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THE PSYCHOBIOLOGICAL CORRELATES OF PANIC ATTACKS DURING *IN VIVO* EXPOSURE

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Abstract. This study examined in detail the psychobiological correlates of panic attacks experienced in panic disorder with agoraphobia. The cognitions, affect, and physiology of the six patients were monitored during *in vivo* exposure to their phobic situations. The results from these case studies showed that catastrophic cognitions are the key component of panic attacks. However, there was no clear-cut evidence to support an interaction among cognitions, affect, and physiology, which has been postulated by cognitive theories to be the central component of panic attacks and panic disorder with agoraphobia.

Keywords: Panic attacks, psychobiological, *in vivo*, exposure, panic disorder, agoraphobia.

Introduction

In the latest version of the *Diagnostic and statistical manual of mental disorders IV* (DSM-IV; American Psychiatric Association, 1994) panic attacks are defined as a discrete period of intense fear or discomfort accompanied by at least 4 symptoms out of a list of 13. The list includes symptoms such as palpitations, sweating, and fear of losing control. The panic attack has a sudden onset and usually peaks within 10 minutes or less. Thus, as defined, panic is a rather ubiquitous phenomenon found in most of the anxiety disorders. However, it is a key feature of panic disorder without agoraphobia (PD) and panic disorder with agoraphobia (PDA).

In recent years, panic attacks have been a focus of behavioural research (Barlow, 1988). One purpose of investigating panic attacks was to increase the understanding of PD and PDA. Initially influenced by the biological models (Klein, 1981; Sheehan & Sheehan, 1983), researchers studied panic attacks to examine and quantify their physiological correlates (Freedman, Ianni, Ettedgui, & Puthezhath, 1985; Margraf, Taylor, Ehlers, Roth, & Agras, 1987; Shear et al., 1992). These investigations were unable to

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provide empirical evidence of the biological markers of PD or PDA. Other possible explanations of this phenomenon were therefore explored, leading to the emergence of cognitive models (Beck, Emery, & Greenberg, 1985; Clark, 1988; Rapee, 1993; Salkovskis, 1988).

Cognitive models highlight the importance of cognitions in the occurrence of panic attacks (Khawaja & Oei, 1998a). According to cognitive theories (Beck et al., 1985; Clark, 1988; Rapee, 1993; Salkovskis, 1988), the physiological changes that initiate panic may occur as a result of an internal (e.g., physical effort, emotional responses, drug intake) or external (e.g., environmental stresses) trigger. These sensations, when interpreted as an indication of imminent danger, create anxiety (Khawaja & Oei, 1998a). This in turn exacerbates the somatic sensations and heightens the anticipation of threat, with the result that the spiralling anxiety develops into a panic attack. The cognitive process of attributing a threatening meaning to autonomic arousal and experiencing the resultant catastrophic cognitions is considered a vital characteristic of panic attacks (Zoellner, Craske, & Rapee, 1996). Catastrophic cognitions are defined as thoughts with the theme of danger or threat (Beck et al., 1985). Empirical evidence has demonstrated that these catastrophic cognitions are involved in panic attacks and PD (Khawaja & Oei, 1998a, b).

Although cognitive explanations emphasize the vital role of cognitive elements in the occurrence of panic attacks, it is important to note that other dimensions are also thought to be involved (for a review see Whittal, Goetsch, & Eifert, 1996). For example, physiological factors are also taken into account when evaluating a panic experience. Somatic sensations are regarded as a primary trigger of panic attacks and evidence of a link between autonomic arousal and panic attacks has been demonstrated by several researchers (e.g., Clark et al., 1988; Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988; Margraf, Barlow, Clark, & Telch, 1993; Margraf & Ehlers, 1989; Rachmann, Lopatka, & Levitt, 1988). Moreover, panic attacks are manifested by severe physical symptoms (Krystal, Woods, Hill, & Charney, 1991; Sanderson, Rapee, & Barlow, 1988). There is consensus among cognitive theories that PD and PDA patients experience panic by misperceiving a benign autonomic reaction as catastrophic (Beck et al., 1985; Clark, 1988). The affect experienced by the individual during a panic attack is also thought to be linked with the physiology and cognitions. Affect is reflected by the subjective distress felt by the person (Whittal et al., 1996). The hypothesized involvement of cognitions, physiology, and affect indicate the importance of investigating the relationship of PDA patients' cognitions with respect to their autonomic arousal and affect.

