10. Panic Attacks in Nonclinical Subjects

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Introduction

Sudden episodes of intense anxiety accompanied by a number of predominantly somatic symptoms (now usually called panic attacks) are the primary feature of the psychological disturbance termed panic disorder in DSM-III-R (APA 1987). This diagnosis was introduced based on the idea that panic attacks are a distinct type of anxiety in need of their own diagnostic entity. However, recent research has shown that panic attacks are not specific to panic disorder since they also occur across a wide range of other psychological disorders and even in nonclinical populations. Barlow et al. (1985) studied 108 patients with the DSM-III diagnoses simple phobia, social phobia, generalized anxiety disorder, panic disorder, agoraphobia with panic attacks, obsessive-compulsive disorder, and major depressive episodes. The great majority of patients in each of these categories (at least 83%) reported having experienced panic attacks. Although the frequency of attacks varied across diagnoses, there were only few differences in terms of the symptom pattern associated with the attacks. Furthermore, symptom severity was similar for patients with situational (predictable, expected) and spontaneous (unpredictable, unexpected) attacks. These results are in line with those of the Munich Follow-Up Study (MFS, Wittchen 1986) in which panic attacks were observed in 9.3% of a representative community sample, a percentage higher than the combined frequencies of panic disorder and agoraphobia with panic attacks.

If it is established that panic attacks are not specific to people suffering from a specific disorder, it is important to study the distribution of the phenomenon in the general population. There have been a first few attempts to approach this question using questionnaire screening methods. Norton et al. (1985, 1986) initiated this line of research. They found surprisingly high prevalences of panic attacks in nonclinical subjects. About one-third of their two samples of undergraduate students reported having experienced at least one panic attack in the past year. They concluded that panic attacks often occur in presumably normal people and that these panic attacks share many similarities with those of patients who have well-defined panic disorders (Norton et al. 1985).

Aside from the question of the distribution of panic attacks in the population, there are at least two other important reasons to study panic attacks in nonclinical subjects. The first reason concerns sampling bias. With the exception of large epidemiological studies such as the Epidemiological Catchment Area Program (ECA, Regier et al. 1984) or the MFS (Wittchen 1986) previous studies of panic attacks have exclusively investigated clinical populations. There are a number of reasons to expect that these samples...
represent a biased selection of the total population of persons with panic attacks. Highly symptomatic individuals are more likely to seek treatment or to be detected in clinical screenings, an effect Motulsky (1978) termed "ascertainment bias." This bias increases the probability of persons with two disorders to be part of clinical samples and thus may lead to mistaken assumptions about the relationship between such disorders. For example, almost all agoraphobic patients seen in clinical settings have panic attacks (cf. Mendel and Klein 1969; Thyer and Himle 1985). In nonclinical community samples, however, the picture looks quite different. Weissman et al. (1986) and Wittchen (1986) found that only a small percentage of all subjects meeting criteria for agoraphobia also exhibited panic disorder (ranging from 6% to 16% in the various ECA and MFS sites) and only another 17% to 50% showed limited panic symptoms in addition to agoraphobia.

Another example for sampling bias is the postulated relationship between panic disorder and mitral valve prolapse (MVP). We have argued elsewhere (Margraf et al., 1988) that the higher prevalence of MVP observed in some studies of panic disorder patients represents a problem of comorbidity rather than a true functional relationship. If the sampling bias inherent in studying clinical samples is eliminated, the association between panic attacks and MVP vanishes. This was shown by Hartman et al. (1982) and Devereux et al. (1986), who studied MVP patients and their family members who had not sought treatment themselves. Family members with and without MVP were not different from each other with respect to number of panic attacks and other symptoms; both groups were far less symptomatic than the original sample of MVP patients who had been referred to the clinic. Thus, sampling bias may strongly influence the results of research on clinical samples. It is therefore important to complement such research by studies of the characteristics of nonclinical subjects with the same disturbance.

