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Ambulatory psychophysiological monitoring of panic attacks

Jürgen Margraf
Department of Psychology, Philipps-University, Marburg, West Germany

Keywords: ambulatory monitoring, panic attacks, anxiety, clinical psychophysiology, cardiovascular psychophysiology

ABSTRACT Ambulatory psychophysiological monitoring is especially relevant to the study of panic attacks. A review of 10 studies shows that most naturally occurring attacks are of moderate intensity with respect to heart rate, blood pressure, and skin temperature. Many attacks occur without changes in these parameters and there seems to be no special association between panic and cardiac arrhythmias other than sinus tachycardia. These findings contrast sharply with the current view of the intense and dramatic nature of panic attacks. In spite of some methodological problems it is argued that ambulatory monitoring yields a more realistic picture of the physiological changes that correspond to panic attacks. The occurrence of attacks shows a circadian pattern with a peak in the morning hours. The combination of ambulatory monitoring and laboratory research is seen as the most productive strategy for understanding the complex phenomenon of panic.

Introduction

Ambulatory psychophysiological monitoring strategies have only recently begun to be applied to the study of clinical phenomena. For a number of reasons these strategies are especially relevant to the study of panic attacks. Panic attacks are rare events. Even in the case of severe panic disorder the majority of the patients experience less than two panic or limited symptom attacks per week (Margraf et al., 1987a). This has led researchers to study experimentally provoked panic attacks. However, the validity of experimental models of panic remains uncertain unless we compare them to naturally occurring attacks (Ehlers et al., 1986, Margraf et al., 1986). More generally, laboratory studies have less ecological validity than studies in the natural environment. In addition, ethical considerations pose limits on the experimental induction of anxiety unless justified by therapeutic reasons as in the case of exposure treatments for phobias. Finally, more than other forms of pathological anxiety, panic attacks are characterized by a multitude of somatic symptoms that should be reflected in physiological measures. It is not surprising then, that in the past few years an increasing number of researchers have employed ambulatory psychophysiological monitoring in the study of panic attacks. This review discusses studies that investigated naturally occurring panic attacks in the patients' normal environment. Ambulatory monitoring studies that provoked panic attacks by exposing agoraphobic patients to feared stimuli are reviewed by Ost (1990, this issue).

Methods

The first report of ambulatory psychophysiological monitoring of panic attacks appeared in 1983. In this study, Taylor et al., (1983) set the standards for much of the later work. They employed minute-by-minute recordings of heart rate and physical activity as well as a standardized diary in a total of 10 panic disorder patients. Since then, cardiovascular measures have attracted the greatest interest (Ehlers et al., 1988a), probably because palpitations number among the most frequent and distressing symptoms of panic attacks (Barlow et al., 1985; Margraf et al., 1987a) and because the measurement technology is relatively advanced (Turpin, 1985). Table 1 gives an overview of the methods employed in the 10
Table 1: Overview of methods.