The relationship among the components of panic attacks (i.e., cognitions, affect, and physiology) has recently been examined using several procedures (Khawaja & Oei, 1998a). The majority of researchers have relied on retrospective methods. Unfortunately, retrospective reports can be biased (Beurs, Lange, & Dyck, 1992; Rapee, Craske, & Barlow, 1990). A more sophisticated method of assessing the various dimensions of panic was introduced by a group of researchers (Kenardy, Evans, & Oei, 1988; 1989; Kenardy, Oei, Weir, & Evans, 1993). These researchers investigated the relationship among PDA patients' cognitions, affect, and physiology by monitoring these responses as they occurred in the natural environment or in phobic situations. Physiology, in the form of heart rates, was monitored by microprocessor in epochs as brief as 5 seconds. Participants were asked to verbalize their thoughts and distress levels (affect) and these

were recorded by a microcassette recorder. In order to interpret the cognitions and to make them more meaningful, they were classified on a number of dimensions by an independent coder using the classification system developed by Last, Barlow and O'Brien (1985). In the case of PD/PDA these classifications are often made in terms of the presence or frequency of negative and/or catastrophic cognitions. The *in vivo* assessment of the ongoing responses increased the reality of the situation.

The initial studies shed light on the relationship among the different variables (i.e., cognitions, affect, and physiology) at the time of the panic attack and elevated anxiety. However, further investigation of this relationship phenomenon by Khawaja, Oei and Evans (1993) failed to show a significant relationship among these variables. It is important to note that the preliminary studies were not free from limitations. The findings were either based on limited data (Kenardy et al., 1988) or on data that was not collected continuously (Kenardy et al., 1989). Two of these studies (Kenardy et al., 1993; Khawaja et al., 1993) conducted statistical analyses on responses recorded during elevated anxiety. These analyses presented an overall global picture with reference to the high anxiety of the patients. However, elevated anxiety, compared to panic attacks, may not be an accurate reflection of PD or PDA. Furthermore, the absence of any significant relationship in the study by Khawaja et al. (1993) may have been due to methodological problems, because the cognitions reported by the patients were classified by an independent normal coder. Further examination of this methodology indicated that the normal coder may not be accurate in reflecting the perspective of the patients who reported the cognitions (Oei & Khawaja, 1999). In view of this finding, it was suggested that the patients who report the cognitions should be involved in the process of coding.

The present research was designed to investigate the components of PD or PDA by addressing the shortcomings of the previous studies. First, the relationship among the variables postulated to be important by the cognitive models of PD or PDA has not been studied extensively by the monitoring of ongoing panic responses recorded during the time the panicker is exposed to the phobic situation. Second, it is important to investigate the interaction among the components of PD or PDA by focusing on panic attacks. The aim of the present study was therefore to examine the relationship among the cognitions, physiology, and affect of the PDA patients by concentrating on their detailed responses during panic attacks. Detailed observations of panic are considered a useful method of collecting meaningful data about the characteristics of panic attack (Whittal et al., 1996). Patients' responses were monitored using cognitive sampling and ambulatory monitoring during *in vivo* exposure to the phobic settings. These procedures have been shown to be useful in collecting the spontaneous data (Margraf, 1990; Kenardy et al., 1989; Turnip, 1990). To increase the interpretability of the information about the inter-relationship of the components of panic attacks, the data for each patient were plotted graphically and examined visually. Finally, because patients who reported the cognitions have been shown to be better at coding their own data (Oei & Khawaja, 1999), the psychobiological correlates of panic attacks were investigated by incorporating these recent developments into the coding system. Based on previous research, it was expected that panic attacks would be characterized by catastrophic cognitions, elevated affect, and physiology. Thus, cognitions, physiology and affect were expected to be interrelated during panic attacks.

Method

Participants

Six patients meeting the DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria for panic disorder with agoraphobia (300.21) participated in the study. The patients reported experiencing between 1 and 3 panic attacks per week. All participants were housewives with secondary education. Their mean age was 43.33 years (SD = 11.63 years; range = 29 to 57 years). The mean duration of their illness was 16.5 years (SD = 11.55 years; range = 1 to 32 years). The participants were not receiving any treatment at the time of the study.