A second important reason to study nonclinical panickers is related to the fact that many of these people are infrequent panickers. They experience fewer attacks than patients who seek treatment for the full-blown syndrome. Infrequent panickers may form the basic population out of which some people will continue to develop the full clinical syndrome. If this is the case, infrequent panickers offer a unique opportunity to study possible vulnerability factors for panic attacks. Most of our current etiological research on panic attacks is correlational in the sense that groups of panic patients and controls are compared in cross-sectional designs. True experimental designs would for instance involve attempts to produce panic disorder in previously "normal" subjects. For obvious reasons such studies cannot be conducted. Thus, we cannot make firm statements about causal antecedents of the disorder. If infrequent panickers are the basic population for panic disorder they should show whatever diathesis for panic exists. In this case we can assume that characteristics of patients that are not found in infrequent panickers are consequences rather than causes of the disorder. On the other hand, infrequent panickers may represent a different basic population than frequent panickers (panic disorder patients). It is possible that even though both groups show identical manifest symptoms (panic attacks) they suffer from different underlying disturbances. In this case, infrequent panickers should be studied to gain insight into such a heterogeneity of the panic attack phenomenon.

Questionnaire Studies

The first studies of panic attacks in presumably normal populations were reported
by Norton et al. (1985, 1986). In their initial study, 186 students were screened using the Hopkins Symptom Checklist (HSCL-90, Derogatis et al. 1973) and a specially designed anxiety questionnaire asking for current levels of anxiety as well as frequency and symptoms of panic attacks. A striking 34.4% of the subjects reported having had one or more panic attacks in the past year, and 2.2% reported having had at least three attacks in the past 3 weeks. The symptoms reported to occur during these attacks were similar to the ones described by clinical samples of panic attack patients (Barlow et al. 1985; Margraf et al. 1987). The most severe symptoms were heart pounding, trembling, sweating, flushing, and dizziness. Subjects describing at least one panic attack in the anxiety questionnaire scored significantly higher than those without attacks on the HSCL-90 subscales anxiety, phobic anxiety, depression, interpersonal sensitivity, somatization, and anger/hostility. There were no significant differences with respect to obsessive-compulsiveness, psychotism, paranoid ideation, or sleep difficulties.

In a second study, Norton et al. (1986) screened 256 students with a refined version of their questionnaire, now termed the Panic Attack Questionnaire (PAQ). Subjects also completed the State-Trait Anxiety Inventory (STAI, Spielberger et al. 1970), the Beck Depression Inventory (BDI, Beck et al. 1961), and the Profile of Mood States (POMS, McNair et al. 1981). In addition, subjects were either given the Fear Survey Schedule (FSS-III, Arrindell 1980) or the Fear Questionnaire (FQ, Marks and Mathews 1979). Very similarly to the first study, 35.9% of the sample reported having experienced at least one panic attack in the past year, and 3.1% reported having had at least three attacks in the past 3 weeks. Panicakers scored significantly higher on state and trait anxiety (STAI, anxiety scale of the POMS), depression (BDI, depression scale of the POMS), fatigue (POMS), and anger (POMS). In contrast, there were no differences compared to nonpanicers on any of the FSS-III or FQ subscales (agoraphobia, social phobia, blood/injury phobia, aggression, animal phobia) or the POMS scales activity and confusion. Panicakers and nonpanicers were similar in the frequency of reported previous treatments for any mental or physical disorder. Similarly, the two groups were comparable with respect to age, sex, or socioeconomic status. Panicakers reported significantly more first-degree relatives who had panic attacks.

As in the first study, the most severe symptoms of panic attacks were palpitations, trembling, sweating, dizziness, and hot/cold flashes. Other characteristics of panic attacks included a sudden onset in the majority of cases (59% under 10 min), an average of eight DSM-III symptoms, and a wide variety of situational contexts in which attacks occurred, especially social situations. The great majority of panicakers reported having experienced at least one life stressor at the onset of their panic attacks. Most frequently mentioned were difficulties at work, family crises, and loss of a significant other. Subjects who experienced some unpredictable attacks were different from those who experienced only predictable attacks on 9 out of 40 comparisons. Those subjects with unpredictable attacks reported more attacks in more different situations, as well as more severe feelings of unreality and tachycardia. Together, these studies show that panic attacks may occur in more persons than previously assumed and that subjects who have panic attacks report more psychopathology than do nonpanicers. In addition, the panic attacks experienced by nonclinical panicers and patients with anxiety disorders are very similar. While these studies have yielded some fascinating data and initiated an important line of research, they also pose some new chal-
challenges. A first problem is to establish the reliability and validity of the PAQ as compared to standard structured interview diagnoses. Norton et al. (1986) reported that 22 out of 24 cases, previously identified as nonclinical panickers by the PAQ, also met DSM-III criteria for panic attacks in a structured interview. However, they did not give information as to what interview was used, whether interviewers were blind to the questionnaire results, and the reliability of their interview and questionnaire methods. A second challenge is to go beyond mere questionnaire assessment of psychopathology and to compare nonclinical panickers, clinical panickers, and normal controls on psychophysiological variables or the response to stressors. More recent studies have attempted to address these issues.

