leqslant 2$;
poor perception: $|SB-OB| > 2$;
no perception: $SB = 0$.

The perception score quantifies the accuracy of the perception of heartbeats on a continuous scale from 0 to 1, with a high score reflecting good perception. The perception categories 'exact perception' and 'no perception' represent the extremes. 'Exact perception' means that the subject was able to perceive all heartbeats in the perception interval. The deviation of ± 2 beats is tolerated because at the time of the tone indicating onset or offset one beat can easily be missed or added (cf. Schandry, 1981). In contrast, 'no perception' reflects a total inability to perceive any heartbeats during the perception interval. Some subjects fell into this category because we asked the subjects to report the number of heartbeats counted, not estimated. Although there is no direct means of checking whether a subject's response is in fact based on the perception of discrete heartbeats, at least for the categories 'exact perception' and 'no perception' this seems to be the case. It is very unlikely that someone will achieve 'exact perception' without perceiving the actual heartbeats. Furthermore, there is no reason for a subject to report no heartbeats if he or she did in fact perceive heartbeats.

Arrhythmia perception

The subjects were instructed to mark all perceptions of irregular cardiac activity on the marker channel of the recorder. The 24-h ECGs were then examined for ventricular extrasystoles (VES), tachycardias (TACHY) and other cardiac irregularities. The arrhythmias were classified according to type and severity (Lown classification). Tachycardias were defined as an episode of increased heart rate that exceeded the baseline rate by 20% within 1 min. If a subject marked a cardiac perception up to 40 s after a ventricular extrasystole and 60 s after the commencement of a tachycardia this was considered an exact perception of an arrhythmia. The hit rate (P-HIT) was defined as the ratio of exactly perceived arrhythmias f(eA) to the total number of arrhythmias f(tA). The hit rate was calculated separately for tachycardias (P-HIT-TACHY) and ventricular extrasystoles (P-HIT-VES):

$$\text{hit rate: (P-HIT)} = f(eA)/f(tA) \times 100\ (\%).$$

The error or 'false alarm' rate (P−FA) represents the proportion of events marked without preceding arrhythmia f(fA) in relation to the total number of marks made f(tM):

$$\text{false alarm rate: (P-FA)} = f(fA)/f(tM) \times 100\ (\%).$$

Hit and false alarm rates were calculated for each subject.

Statistical evaluation

Group differences were tested with a non-parametric test of significance (5%, two-tailed). If the Kruskal–Wallis rank variance analysis (*H* test) yielded significant group differences, then simultaneous *post-hoc* comparisons were made with the Nemenyi test. Changes in the heartbeat perception scores within the groups were tested with the Friedman rank variance analysis. The distribution of the two response categories 'exact perception' and 'no perception' was evaluated descriptively. Due to low cell populations, statistical analysis was not possible.

RESULTS

Heart rate

The diabetics with autonomic neuropathy tended to have the highest heart rate in the perception intervals (Fig. 1). Under physical stress (standing, physical exercise), however, these differences largely disappeared; this was because the increase in heart rate was more marked in the two control groups than in the group of diabetics with autonomic neuropathy. The difference in heart rate between the groups is significant only for the condition 'anxiety' (*H* test: $P = 0{\cdot}03$; *post-hoc* comparisons: HC/DWN NS; HC/DAN $P = 0{\cdot}04$; DAN/DWN NS).

Spontaneous heart rate variability

Spontaneous heart rate fluctuation (R–R interval variation) was evaluated with a method proposed by Airaksinen *et al.* (1986). The evaluation is made at rest, whereby the difference between the shortest and longest interbeat interval within a period of 12 seconds is determined. The diabetics with autonomic neuropathy had a significantly lower spontaneous variation in heart rate than the non-neuropathic diabetics or the healthy controls (mean ±standard deviation; HC: $0{\cdot}15 \pm 0{\cdot}06$; DWN: $0{\cdot}12 \pm 0{\cdot}05$; DAN: $0{\cdot}06 \pm 0{\cdot}03$; *H* test: $P < 0{\cdot}01$; *post-hoc* comparisons: HC/DWN NS; HC/DAN $P < 0{\cdot}01$; DAN/DWN $P = 0{\cdot}01$).