Measures

Ambulatory monitoring. Ambulatory monitoring was recorded using the Vitalog PMS-8 (Vitalog Corp., 1982). The Vitalog is an ambulatory microprocessor that records heart rate and activity level when worn by the participant (in epochs as brief as 5 seconds). Physical activity ranges from 0 (no movement) to 6 (walking at a fast pace). The event marker of this device is a button, which can be pressed to record any panic attack onset. The Vitalog is a small and portable device that is carried in a pouch on the waist. These procedures were similar to those reported in the earlier studies (e.g., Khawaja et al., 1993).

Cognitive sampling. The spontaneous cognitions of particpants were sampled every minute, cued by a two second repeated tone generated by a timing watch. Cognitions occurring immediately prior to the tone cue were to be reported by the participant. After the first cue, following a 10 second interval, there was a second cue on which participants were to report their subjective distress level (SUD) on a scale from 0–10. A microrecorder recorded participants' cognitions and subjective distress level throughout the exposure. These procedures were similar to those used in the earlier studies (Kenardy et al., 1989; Khawaja et al., 1993).

Procedure

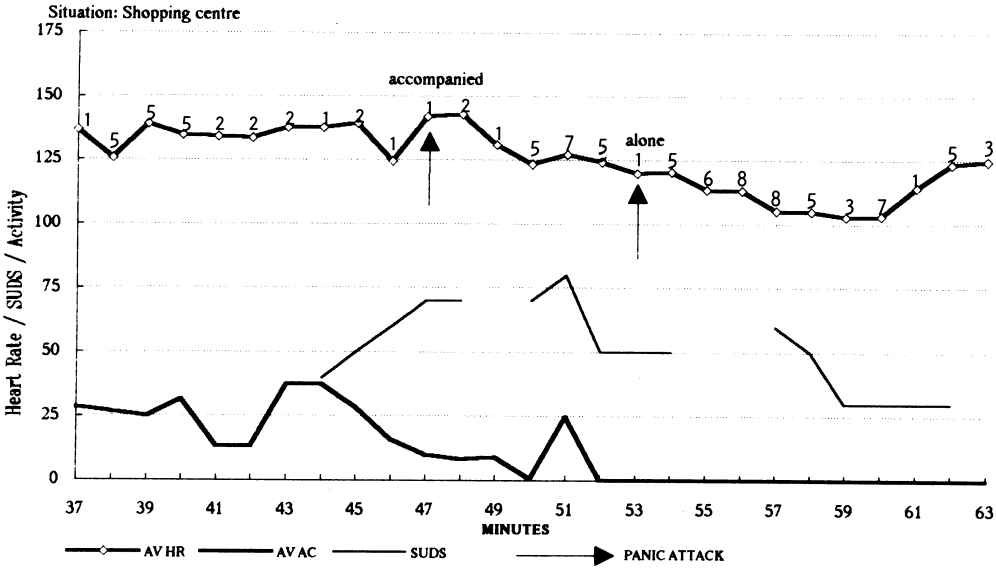
Patients reporting panic attacks were selected for the present study. The selection and experimental procedures are described in detail in Khawaja et al., 1993. Briefly, participants were selected from the two clinics associated with the University of Queensland: the Anxiety Disorder Clinic and the Psychology Clinic. The patients from the Anxiety Disorder Clinic were referred by health practitioners. These patients went through routine pre-assessment in the clinic. A psychologist or a psychiatrist interviewed the patients using a semi-structured interview according to the DSM-III-R. The duration of the interview was approximately one hour. These procedures are similar to those outlined in previous studies (Kenardy et al., 1989; Oei, Moylan, & Evans, 1991). Participants recruited from the Psychology Clinic responded to advertisements appearing in the local newspapers of the Brisbane metropolitan area. Those who appeared to be suitable, on the basis of a screening interview conducted by telephone, were mailed a package containing brief information about the study, a consent form, and a set of

questionnaires. They were asked to bring the completed questionnaires to the Psychology Clinic, University of Queensland. On their visit to the clinic, the first author on the basis of SCID-NP interviewed them. Participants were informed about the nature of the study and it was emphasized that they were free to discontinue at any time, without prejudice.