<table>
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<tr>
<th>Study</th>
<th>Subjects</th>
<th>Number of attacks</th>
<th>Equipment, recording period</th>
<th>Psychophysiol. measures</th>
<th>Physical activity</th>
<th>Remarks</th>
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<tr>
<td>(1) Taylor et al., 1983</td>
<td>10 PD, 8</td>
<td>PMS, 24 hrs</td>
<td>HR</td>
<td>yes</td>
<td>Event marker, high anxiety control periods</td>
<td></td>
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<tr>
<td>(2) Freedman et al., 1985</td>
<td>12 PD, 11 Con</td>
<td>cassette system, 2 x 12 hrs</td>
<td>HR, ST, AT</td>
<td>no</td>
<td>Blinded ratings of recordings, cut-off between panic and lim. sym. attacks was 3 symptoms</td>
<td></td>
</tr>
<tr>
<td>(3) Taylor et al., 1986</td>
<td>12 PD, 12 Con</td>
<td>PMS (120 hrs), Medcomp (24hrs)</td>
<td>HR, ECG</td>
<td>yes</td>
<td>Diagnosis of patients uncertain</td>
<td></td>
</tr>
<tr>
<td>(4) White &amp; Baker 1986</td>
<td>2 PD, 3</td>
<td>?</td>
<td>HR, BP</td>
<td>no</td>
<td>HR data for 25 panic and 19 lim. sym. attacks, cut-off between panic and lim. sym. attacks was 3 symptoms</td>
<td></td>
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<tr>
<td>(5) Mugger et al., 1987</td>
<td>27 PD, 19 Con</td>
<td>PMS, 72 hrs</td>
<td>HR</td>
<td>yes</td>
<td>HR data for 25 panic and 19 lim. sym. attacks, cut-off between panic and lim. sym. attacks was 3 symptoms</td>
<td></td>
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<tr>
<td>(6) Shear et al., 1987</td>
<td>23 PD, 18 (46 lim. sym. attacks)</td>
<td>?</td>
<td>HR, ECG</td>
<td>no</td>
<td>175 non-panic anxiety intervals recorded</td>
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<td>(7) Galley et al., 1988</td>
<td>10 PD, 10 Con</td>
<td>Avionics, PMS, Medilog</td>
<td>HR, ECG</td>
<td>yes</td>
<td>HR data for 31 attacks, cut-off between panic and lim. sym. attacks was 3 symptoms</td>
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<tr>
<td>(8) Hibbert &amp; Pillsbury 1988</td>
<td>4 PD, 3 Con</td>
<td>Avionics, PMS, Medilog</td>
<td>HR, PP, ECG</td>
<td>no</td>
<td>Diagnosis of patients uncertain, event marker, no results on RR and ECG presented</td>
<td></td>
</tr>
<tr>
<td>(9) Shear et al., unpublished study</td>
<td>22 PD, 25 Con</td>
<td>Pressure-meter II, 24 hrs</td>
<td>HR, BP, ECG</td>
<td>no</td>
<td>Event marker, control group composed of subjects from earlier study, ECG results not reported</td>
<td></td>
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<tr>
<td>(10) Mugger et al., unpublished study</td>
<td>118 PD, 277 (404 lim. sym. attacks)</td>
<td>PMS, 72 hrs</td>
<td>HR</td>
<td>yes</td>
<td>HR data for 45 panic and 85 lim. sym. attacks</td>
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Abbreviations:

Studies of unprovoked panic attacks in the natural environment reviewed here. All studies investigated cardiovascular measures and employed standardized diaries for self-report measures. All subjects were drug-free for a sufficient time prior to and during ambulatory monitoring with the exception of the White and Baker (one patient received 0.5 mg of alprazolam every 6-8 hours) and Hibbert and Pillsbury (no information given) studies. In most studies, patients met DSM-III or DSM-III-R criteria for panic disorder or agoraphobia with panic attacks. Unfortunately, half of the studies did not record physical activity levels. This makes it difficult to draw firm conclusions, especially with respect to cardiovascular variables.
Results

Heart rate

The largest body of data is available for heart rate. The results show that many, but by far not all panic attacks are accompanied by increases in heart rate, usually of moderate intensity. Taylor et al. (1983) used computerized algorithms to determine high heart rate levels disproportionate to concurrent physical activity levels. Three of the eight attacks recorded met the criteria with maximum heart rates ranging from 115 to 148 beats per min (bpm). During one other attack the patient had a high heart rate, but was also physically active. The remaining four panic attacks occurred at heart rates that were not higher than expected from activity levels (between 80 and 100 bpm). In their second study, Taylor et al. (1986) had blind raters evaluate heart rate and activity patterns. During 16 of 25 (64%) panic attacks and three of 16 (19%) limited symptom attacks, heart rate increases measured at least 20 bpm and were greater than expected from concurrent activity levels. The mean maximum heart rate during these attacks was 117 bpm. The patients rated these attacks as more intense than attacks occurring without heart rate changes. However, symptom patterns including cardiovascular complaints were not significantly different.

Margraf et al. (1987a) used a different approach to data analysis. They studied the average pattern of heart rate changes during 41 panic and limited symptom attacks ("panic"), the 15 min preceding the attacks ("baseline"), and matched control periods recorded 24 hours after the attacks ("matched"). The 17 panic and limited symptom attacks classified by the patients as 'spontaneous' (no situational trigger perceived) were, on the average, not characterized by heart rate elevation. Mean heart rates for spontaneous panic attacks during baseline, panic, and matched periods were 82, 83, and 81 bpm, respectively (81, 79, 81 for spontaneous limited symptom attacks). Mean peak heart rates showed the same pattern of results (99, 99, 95 bpm for panic and 100, 94, 97 bpm for limited symptom attacks). Panic attacks occurring in feared situations were accompanied by elevated heart rates compared to matched periods (mean bpm for eight 'situational' panic attacks: 90 during panic and 82 during matched periods; eight 'situational' limited symptom attacks: 94 and 81 bpm, respectively). Interestingly, heart rates were already elevated during the 15 min period preceding the actual attacks (88 and 92 bpm for panic and limited symptom attacks, respectively). There were no significant differences between baseline and panic periods. As with 'spontaneous' attacks, peak heart rates showed a similar pattern of results (99, 110, 101 for panic and 103, 108, 99 bpm for limited symptom attacks during baseline, panic and matched periods). Across all attacks, the average heart rate difference between panic and matched periods was only 5 bpm. During baseline and matched periods physical activity and heart rate correlated significantly (r = 0.47 and 0.56). However, there was no significant correlation during panic periods (r = 0.27, and r = 0.21, respectively) and changes in activity were not statistically significant. These results support the notion that the small heart changes during panic periods represent an effect of anxiety rather than of physical workload.