Orthostatic reaction

Cardiac orthostatic reaction was defined as the percentage change in heart rate between the consecutive experimental conditions, 'supine I' and 'standing'. The mean increase in heart rate was 25% (±25) in the healthy controls, 22% (±17) in the non-neuropathic diabetics and 10% (±33) in the diabetics with autonomic neuropathy. The group differences in the orthostaticly induced heart rate changes are significant (*H* test: $P = 0{\cdot}03$; *post-hoc* comparisons: HC/DWN NS; HC/DAN NS; DAN/DWN $P = 0{\cdot}04$).

Heartbeat perception

Fig. 2 shows the perception scores for the three groups in the seven experimental situations. The diabetics with autonomic neuropathy had the worst overall perception scores, and this did not change within the test situations (Friedman test: NS). The diabetics without neuropathy, on the other hand, started with relatively poor scores, but improved markedly with the number of trials (Friedman test: $P < 0{\cdot}01$). They performed best in the anxiety condition and worst in the standing condition. The healthy control group achieved the best perception scores in all test conditions, with the poorest result in the sitting condition and the best in the anxiety condition (Friedman test: $P = 0{\cdot}05$). The differences between the groups are close to significance in the test conditions 'standing' (*H* test: $P = 0{\cdot}10$), 'mental stress' ($P = 0{\cdot}12$), 'physical exercise' ($P = 0{\cdot}12$) and 'supine II' ($P = 0{\cdot}08$). However, if the perception score is averaged across the two supine situations to stabilize the score, the group differences are significant (HC: $0{\cdot}70 \pm 0{\cdot}19$; DWN: $0{\cdot}57 \pm 0{\cdot}23$; DAN: $0{\cdot}50 \pm 0{\cdot}33$; *H* test: $P = 0{\cdot}04$; *post hoc* comparisons: HC/DWN NS; HC/DAN $P = 0{\cdot}08$; DAN/DWN NS).

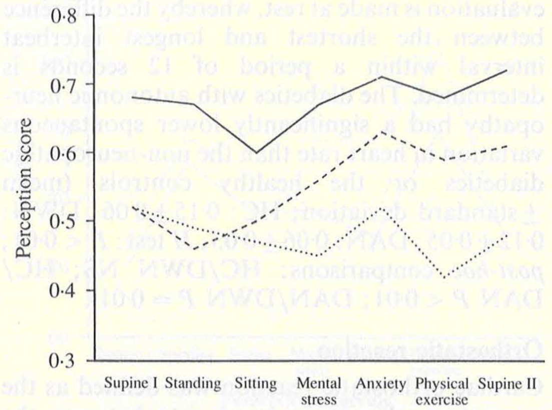

FIG. 2. Heartbeat perception score (PS) under the seven conditions of the heartbeat perception task (for significance levels see text). ——, Healthy controls; ---, diabetics without neuropathy; · · · ·, diabetics with autonomic neuropathy.

Similar results are revealed by the distribution of the two perception categories 'exact perception' and 'no perception' (Table 2). The healthy control group performed best, the diabetics with autonomic neuropathy worst. The relatively good performance of the healthy controls is reflected in the frequency of exact perceptions. Compared with both diabetic groups, more than twice as many of them were able to achieve an exact perception at least once (50 % compared with 21 and 23 %), and overall they had exact perceptions about twice as often as either diabetic group (14 % compared with 6 and 9 %). On the other hand, the impaired heartbeat perception ability of the diabetics with autonomic neuropathy is evident in the frequency of the 'no perception' responses. In 27 % of the perception intervals they were unable to perceive any heartbeats, whereas this happened for the healthy controls and the diabetics without neuropathy in less than 10 % of the intervals. Moreover, 31 % of the diabetics with autonomic neuropathy were unable to perceive any heartbeats at all at least once, compared with only 17 % of the healthy controls and 14 % of the diabetics without neuropathy.

Examination of the results in the different heartbeat perception conditions reveals that the healthy controls performed best (see frequency of exact perceptions) in the situations 'anxiety', 'physical exercise' and 'supine II', and worst in the situations 'standing' and 'sitting'. The

Table 2. *Heartbeat perception responses in the two categories 'exact perception' and 'no perception'*

	Exact perception			No perception		
	HC	DWN	DAN	HC	DWN	DAN
Supine I	2	1	1	1	1	3
	11 %	7 %	8 %	6 %	7 %	23 %
Standing	0	0	2	0	2	3
	0 %	0 %	15 %	0 %	14 %	23 %
Sitting	0	1	1	2	2	3
	0 %	7 %	8 %	11 %	14 %	23 %
Mental stress	1	1	2	1	0	4
	6 %	7 %	15 %	6 %	0 %	30 %
Anxiety	4	1	0	1	0	3
	22 %	7 %	0 %	6 %	0 %	23 %
Physical exercise	5	1	0	2	0	4
	28 %	7 %	0 %	11 %	0 %	30 %
Supine II	5	1	2	1	0	4
	28 %	7 %	15 %	6 %	0 %	30 %
Total responses	17	6	8	8	5	24
	14 %	6 %	9 %	6 %	5 %	27 %
Total subjects	9	3	3	3	2	4
	50 %	21 %	23 %	17 %	14 %	31 %

HC = healthy controls; DWN = diabetics without neuropathy; DAN = diabetics with neuropathy.