The selected participants reported to the Clinic on the day of the experiment. The procedure was explained in detail and all queries were answered (see Khawaja et al., 1993, for a full description of the procedure). Prior to the exposure, they were provided with training in reporting cognitions and subjective distress levels. They were further instructed to record any panic attack onset by pressing the event marker, or reporting aloud. Panic attacks were explained to the subjects according to the DSM-III-R criteria. After satisfactory training, a baseline recording was done for a period of 10 minutes. During this phase, each participant was asked to report her cognitions and subjective distress levels, first while sitting alone in a clinic room for 5 minutes, and then walking around the clinic at a normal pace with the experimenter for another 5 minutes. Heart rates and activity were automatically being recorded. Subsequently, assessment was conducted during a period of exposure to situations of self-reported high anxiety and avoidance. The first author accompanied participants to their feared situations. They were then asked to remain on their own in the *in vivo* situation for approximately 15 to 20 minutes. Participants were then debriefed at the end of the exposure. The total duration of the experiment was approximately 70 minutes. A second appointment, within the next 3–5 days, was arranged with the participant. During this interval the tapes with the participants' recorded cognitions were transcribed. On the second visit, participants were asked to classify the cognitions they had reported during the exposure, using the simplified version of the Last and colleagues' classification system (Last et al., 1985). This simplified form is fully described elsewhere (Oei & Khawaja, 1999). Participants were instructed to categorize their own thoughts, which they had reported when taken out to a situation that was anxiety provoking for them. It was further stated in the instructions that their thoughts could be classified into different types on the basis of the categories listed (i.e., catastrophic, negative affect, escape, approach, coping, positive affect, descriptive, and irrelevant). Finally, subjects were offered group cognitive behaviour therapy at the end of the study.

Results

Seven panic attacks were reported by the six participants. One participant (Subject 1) experienced a panic attack twice during the exposure phase. In order to study the cognitive, affective, and physiological correlates of panic attacks in detail, the data were examined visually. Responses 10 minutes prior to, until 10 minutes after, the reported onset of the panic attacks were selected. As panic attacks have a sudden onset and generally last for a few minutes, this duration was considered a reasonable time to investigate the attacks. Figures 1 to 6 present responses of the six subjects as they occurred during the selected time period. These responses consist of cognitions, heart rate, SUDS, and physical activity. Heart rate and SUDS, which were recorded every 5 seconds, were averaged across each minute. Physical activity, which originally ranged from 0–6, was multiplied by 10 in order to prepare the graphs. The averaged heart



Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 1. Panic attacks of Subject 1

rates and physical activity per minute were plotted in the graph along with the coinciding cognition and SUD. The graphs also show the situations in which the patients experienced the panic attacks. These situations were extremely fearful for the participants as compared with the other phases of the exposure.

As can be seen from the Figures, six out of the seven panic attacks were experienced when the patients were alone. The first panic attack of Subject 1 occurred whilst accompanied by the experimenter. Cognitions coinciding with the onset of panic attacks had a catastrophic content (see Figures 1 to 6). As shown by the graphs, the thoughts reported before and after the panic attacks were generally of a negative type (e.g., catastrophic, negative affect, and escape). Inspection of the data in Figures 1 to 6 indicates that positive (coping and approach) and neutral (description and irrelevant) cognitions were also occasionally reported.

The data reveal that the distress levels were not reported consistently by the participants. SUDS were moderate at the time of four of the panic attakcs (Figures 1, 2, 3, and 5). High SUDS (7 or above on the scale of 1 to 10) were reported by two participants (Figures 1 and 6). One participant did not report her subjective anxiety levels (see Figure 4). There was no fluctuation in the heart rate at the time of five panic attacks (Figures 1, 2, 3, 5, and 6). In the case of Subject 4, there was a sudden increase in the heart rate one minute before the onset of the panic attack (see Figure 4). A sharp acceleration in the heart rate occurred only in the first panic attack of Subject 1 (see Figure 1). Physical activity was either absent (Figures 1, 3, and 5) or minimal (Figures