**Laboratory studies**

Beck and Scott (1987) compared ten subjects who had panic disorder with ten infrequent panickers. All subjects were recruited from the community using media announcements. Infrequent panickers were defined as subjects reporting four of the 12 DSM-III symptoms during typical attacks, but never having experienced three attacks in 3 consecutive weeks. The screening instrument was the Anxiety Disorder Interview Schedule (ADIS-R, DiNardo et al. 1983). Laboratory paradigms were four 2-min tasks (neutral imagery, hospitalization imagery, signal detection, paced arithmetic). Assessments included continuous measurement of trapezous electromyogram (EMG), skin conductance level (SCL), and heart rate, as well as thought-listing and ratings of the DSM-III panic symptoms immediately following each task. Overall, there were relatively few differences between panic disorder subjects' and infrequent panickers' responses to the test paradigms.

Panic disorder subjects showed more EMG and SCL reactivity to the two imagery tasks, infrequent panickers more EMG and SCL reactivity to paced arithmetic. There were no differences in heart rate reactivity or on the thought-listing measure. With respect to panic symptoms, panic disorder subjects scored significantly higher on four and infrequent panickers scored higher on six out of 52 comparisons.

Sandler et al. (1987) reported a comparison of nonclinical panickers and control subjects without panic attacks. Eighty subjects were recruited through screening of college students with a panic attack questionnaire. Subsamples included frequent panickers, infrequent panickers, and panic-free controls. Cardiovascular reactivity to a psychological (challenge reaction time task) and a physical (cycling) stress task was assessed by measuring heart rate and blood pressure at intervals before, during, and after the tasks. All of the measures showed progressive declines during the pretask baseline periods, increases during the stress tests, and declines during the post-task recovery phases. There were no differences between groups with respect to cardiovascular reactivity. This is similar to what is seen in most so-called panic induction studies of clinical samples (cf. Ehlers et al. 1986a, 1986b, 1988; Margraf et al. 1986a). In contrast to these studies, however, differences in baseline heart rate levels were lacking. The only apparent difference was some modest evidence for slower recovery from exercise in those subjects reporting the most frequent occurrence of panic attacks.

It is interesting to note that neither Sandler et al. (1987) nor Beck and Scott (1987) found differences in cardiovascular reactivity. There were few reactivity differences between frequent and infrequent panickers in Beck and Scott's study and no reactivity differences between panickers and nonpanickers in Sandler et al.'s
study. This is in contrast to the consistent and strong differences on several of the questionnaire measures in the two Norton studies. We have recently conducted two studies trying to combine questionnaire and laboratory measures in a comparison of nonclinical panickers and controls.

The Marburg and Tübingen Studies

During late 1986 and early 1987 we conducted two independent studies of nonclinical panickers. The first study (Ehlers and Meisner, in preparation) involved a sample of 170 undergraduate students at Philipps University in Marburg in the central part of West Germany. The second study (Margraf, Wrobel, and Jakschik, in preparation) involved a sample of 136 undergraduate students at the university of Tübingen in southern Germany. These studies pursued three specific goals:

1. To replicate the Norton et al. (1985, 1986) findings using a German translation of their PAQ
2. To determine the reliability and validity of the questionnaire screening method as compared to a standard structured interview approach
3. To compare nonclinical panickers and controls on a questionnaire battery and a psychophysiological laboratory assessment

Figure 1 gives an overview of the studies. Each study consisted of three phases. First, a large group of undergraduate students was screened using our German translation of the PAQ. The definition of a panic attack given to the subjects is shown in Table 1.

Table 1. Definition of a panic attack given in the version of the panic attack questionnaire used in the German studies

A panic attack (anxiety attack) is a discrete period of sudden onset of intense apprehension, fear, or terror, often associated with feelings of impending doom. The following symptoms may be experienced:

- Racing, pounding, or irregular heart beat
- Dizziness or lightheadedness
- Shortness of breath
- Sweating
- Chest pain or discomfort
- Trembling or shaking
- Hot and cold flashes
- Choking or smothering sensations
- Numbness or tingling in parts of the body
- Fear of dying
- Faintness
- Nausea or abdominal distress
- Feelings of unreality or being detached
- Fear of losing control or going crazy