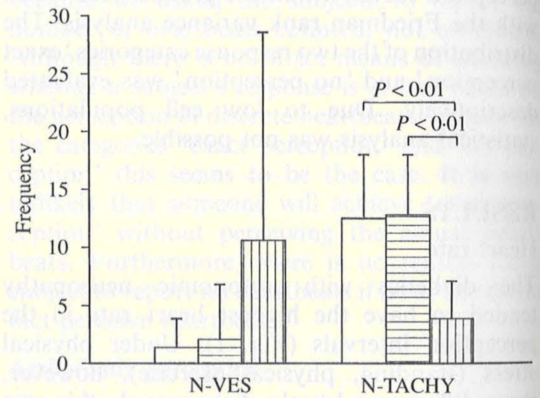

FIG. 3. Frequency of ventricular extrasystoles (N-VES) and tachycardias (N-TACHY) (means and standard deviations). □, Healthy controls; ▤, diabetics without neuropathy; ▥, diabetics with autonomic neuropathy.

diabetics without neuropathy did not improve their frequency of exact perceptions in the situations where the healthy controls did. However, all of them were able to perceive at least some heartbeats in these situations, leading to a reduction in 'no perception' responses and an improvement in the perception score (see Fig. 2). The diabetics with autonomic neuropathy, on the other hand, were unable to improve their

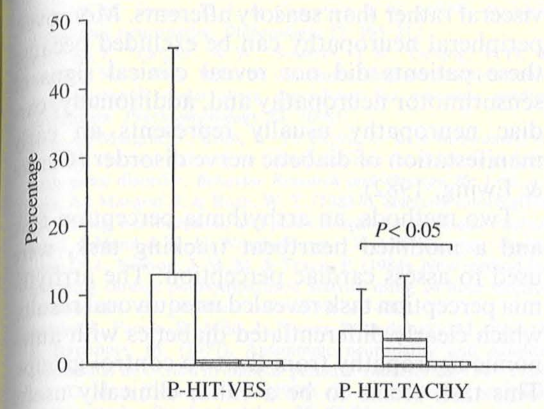

FIG. 4. Percentage of correctly perceived ventricular extrasystoles (P-HIT-VES) and tachycardias (P-HIT-TACHY) in the ambulatory 24-h ECG (means and standard deviations). □, Healthy controls; ▤, diabetics without neuropathy; ▥, diabetics with autonomic neuropathy.

cardiac perception in the 'anxiety' and 'physical exercise' conditions. The frequency of intervals with imperceptibility of heartbeats did not change and, additionally, there were no intervals with exact perceptions.

Frequency of arrhythmias

On average, the diabetics with autonomic neuropathy had more ventricular extrasystoles and fewer tachycardias than the subjects in the two control groups (Fig. 3). While these differences are significant for tachycardias (*H* test: $P < 0{\cdot}01$; *post-hoc* comparisons: HC/DWN NS; HC/DAN $P < 0{\cdot}01$; DAN/DWN $P < 0{\cdot}01$), they are not significant for ventricular extrasystoles.

Number of cardiac events marked

Perceptions of cardiac activity were rare in all of the test groups. The mean number of events marked was 2·3 (±4·2) in the control group, 1·6 (±3·0) in the non-neuropathic diabetics and 0·15 (±0·4) in the diabetics with autonomic neuropathy. The difference between the healthy controls and the diabetics with autonomic neuropathy is significant (*H* test: $P = 0{\cdot}03$; *post-hoc* comparisons: HC/DWN NS; HC/DAN $P = 0{\cdot}04$; DAN/DWN NS).