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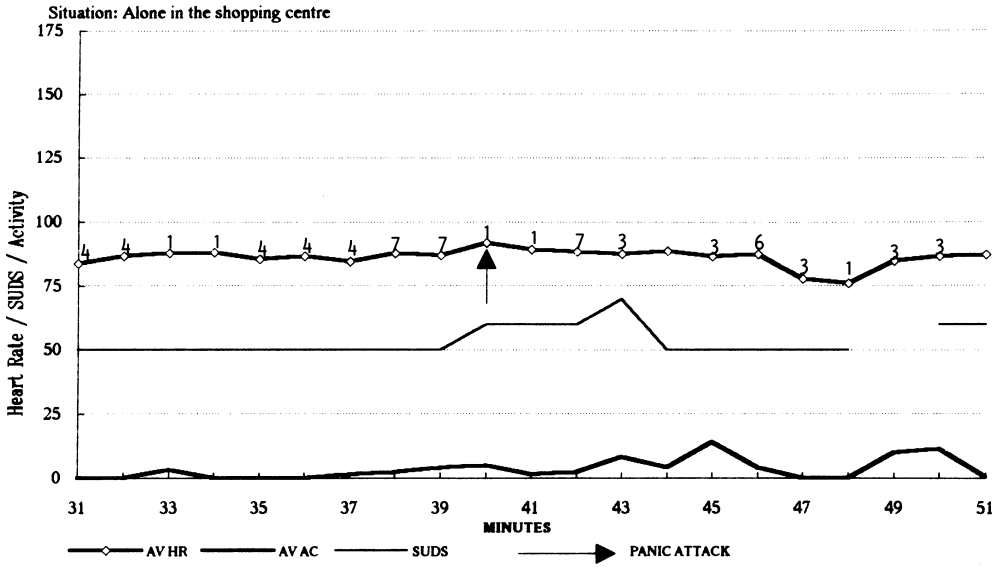

Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 2. Panic attacks of Subject 2

1, 2, and 6) at the time of six of the panic attacks. The first participant (see Figure 1) was walking slowly in the shopping centre before reporting a panic attack. The fourth participant (see Figure 4) walked for a few minutes towards the bus stop before reporting the panic attack.

Concurrent relationships

Overall, the data show that only one panic attack, the first panic attack of Subject 1 (see Figure 1), was marked by a catastrophic cognition, high subjective distress, and an elevated heart rate without an impact of physical activity. The panic attack experienced by Subject 4 was marked by a catastrophic cognition along with an increased heart rate one minute before the onset of the attack (see Figure 4). However, the heart rate of this participant may have been confounded by physical activity. Catastrophic cognitions and moderate SUDS were the main features of the remaining panic attacks (Figures 1, 2, 3, 5, and 6).

Discussion

The present study presents an in-depth analysis of panic attacks as they occurred during *in vivo* exposure using ambulatory monitoring and cognitive sampling. Graphic presentation of the data highlights the vital features of the panic attacks. The majority (85%)