White and Baker (1986) observed no significant heart rate increase in three panic attacks. Freedman et al. (1985) found heart rate increases in all of the eight panic attacks recorded. Peak heart rates ranged between 80 and 120 bpm. Gaffney et al. (1988) reported that out of 31 spontaneous attacks 17 showed no change, eight were associated with increased heart rate, and six were accompanied by increases in both heart rate and physical activity. Mean heart rate for 10-min epochs 1 hour before, during, and 1 hour after the panic attacks were 93, 108, and 94 bpm, respectively, with standard deviations ranging from 25-30 bpm. This difference reached borderline significance (p < 0.06). Shear et al. (1987) found mean heart rates of 108 (+/- 14) bpm during 18 panic intervals and 91 (+/- 13) bpm during the preceding baseline intervals. In their second study, Shear et al. (unpublished) reported lower values. The mean heart rate during panic was 86 (+/- 17) bpm with a range from 64 to 102 bpm. This represented a mean increase of 15 bpm (range 3-25) over the 5-min period before the panic attack. Unfortunately, in all but Gaffney's studies measures of actual physical activity were lacking.

In the largest study to date, Margraf et al. (in preparation) replicated most of their pre-
arious psychophysiological findings in a sample of 130 attacks. In this study, there were no significant differences in heart rate or physical activity between spontaneous and situational or between panic and limited symptom attacks. Heart rate was significantly higher during panic than during matched control periods (recorded 24 hours later). Baseline periods showed similar significant heart rate elevations meaning that panic attacks occurred out of a background of heightened arousal (this was true for both 'spontaneous' and situational attacks). Across all attacks, mean heart rates were 89, 89, and 84 bpm for baseline, panic, and matched periods, respectively (105, 105, 100 bpm for peak heart rates). During panic periods, heart rates ranged from 60 to 148 bpm. When compared to matched periods, 14 attacks occurred without heart rate changes, 50 with decreases, and 66 with increases. The range of heart rate differences between panic and matched periods was -24 to +67 with a mean of 5 bpm. Again, heart rate changes during panic periods could not be attributed to differences in physical activity. Interestingly, the patients' report of palpitations was reflected in the heart rate recordings. During attacks with palpitations heart rates were about 9 bpm higher than during matched periods. For attacks without palpitations this difference was less than 2 bpm. An additional purpose of this study was to test the reactivity of measurement. It is conceivable that patients are reassured by wearing the physiological monitoring equipment. This could in part explain the rather moderate intensity of the changes associated with panic attacks. For this purpose, patients kept their panic attack diaries for three additional days without wearing the monitor. A comparison of days with and without monitor, however, yielded no difference in the number of panic attacks experienced. Finally, there were no heart rate or activity differences between the attacks of patients with or without agoraphobia.

Taken together, these studies suggest that a fair number of panic attacks occur with measurable changes in heart rate that are not caused by differences in physical workload. However, these changes are typically of moderate intensity and the variability between attacks is pronounced. A large subgroup of attacks occur without heart rate increases. So far there are no indications of particular measurement reactivity and the patients' reports of palpitations are reflected in the heart rate data.