The questions on the following pages refer to panic attacks in situations that were not life-threatening. The attacks have to be accompanied by at least four of the symptoms listed above.
In phase two, we selected subsamples of PAQ-determined panic attacks and nonpanickers or “controls” (about 50% each) using strict criteria (panickers: reporting at least one spontaneous attack, at least four symptoms, attacks not only in social situations; controls: no attacks or anxiety symptoms). We then conducted blind diagnostic interviews to determine the agreement between the interview and questionnaire methods. The interview was a German translation of the Structured Clinical Interview for DSM (SCID) by Spitzer and Williams (1986). In the third phase, subjects meeting both PAQ and SCID criteria for panic attacks (“nonclinical panic attacks”) and controls were compared using an extensive questionnaire battery and a psychophysiological laboratory assessment involving a baseline and a hyperventilation task. In addition, two substudies assessed the retest reliability of the PAQ and the interrater reliability of the SCID in our hands. In the following, we will present the results of the preliminary analyses conducted so far.

The retest reliability of the PAQ proved to be generally good. Similarly, the interrater reliability of the SCID in our hands was good. Table 2 summarizes the results of the retest reliability study.

Information about the occurrence, number, and intensity of attacks as well as stress at the onset of panic, avoidance behavior, and family history was given reliably. In contrast, subjects were not able to give reliable information about whether they had ever experienced unexpected (“spontaneous”) attacks, panicked only in social situations, or experienced most of their symptoms within 10 min.

Table 3 compares the number of subjects reporting panic attacks on the PAQ in our two studies with the numbers reported by Norton et al. (1985, 1986). The mean ages of our samples were 24 (study 1) and 25 years (study 2), while 69% (study 1) and 65% (study 2) of all subjects were female.

While we found a somewhat higher percentage of panic attacks for the past year, results for the past 3 weeks closely resemble those of Norton et al. (1985, 1986). Thus, their finding of a high percentage of nonclinical panic attacks is replicated using the questionnaire method. However, when using the structured interview approach, a different picture emerge. We found that only 12 out of 23 (study 1, Marburg) and 15 out of 29 (study 2, Tübingen) PAQ-determined panic attacks also met SCID criteria for panic attacks. While we thus had a high rate of false positives, there were only a few false negatives:

<table>
<thead>
<tr>
<th>Item (or groups of items)</th>
<th>Reliability coefficient</th>
<th>Statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ever had panic attack</td>
<td>0.80</td>
<td>Kappa</td>
</tr>
<tr>
<td>Ever had 3 attacks in 3 weeks</td>
<td>0.89</td>
<td>Kappa</td>
</tr>
<tr>
<td>Ever worried for 4 weeks about attack</td>
<td>0.82</td>
<td>Kappa</td>
</tr>
<tr>
<td>Number of attacks past year</td>
<td>0.85</td>
<td>Spearman</td>
</tr>
<tr>
<td>Most symptoms within 10 min</td>
<td>0.33</td>
<td>Kappa</td>
</tr>
<tr>
<td>Ever had unexpected panic</td>
<td>0.53</td>
<td>Kappa</td>
</tr>
<tr>
<td>Panics only in social situations</td>
<td>0.43</td>
<td>Kappa</td>
</tr>
<tr>
<td>Stress at onset of panic (6 items)</td>
<td>0.67-1.0</td>
<td>Kappa</td>
</tr>
<tr>
<td>Age of onset, treatment, self-medication, family history, avoidance behavior (14 items)</td>
<td>0.65-0.72</td>
<td>Kappa, Spearman</td>
</tr>
<tr>
<td>Average duration, anxiety, number of symptoms</td>
<td>0.70-0.76</td>
<td>Spearman, Pearson</td>
</tr>
</tbody>
</table>
pooled across both studies only 4 out of 42 PAQ nonpanickers met SCID criteria for panic. Overall rates of agreement were as low as 74% and 65% (kappa: 0.50 and 0.32, studies 1 and 2, respectively). A post hoc analysis of those subjects who indicated panic attacks on the PAQ, but did not meet SCID criteria, revealed that disagreement was not of a pure “chance” nature. Rather, it seemed that these false positives reported milder variants of the same phenomenon (cf. the concept of limited symptom attacks in DSM-III-R) and that the interview had a more conservative cut-off between panic and non-panic.