Perception of arrhythmias

In the control group the hit rate was 13 % (±33) for ventricular extrasystoles and 5 % (±6) for tachycardias (Fig. 4). In the non-neuropathic diabetics, the hit rate for tachycardias was quite similar (4 % ± 9), whereas this group was unable to perceive ventricular extrasystoles correctly. The diabetics with autonomic neuropathy, on the other hand, were unable to perceive either tachycardias or ventricular extrasystoles. The group differences are significant for the tachycardia hit rate (*H* test: $P = 0{\cdot}04$; *post-hoc* comparisons: HC/DWN NS; HC/DAN $P = 0{\cdot}04$; DAN/DWN NS), but not for the perception of ventricular extrasystoles.

The mean false alarm rate (P − FA) was 59 % (±40) in the control group, 57 % (±34) in the non-neuropathic diabetics and 100 % (±0) in the diabetics with neuropathy. However, the differences between the groups are not significant.

DISCUSSION

In diabetics with autonomic neuropathy, the efferent and afferent innervation of the heart is disturbed. This leads to an alteration in cardiac activity and to disturbed perception of heartbeats and arrhythmias.

Consistent with hypothesis 1, the diabetics with autonomic neuropathy had a reduced spontaneous variability of the inter-beat intervals, a lower orthostatic increase in heart rate, less frequent tachycardias and more frequent ventricular extrasystoles than the non-neuropathic diabetics or the healthy controls. An increased basal heart rate could be confirmed only as a tendency. Our findings were also consistent with the suppositions formulated in hypothesis 2. The healthy subjects achieved the best heartbeat perception scores in all test situations and the highest overall number of exact perceptions. As expected, they performed best in situations with psychologically or physically induced arousal and worst in a standing or sitting position. The diabetics without neuropathy started out with relatively poor heartbeat perception, comparable to that of the diabetics with neuropathy; however, they were able to improve their performance markedly in the situations with arousal induction. This differentiated them clearly from the diabetics with autonomic neuropathy. The latter were unable to improve their heartbeat perception. Most characteristic for the diabetics with neuropathy was that many of them had a total imperceptibility of heartbeats. Overall their perception of heartbeats was less accurate than that of the two control groups. Furthermore, consistent with hypothesis 3, the diabetics with autonomic neuropathy marked fewer cardiac events in the ambulatory 24-h ECG, and their cardiac sensations showed absolutely no connection with actual cardiac arrhythmias such as ventricular extrasystoles and tachycardias. This total inability to perceive arrhythmias clearly differentiates the neuropathic diabetics from the healthy controls and in general from the non-neuropathic diabetics.

Our findings confirm that autonomic deafferentation in diabetes mellitus leads to an impairment in the transmission of cardiac signals. In intact cardiac perceptivity, cardiac signals are decoded and processed in the 'noise' of the internal and external afferences, similar to external signals (Pennebaker, 1982). Autonomic deafferentation induces a reduction in the cardiac signal rate, impairing the cardiac signal-to-noise ratio. Under ambulatory conditions (24-h ECG, arrhythmia perception task), i.e. normal noise level, the cardiac signal can no longer be perceived correctly, resulting in a total loss of arrhythmic perception. The conditions of the laboratory, on the other hand (heartbeat perception task, protection from external stimuli, concentration on the heart), reduce the general noise level and hence the signal-to-noise ratio is artificially improved. Under such conditions, the effects of the autonomic neuropathy are somewhat less distinct, but still observable. Within the laboratory, the healthy controls and the diabetics without neuropathy achieved their best results in situations with arousal induction. Presumably, in these conditions the cardiac signal is strengthened by beta-adrenergic influences on the myocardium (Katkin, 1985). In diabetics with autonomic neuropathy the afferent transmission of the cardiac signal is impaired, and therefore these patients do not show a similar effect.

According to Brener (1974), perception and control of visceral activities could be transmitted via a central feedback pathway, an interoceptive afferent pathway and/or an exteroceptive afferent pathway (cf. Jones *et al.* 1987). The confirmed impairment of cardiac perception due to neuropathic denervation of the heart suggests that cardiac perceptions are transmitted via visceral rather than sensory afferents. Moreover, peripheral neuropathy can be excluded because these patients did not reveal clinical signs of sensorimotor neuropathy and, additionally, cardiac neuropathy usually represents an early manifestation of diabetic nerve disorder (Clarke & Ewing, 1982).

Two methods, an arrhythmia perception task and a modified heartbeat tracking task, were used to assess cardiac perception. The arrhythmia perception task revealed unequivocal results, which clearly differentiated diabetics with autonomic neuropathy from the two control groups. This task seems to be a valid, clinically useful and naturalistic tool for evaluation of cardiac perception. The additionally conducted modified tracking task basically confirmed the above findings. Therefore, in the context of the whole study, it is justifiable to report these results in spite of the recently questioned validity of the tracking method (Flynn & Clemens, 1988).