Cardiac arrhythmias
The possible contribution of cardiac arrhythmias has been a second focus of interest in ambulatory monitoring of panic attacks. On the whole, the results argue against a relationship between panic attacks and arrhythmias other than sinus tachycardia. In the study of Taylor et al. (1986) six of the panic attacks occurred during simultaneous ECG monitoring. During these attacks, the heart rate, when elevated, represented a sinus tachycardia rather than any other type of atrial or ventricular arrhythmia. There was no difference in the overall frequency of arrhythmias between patients and controls. Similar results were reported by Gaffney et al. (1988). In this context, a study of 67 patients with 'neurocirculatory asthenia' and 33 healthy controls may be of interest (Tizioni et al. 1980). During 24-hour ECG monitoring most patients but only few controls showed episodes of sinus tachycardia in the absence of self-reported unusual physical effort. However, the study did not use actual measures of physical activity and results were not statistically tested. Moreover, the results cannot be generalized to panic disorder patients as only 39% of the patients of this study reported anxiety. Shear et al. (1987) observed a high mean daily density of ventricular premature complexes in their panic patients but reported that complex ventricular arrhythmias were 'distinctly uncommon' and that the great majority of panic episodes occurred without arrhythmias. Unfortunately, this study did not have a control group. The two controlled studies of arrhythmias mentioned above (Taylor et al., 1986; Gaffney et al., 1988) showed no overall difference between patients and normal controls. Similarly, a study by Harbauer-Raum (1987) found no difference in the frequency of cardiac arrhythmias between 23 patients with cardiac neurosis and 16 healthy controls. In contrast, 21 patients with mitral valve prolapse had more frequent ventricular premature beats. Since there is a strong overlap between panic disorder and cardiac neurosis, this study gives further evidence against a higher frequency of arrhythmias in panic disorder. The
study by Herbauer-Raum (1987) is especially interesting because subjects were also asked to press the event marker whenever they perceived an arrhythmia. Cardiac neurotics were significantly better in perceiving their arrhythmias and responded to them with greater anxiety than either of the other groups. This result clearly deserves replication in a better diagnosed sample of panic disorder patients. Taken together, the results of ECG monitoring argue against a special association of arrhythmias other than sinus tachycardia with panic attacks or an elevated prevalence of such arrhythmias in panic patients.

Other measures
The third cardiovascular variable studied was blood pressure. White and Baker (1986) reported large and sustained rises especially in systolic blood pressure associated with panic attacks in two otherwise normotensive patients. Shear et al. (unpublished) found mean systolic blood pressures of 125, 109, and 113 mm Hg during panic attacks, limited symptom attacks and non-panic anxiety, respectively. This compared to 104 mm Hg during non-anxious periods. The values for diastolic blood pressure were 83, 78, 76, and 69 mm Hg. There were no significant differences in 24-hour blood pressure levels between patients and controls (109/76 vs. 114/77, respectively). Similar to the heart rate results, blood pressure increases during panic were only moderate, and for a given patient the values during panic were not necessarily the highest in a 24-hour recording. Moreover, panic values were only minimally higher than average blood pressures in the normal control group during work.

A last group of measures has been recorded only in single studies. Freedman et al. (1985) observed an increase in finger temperature prior to the event mark in panic attacks. Maximum temperature increases per minute were 0.3 to 2.3 °C during the 5 min before the event mark but only 0 to 0.6 °C during control periods. Temperature usually decreased at the time of the event mark. This pattern of temperature changes did not occur during high-anxiety control periods and is different from the changes found in experimental panic induction studies (Bloom et al., 1976; Ekman et al., 1983; Ehlers et al., 1986; Margraf et al., 1986).

The authors discuss the possibility that the finger temperature changes could have been associated with changes in anger rather than anxiety. Four of the eight attacks were reported to have been triggered by arguments in which the patients felt angry and experimental induction of anger has been shown to increase finger temperature (Ekman et al., 1983). Finally, Hibbert and Pilbury (1988) reported decreases in transectaneously measured pCO2 that were related to episodes of panic attack symptoms. However, there was no one-to-one relationship even in this limited sample of five panic attacks. At present, this pilot study primarily shows the feasibility of ambulatory pCO2 monitoring. Considering the emphasis that many clinicians place on hyperventilation in panic attacks this technology deserves further application.

Circadian rhythms
Six studies gave information on the time of occurrence of panic attacks (Taylor et al., 1986; White and Baker, 1986; Margraf et al., 1987a; in preparation; Hibbert and Pilbury, 1988; Shear et al., unpublished). Figure 1 gives the percentage of all panic and limited symptom attacks occurring at different times of day. Since Hibbert and Pilbury (1988) did not monitor their cases for a full 24 hours these results are not included here. The upper part of the figure shows the results for situational and spontaneous attacks separately. Since the distinction between these types of attacks was not made in the studies by White and Baker (1986) and Shear et al. (unpublished) their data are not included in this part of the figure. The lower part of Figure 1 shows the data for all attacks recorded in all five studies.