The comparison of nonclinical panickers (PAQ and SCID criteria) and controls on the questionnaire battery yielded a number of pronounced differences. Since study 2 (Tübingen) used a more comprehensive battery, the pattern of its results is shown in Fig. 2. The results of study 1 were generally similar. The questionnaires used in study 2 were the Panic and Agoraphobia Profile (PAP, cf. Margraf and Ehlers 1987), Fear Survey Schedule (FSS, Arrindell 1980), Symptom Checklist-90 (SCL, Derogatis 1977), Self-report Inventory of somatic symptoms (SISS, King et al. 1986), state-trait anxiety inventory (STAI, Spielberger et al. 1970, trait form), Beck Depression Inventory (BDI, Beck et al. 1981), and the Mobility Inventory (MI, Chambliss et al. 1985).

The different questionnaires use very different scales. For a standardized presentation, we computed the difference between the means of panickers and controls divided by the standard deviation of the control group. The bars in Fig. 2 thus indicate the difference between the two groups in units of the standard deviation of the controls. The upper part of Fig. 2 shows those scales on which the two groups differed significantly ($P < 0.05$), the lower part scales without significant differences. It is important to note that questionnaires measuring similar constructs also yielded similar results. Therefore, such scales were grouped together.

Nonclinical panickers reported considerably higher levels of phobophobia, agoraphobic fears (but not avoidance behavior), somatization, anxiousness, depression, and injury phobia than nonpanickers of comparable age, sex, and socioeconomic background. Of the two depression scales, the BDI that focuses more on
Fig. 2: Comparison of paniciners and controls using a questionnaire battery (study 2, Tübingen). The bars represent the difference between the scores of the two groups divided by the standard deviation of the control group. Scales measuring similar constructs are grouped together. The abbreviations referring to the questionnaires containing the scales are explained in the text. The upper half of the figure shows scales yielding significant differences between paniciners and controls: phobophobia (PAP), agoraphobic fears (FSS), phobic anxiety (SCL), total somatization disorder score (SISS), somatization (SCL), neurotic symptoms (SISS), anxiety (SCL), trait anxiety (STAI), depression (BDI), depression (SCL), psychoticism (SCL), and blood/injury phobia (FSS). The scales listed in the lower half of the figure yielded no significant differences between paniciners and controls: mobility alone and mobility accompanied (MI), general avoidance (PAP), social fears (FSS), interpersonal sensitivity (SCL), fear of social embarrassment (PAP), aggression (FSS), hostility (SCL), separation anxiety (PAP), obsessive-compulsiveness and paranoid ideation (SCL), animal phobia (FSS), cardiovascular, gastrointestinal, and muscular awareness (SISS), fear of loss of control and fear of somatic distress (PAP).
the cognitive concomitants of depression yielded a stronger difference than the depression scale of the SCL-90 that contains more vegetative symptoms. The difference on the SCL-90 psychoticism scale is probably due to several ambiguous items that can be interpreted as signs of psychotic ideation as well as indicating typical panic symptoms (e.g., a fear of going crazy, losing control over one's body, derealization). Somewhat surprisingly, there were no differences in terms of self-reported avoidance behavior, social anxiety, hostility, or aggression. Separation anxiety, which has been linked causally to the development of panic attacks (Klein 1980; cf. Margraf et al. 1986b for a critique), was not heightened in nonclinical panickers. It should be noted that the separation anxiety scale used here has been shown to be highly sensitive to the separation anxiety found in clinical panickers as well as in agoraphobics (Margraf and Ehlers 1987).

The results for the baseline and hyperventilation tasks of the psychophysiological laboratory assessment are summarized in Fig. 3 (study 1) and 4 (study 2). We chose hyperventilation (60 cycles/min, 2 min) as the stress task because it has frequently been associated with panic attacks. Separate repeated measures ANOVA's (using the Greenhouse-Geisser correction when appropriate) for the different dependent variables showed significant baseline differences between panickers and controls in self-reported anxiety and panic symptoms, but not control symptoms which are not usually associated with anxiety, heart rate, systolic blood pressure, and diastolic blood pressure. Blood pressure results are not included in the figures. The responses to the hyperventilation task were similar in both groups with the exception of a greater increase in self-rated anxiety in panickers. In study 2 (Tübingen) the EKG was monitored continuously throughout the different paradigms and a rather strong heart rate increase in response to hyperventilation was observed. This was not the case in study 1 (Marburg) because heart rate could not be measured during but only before and after paradigms.