The cardiac perception deficiency as demonstrated in this study is clinically significant not only for perception of heartbeats and arrhythmias, but also for other cardiac sensations. The loss of cardiac pain may lead to silent myocardial infarction, and the loss of hypoglycaemic awareness in diabetes mellitus even to hypoglycaemic coma. Additionally, the impaired visceral perception has indirect effects on psychological and affective variables (Pauli *et al.* 1989).

This work was supported by grant no. Str 273/2-3 from the Deutsche Forschungsgemeinschaft.

REFERENCES

Airaksinen, K. E. J., Niemen, P. J., Pirttiaho, H. I. & Takkunen, J. T. (1986). Standard EKG: a useful screening method for diabetic autonomic neuropathy. *Diabetes Care* **9**, 103–104.

American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III*), *3rd ed.* APA: Washington, DC.

Bestler, M., Schandry, R., Weitkunat, R. & Alt, E. (1990). Kardiodynamische Determinanten der Herzwahrnehmung. *Zeitschrift für Experimentelle und Angewandte Psychologie* **37**, 361–377.

Brener, J. (1974). A general model of voluntary control applied to the phenomena of learned cardiovascular change. In *Cardiovascular Psychophysiology* (ed. P. A. Obrist, A. H. Black, J. Brener and L. V. DiCara), pp. 365–391. Aldine: Chicago.

Brener, J. & Jones, J. M. (1974). Interoceptive discrimination in intact humans: detection of cardiac activity. *Physiology and Behavior* **13**, 763–767.

Clarke, B. F. & Ewing, D. J. (1982). Resting heart rate and diabetic autonomic neuropathy. *New York State Journal of Medicine* **82**, 908–913.

Clarke, B. F., Ewing, D. J. & Campbell, I. W. (1979). Diabetic autonomic neuropathy. *Diabetologia* **17**, 195–212.

Davis, M. R., Langer, A. W., Sutterer, J. R., Gelling, P. D. & Marlin, M. (1986). Relative discriminability of heartbeat-contingent stimuli under three procedures for assessing cardiac perception. *Psychophysiology* **23**, 76–81.

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

Ehlers, A., Margraf, J. & Roth, W. T. (1988*b*). Selective information processing, interoception, and panic attacks. In *Panic and Phobias 2* (ed. I. Hand & H.-U. Wittchen), pp. 129–148. Springer: Berlin.

Ewing, D. J., Neilson, J. M. M. & Travis, P. (1984). New method for assessing cardiac parasympathetic activity using 24 hour electrocardiograms. *British Heart Journal* **52**, 396–402.

Faerman, I., Faccio, E., Milei, J., Nunez, R., Jadzinsky, M., Fox, D. & Rapaport, M. (1977). Autonomic neuropathy and painless myocardial infarction in diabetic patients. *Diabetes* **26**, 1147–1158.

Flynn, D. M. & Clemens, W. J. (1988). On the validity of heartbeat tracking tasks. *Psychophysiology* **25**, 92–96.

Jones, G. E. & Hollandsworth, J. G. (1981). Heart rate discrimination before and after exercise-induced augmented cardiac activity. *Psychophysiology* **18**, 252–257.

Jones, G. E., O'Leary, R. T. & Pipkin, B. L. (1984). Comparison of the Brener–Jones and Whitehead procedures for assessing cardiac awareness. *Psychophysiology* **21**, 143–148.

Jones, G. E., Jones, K. R., Cunningham, R. A. & Caldwell, J. A. (1985). Cardiac awareness in infarct patients and normals. *Psychophysiology* **22**, 480–487.

Jones, G. E., Jones, K. R., Rouse, C. H., Scott, D. M. & Caldwell, J. A. (1987). The effect of body position on the perception of cardiac sensations: an experiment and theoretical implications. *Psychophysiology* **24**, 300–311.

Kanfer, F. H. & Hagerman, S. (1981). The role of self-regulation. In *Behavior Therapy for Depression* (ed. L. P. Rehm), pp. 143–179. Academic Press: New York.

Katkin, E. S. (1985). Blood, sweat, and tears: individual differences in autonomic self-perception. *Psychophysiology* **22**, 125–137.

Katkin, E. S., Morell, M. A., Goldband, S., Bernstein, G. L. & Wise, J. A. (1982). Individual differences in heartbeat discrimination. *Psychophysiology* **19**, 160–166.