The curves are rather similar for situational and spontaneous attacks with the exception of more spontaneous attacks during the night. This must probably reflects the fact that patients perform fewer phobic activities during the night (e.g. shopping). More interesting is that the occurrence of attacks shows a clear circadian pattern with a peak in the morning hours. This finding may be relevant in the light of the relationship between anxiety and depressive disorders since the mood states of depressed patients often show a similar pattern.
Figure 1 Percentage of panic and limited symptom attacks by time of occurrence (tabulated from Taylor et al., 1986, Margraf 1987a, in preparation). The upper part of the Figure shows the results for situational and spontaneous attacks, respectively. The lower part of Figure 1 shows the combined data for all 511 attacks recorded in the studies of Taylor et al. (1986), White & Baker (1986), Margraf et al. (1987a, in preparation), and Shear et al. (unpublished).

Discussion

Ambulatory psychophysiological monitoring has clarified some of the physiological changes that accompany panic attacks. The findings reviewed in the present paper contrast sharply with the currently predominant view of an intense and dramatic nature of such attacks. This view is primarily based on clinical descriptions of patients applying for treatment and on case reports of panic attacks recorded 'by chance' in the laboratory. These case reports described very large and abrupt physiological changes, especially in heart rate (Lader and Mathews, 1970; Cohen et al., 1985; Margraf et al., 1987b). Increases of 40 to 50 bpm within 30 to 90 sec as recorded in these reports are clearly an exception compared to naturally occurring panic attacks. In fact, most unprovoked panic attacks recorded in the natural environment are of moderate intensity with respect to heart rate, blood pressure, and skin temperature. Heart rate levels reached during panic attacks are much lower than those during mild exercise. Many attacks occur even without changes in the parameters measured and there seems to be no special association between panic and cardiac arrhythmias other than simple sinus tachycardias.

How can we explain the apparent contradiction between ambulatory monitoring and clinical or case reports? The surprising difference
in results reminds one of the discussion of discordance and desynchrony of different measures of fear (Hodgson and Rachman, 1974; Hirnadi et al., 1985). However, in ambulatory monitoring there was rather good agreement between self-report and physiological measures. For instance, the patient's report of palpitations was borne out by heart rate recordings. Attacks with palpitations occurred with substantially greater heart rate changes than attacks without palpitations (Margraf et al. in preparation). The self-report data support the notion that panic attacks on the average are of only modest intensity. Taylor et al. (1986) and Margraf et al. (1987a, in preparation) found only moderate levels of anxiety and panic symptoms for the majority of attacks. Similar agreement exists with respect to the comparison of so-called spontaneous and situational attacks. Other than situational context there were no consistent differences in self-report and psychophysiological variables between these types of attacks. Finally, a lack of differences between the attacks of panic patients with and without agoraphobia was found in both self-report and psychophysiological variables. Thus, it seems that the differences between clinical and ambulatory monitoring findings are more than just the "normal" desynchrony.

Is it possible that ambulatory monitoring studies underestimate physiological changes? Most studies monitored only two or three variables. The sensations leading to symptom report may correspond to changes in cardiac functions not yet studied. For a more complete picture of the cardiovascular psychophysiology of panic attacks other parameters such as stroke volume, contractility of the heart, renal blood flow, coronary artery status, retinal arterial status, coronary ischemia, and oxygen consumption would have to be studied. A second problem is that the timing of panic attacks relies exclusively on self-report. Especially in the case of diaries this may be unreliable. In our own studies we found an excessive proportion of attacks that were recorded on the full or half hour. Event markers are a better method but still a form of self-report. It is nonetheless unlikely that these first two problems explain the modest nature of the physiological changes observed in ambulatory monitoring studies as the results on anxiety levels and panic symptoms concurred with the psychophysiological variables. Theoretically, patient selection bias could result from the inconvenience associated with wearing the monitor. In our experience, however, it is extremely rare that a patient declines participation in the study for this reason. This coincides with Hippiot's (1986) observation that on the whole panic patients are grateful for a physical approach to investigation and are happy to be monitored. Finally, there is a possibility that patients change their daily activities because of wearing a monitor (cf. Shear, 1985). Nevertheless, there were no differences in panic attack frequency on days with and without monitor in the one study that addressed this question (Margraf et al., in preparation).