Overall, the results of the Marburg and Tübingen studies replicate earlier findings: There is a high number of persons with panic attacks in nonclinical samples. These persons also show more self-reported psychopathology, but not the cardiovascular differences typical for clinical cases of panic disorder. These replications are complemented by data on the reliability and validity of the questionnaire screening method and results from a more comprehensive battery of questionnaires. In addition, hyperventilation was again shown to produce increases in anxiety, panic symptoms, and heart rate. Nonclinical panickers showed higher baseline anxiety and a greater response to hyperventilation on the anxiety rating scale than nonpanickers.

Conclusions

Taken together, published studies of nonclinical or infrequent panickers and our own preliminary results suggest that Norton et al. (1985, 1986) identified a valid phenomenon. Panic attacks occur relatively frequently in nonclinical subjects. As in clinical studies, the exact proportion depends in part on the measures or criteria we use to determine panic attacks. In our studies, at least 50% of questionnaire-determined panickers did not meet SCID criteria for panic attacks. This occurred in spite of the fact that the subjects invited for the interview had not only indicated a panic attack but also reported on the PAQ at least one spontaneous attack, at least four symptoms during attacks, and panic attacks not only in social situations. Thus, the proportions of panickers given in Table 3 are probably upper limits of the prevalence of panic attacks.
attacks in nonclinical subjects. In spite of the high number of "false positive" results, the low proportion of "false negative" results and its good retest reliability make the PAQ a valid screening device. However, if one wants to assure compatibility with the diagnostic standards in clinical studies, a structured interview has to complement the questionnaire in its present form. Nevertheless, it may be
possible to develop future forms of the PAQ that agree better with instruments such as the SCID. It is also possible that the low agreement was due to the lack of reliability of certain criteria for the diagnosis of panic in DSM-III-R. The fact that information about the “spontaneity” and the rapidity of onset of panic attacks was not given reliably raises doubt as to the usefulness of these criteria.
What are nonclinical panickers like? There are a number of variables in the self-report domain that differentiate nonclinical panickers from controls. These are primarily measures of phobophobia, agoraphobic fears, somatization, anxiousness, and depression. The physiological variables assessed so far as well as reactivity to laboratory stressors differentiate much less well or not at all between panickers and controls in nonclinical samples. The most consistent difference found in our laboratory assessments were tonically elevated levels of self-reported anxiety and symptoms. Reactivity to stress tasks differentiated only poorly and cardiovascular measures differentiated not at all in our studies and in that of Sandler et al. (1987). Even the significant differences on laboratory parameters reach a magnitude of only about one standard deviation (of the control group) and are thus much smaller than some of the questionnaire differences.

Several of the features of nonclinical panickers have previously been found in clinical panic disorder patients (e.g., phobophobia, somatization, general anxiousness, depression). However, further studies are needed that directly compare nonclinical and clinical samples. It is an open question whether the infrequent panickers studied by Beck and Scott (1987) represent a clinical or a nonclinical population since all subjects were recruited through media announcements for people with panic attacks. In our experience, infrequent panickers who respond to such advertisements are more similar to self-selected clinical cases than to nonclinical subjects recruited from community screenings. This could be one reason for the lack of differences between the two samples reported by Beck and Scott (1987).

The results of studies of nonclinical panickers are consistent with the psycho-physiological, cognitive, or psychological models of panic proposed by several researchers (e.g., Barlow 1986; Clark 1986; Margraf et al. 1986a, 1986b; van den Hout, 1988) in showing a number of postulated causal factors for the development of panic (e.g., fear of anxiety symptoms, anxiety response to hyperventilation) to be present in this population. They are not consistent with views that assume separation anxiety or active avoidance behavior as necessary antecedents of panic attacks.

If we want to use these results to make more causal statements about the development of panic attacks, we need prospective longitudinal studies. These studies have to determine whether infrequent panickers are the basic population out of which some subjects go on to develop the full-blown clinical picture of panic disorder or even agoraphobia with panic attacks or whether the phenomenon of panic is heterogeneous, representing different subgroups of underlying causes. Either possibility is of high scientific and clinical interest. In the first case, we have a fascinating opportunity to study possible vulnerability factors in subjects at a high risk to develop panic disorder. In the second case, we may gain insights into differential etiologies of panic attacks possibly connected to clinical outcome in the long run. In addition, the longitudinal study of nonclinical panickers may give us information about possible factors protecting most of them from becoming clinical “cases”. We have recently started such a prospective longitudinal follow-up study of infrequent panickers at the Clinical Research Unit of the Department of Psychology at Philipps University. On the whole, the studies presented in this chapter illustrate the usefulness of supplementing the usual study of clinical samples by investigating of panic attacks in nonclinical subjects.

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