Klosiewski, M. (1984). Hypoglycemia – what does the diabetic experience? *The Diabetes Educator* **10**, 18–21.

Krasnow, A. Z. & Bloomfield, D. A. (1978). The relationship between subjective symptomatology and objective findings from ECG monitoring studies in a large unselected population. In *Ambulatory ECG Monitoring* (ed. S. Stern), pp. 149–169. Year Book Medical Publishers: Chicago.

Kunz, G., Raeder, E. & Burckhardt, D. (1977). What does the symptom 'palpitation' mean? – Correlation between symptoms and the presence of cardiac arrhythmias in the ambulatory ECG. *Zeitschrift für Kardiologie* **66**, 138–141.

Lautenbacher, S., Stalmann, H., Haslbeck, M. & Strian, F. (1987). Neuroendokrine Einflüsse auf die Wahrnehmung der Herzaktion. *Deutsche Medizinische Wochenschrift* **112**, 1492–1497.

Lehmann, W. P., Haslbeck, M., Müller, J., Mehnert, H. & Strian, F. (1985). Frühdiagnose der autonomen Diabetes-Neuropathie mit Hilfe der Temperatursensibilität. *Deutsche Medizinische Wochenschrift* **110**, 639–642.

Levine, B. J., Zeldis, S. M., Morganroth, J. A. & Kastor, J. A. (1978). Lack of correlation between symptoms and significant arrhythmias on long-term electrocardiographic analysis. *Clinical Research* **26**, 247A.

Masaoka, S., Lev-Ran, A., Hill, L. R., Vakil, G. & Hon, E. H. G. (1985). Heart rate variability in diabetes: relationship to age and duration of the disease. *Diabetes Care* **8**, 64–68.

Montgomery, W. A. & Jones, G. E. (1984). Laterality, emotionality, and heartbeat perception. *Psychophysiology* **21**, 459–465.

Pauli, P., Strian, F., Lautenbacher, S., Karlbauer, G. & Hölzl, R. (1989). Emotionale Auswirkungen der autonomen Deafferentierung bei Diabetes-Neuropathie. *Zeitschrift für Klinische Psychologie* **18**, 168–277.

Pennebaker, J. W. (1982). *The Psychology of Physical Symptoms.* Springer: New York.

Pennebaker, J. W. & Hoover, C. W. (1984). Visceral perception versus visceral detection: disentangling methods and assumptions. *Biofeedback and Self-Regulation* **9**, 339–352.

Pennebaker, J. W., Cox, D. J., Gonder-Frederick, L., Wunsch, M. G., Evans, W. S. & Pohl, S. (1981). Physical symptoms related to blood glucose in insulin-dependent diabetics. *Psychosomatic Medicine* **43**, 489–500.

Rouse, C. H., Jones, G. E. & Jones, K. R. (1988). The effect of body composition and gender on cardiac awareness. *Psychophysiology* **25**, 400–407.

Runge, M. & Kühnau, J. (1983). Die autonome kardiale Neuropathie. *Deutsche Medizinische Wochenschrift* **108**, 109–113.

Schandry, R. (1981). Heart beat perception and emotional experience. *Psychophysiology* **18**, 483–488.

Stalmann, H., Hartl, L. & Pauli, P. (1987). Verhaltens-medizinische Aspekte der kardialen Neuropathy bei Diabetes mellitus. In *Verhaltensmedizin und Diabetes mellitus* (ed. F. Strian, R. Hölzl and M. Haslbeck), pp. 145–155. Springer: Berlin.

Stalmann, H., Hartl, L., Pauli, P., Strian, F. & Hölzl, R. (1988). Perception of arrhythmias in mitral valve prolapse, cardiac phobia and panic attacks (abstract). *Journal of Psychophysiology* **2**, 160.

Tyrer, P., Lee, I. & Alexander, J. (1980). Awareness of cardiac function in anxious, phobic and hypochondriacal patients. *Psychological Medicine* **10**, 171–174.

Völker, R., Alt, E., Schmidt, G. & Wirtzfeld, A. (1986). Korrelation subjektiver Symptome und objektiver Befunde im Langzeit-EKG. *Herz/Kreislauf* **18**, 323–328.

Whitehead, W. E., Drescher, V. M., Heiman, P. & Blackwell, B. (1977). Relation of heart-rate control to heart beat perception. *Biofeedback and Self-Regulation* **2**, 371–392.