At present, it seems more likely that methodological problems with the clinical and case reports explain the discrepancy. First, recollection bias may play a role in clinical descriptions. Margraf et al. (1987a, in preparation) showed that panic patients endorse considerably more symptoms in retrospective accounts of their panic attacks (in questionnaires or diagnostic interviews) than in concurrent diary reports made at the actual time of the panic attack. Second, case reports of laboratory recorded spontaneous attacks may represent a publication bias. It is conceivable that these attacks are only recognized and published if they occur with noticeable changes. Indeed, the only group laboratory study of unprovoked panic attacks failed to find any consistent pattern of physiological changes during panic attacks (Cameron et al., 1987). These authors monitored several psychophysiological and biochemical measures in patients with frequent panic attacks who stayed in bed for extended periods of time. Similarly, the average heart rate increase observed in studies of experimentally induced panic is rather moderate (Ehlers et al., 1986, 1988a; Margraf et al., 1986).

For a full understanding the relationship between symptom report and physiological changes has to be addressed on a more general level. The symptom report of panic patients reflects at least to some degree their appraisal of body sensations rather than actual physiological responses. This is in line with what we have termed the psychophysiological model of panic attacks (Margraf et al., 1986; Ehlers et al., 1988b; Ehlers and Margraf, 1989).
suggestion following from this model is that
interceptive processes and their association
with threat need to be studied in panic
patients. Recent research supports the role of
these variables in panic disorder. Ehlers and
her associates established that panic patients
show significantly more accurate cardiac
awareness than patients with other anxiety
disorders or normal controls and that panic
patients respond with larger increases in anxi-
ety and physiological arousal to perceived
heart rate acceleration (Ehlers, 1989; Ehlers
et al., 1988b; 1988c). As discussed before,
similar results have been found in 'cardiac
neurotics' (Furhauer-Raum, 1987). A large
corpus of research has established specific
characteristics of panic patients in the
appraisal of bodily sensations (reviewed by
Ehlers and Margraf, 1989, Margraf and Ehlers,
1989). At the very beginning of a panic attack,
patients usually notice some kind of unpleasant
body sensation (Hibbert, 1984; Ley, 1985;
Zucker et al., 1989). This is supported by the
finding from the ambulatory monitoring stud-
ies reviewed here that heart rate levels are
already elevated before the actual panic attack
is reported.

The circadian pattern of occurrence seen in
both situational and spontaneous attacks is
reminiscent of the changes in mood observed
in many depressed patients. It is also similar to
the diurnal variation of cortisol secretion
that has been linked to some depressive states (e.g.
Sachar, 1982). This is especially interesting in
the light of the discussion of the relationship
between depression and anxiety (e.g. Angst
and Dobler-Mikola, 1985; Stavrakaki and
Varga, 1986; Tyers et al., 1987). However, the
circadian rhythms presented here have not
been established across a large number of patients
rather than within each individual patient.
Since this method could cause artefacts it
would be premature to draw firm conclusions.
In any case, possible circadian rhythms deserve
further investigation.

In conclusion, the earlier clinical and case
reports appear to be misleading. The intense
attacks described in these reports seem to
represent extreme prototypes rather than the
average panic attack. Ambulatory monitoring
has helped us to gain a more realistic under-
standing of the physiological changes that
correspond to symptom reports in panic attacks.

It has also identified a possible circadian
rhythm in the occurrence of these attacks. In
addition, this strategy provides a standard
against which potential experimental models
of panic attacks can be compared. Considering
the specific advantages and disadvantages that
each approach has, the combination of ambu-
latory monitoring and laboratory research
seems to be the most productive strategy for
understanding the complex phenomenon of
panic. The recent advances in the technology
of ambulatory monitoring should provide a
further argument for the wider use of this
approach.

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Address for correspondence

Dr Jürgen Margraf,
Fachbereich Psychologie,
Gutenbergstr. 18,
D-3550 Marburg,
West Germany.

References

Vf. A continuum from depression to anxiety disorders?
European Archives of Psychiatry and Neurological Sci-
cences, 235, 173-186.

Barlow, D. H., Vernilye, J., Blanchard, E. B., Verni-
phenomenon of panic. Journal of Abnormal Psychology,
94, 320-328.

Bloom, L., Houston, B., & Burish, T. (1976) An evalua-
tion of finger pulse volume as a psychophysiological
measure of anxiety. Psychophysiology, 13, 40-42.

Cameron, O. G., Lee, M. A., Curtis, C. G., & McCann,
D. S. (1977) Endocrine and physiological changes during
'spontaneous' panic attacks. Psychoneuroendocrinology,
12, 221-231.

Psychophysiology of relaxation-associated panic attacks.

Ehlers, A. (1989) Interaction of psychological and physio-
logical factors in panic disorder. In: P. F. Leviboni, &
P. Wilson (Eds.) Proceedings of the 24th International
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