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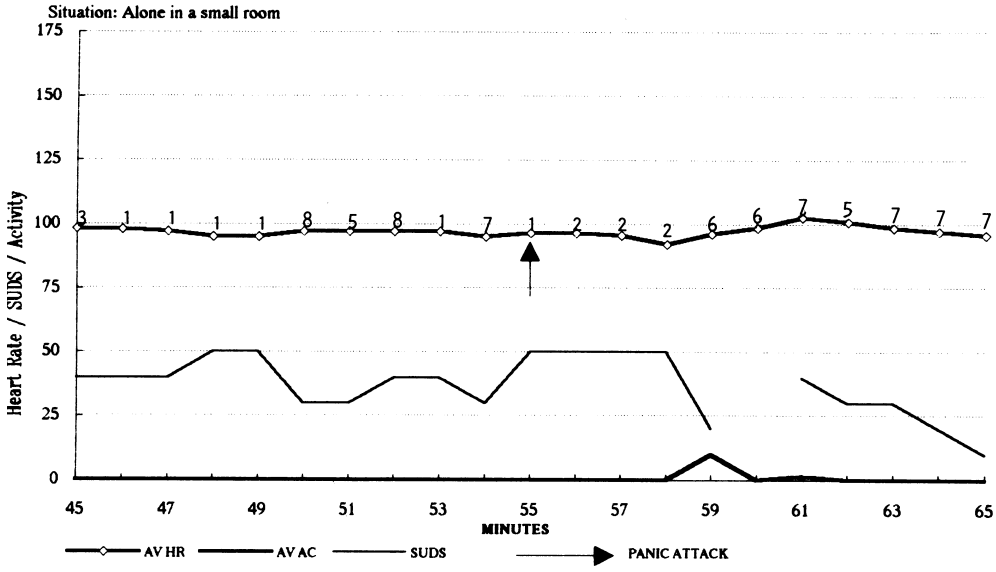
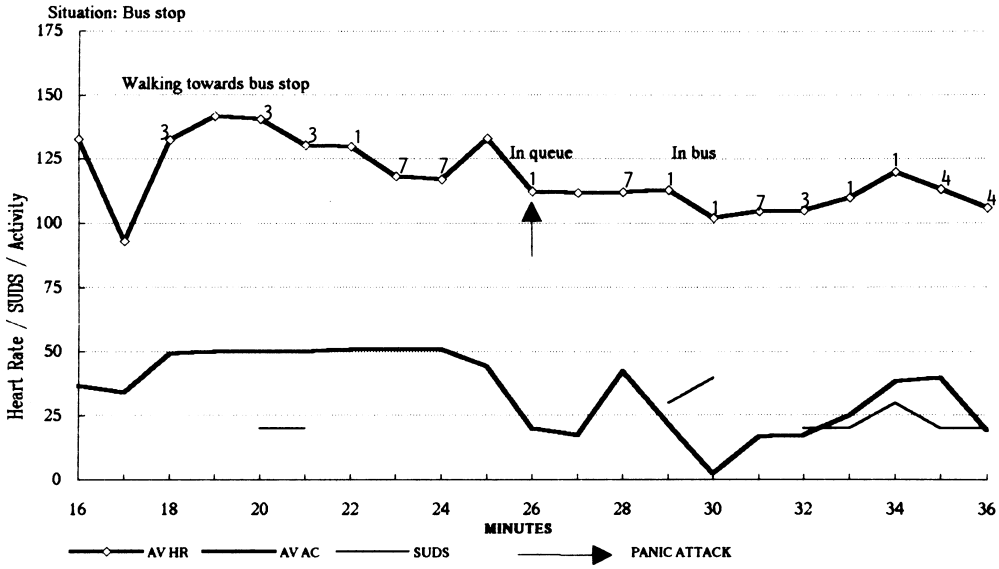


Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 3. Panic attacks of Subject 3

of the attacks occurred after the researcher had left the patients alone in the phobic situations. Apparently, the aversiveness of the environment increased when patients were on their own and without a safety figure (Rachmann, 1984). This may have increased their attention to the potential dangers of the situations and/or to their fearful bodily sensations (Borden, Lowenbruan, Wolff, & Jones, 1993). A lack of perceived control over these internal and/or external-threatening events may have aggravated their autonomic arousal (Rapee, Craske, Brown, & Barlow, 1996). Anticipation of the panic and an exaggerated appraisal of the negative consequences of anxiety may thus have escalated into a panic attack (Telch, Brouillard, Telch, Agras, & Taylor, 1989). This is consistent with the outcome of recent studies that have indicated that absence of the safe person led to a marked increase in anxiety, fear, heart rate, panic, and catastrophic cognitions in PDA patients (Carter, Hollon, Carson, & Shelton, 1995; Rapee, Telfer, & Barlow, 1991).

Cognitions were revealed as the prominent feature of panic attacks. Ideation reported at the time of the panic attack had a catastrophic content, reflecting the patients' tendency to misinterpret internal and external cues as dangerous and likely to lead to a disaster (Beck, 1988; Clark, 1988; Westling & Ost, 1993). This finding is consistent with the cognitive theories of PD/PDA (for reviews see McNally, 1994; Taylor, 1995; Khawaja & Oei, 1998). Furthermore, the attack onsets were generally preceded, and followed by, negative cognitions with a theme of danger, unpleasant mood, or escape from the *in vivo* situation. This indicates that anxiety was marked immediately before



Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 4. Panic attacks of Subject 4

and after the panic attacks. The content of the negative cognitions shed light on the physical symptoms and the unpleasant emotions experienced by patients (Marks, Basoglu, Alkubaisy, Sengun, & Marks, 1991). For example, cognitions with a content of escape were suggestive of participants' compulsion to leave the *in vivo* situation. This finding is consistent with a previous study by Genest, Bowen and Dudley (1990), who hypothesized that escape or avoidance may be linked to high levels of anxiety. Barlow and colleagues (Barlow, Brown, & Craske, 1994) have described the "urge to escape" as an important feature of panic, which prepares the organism for action. However, because it is a subjective experience, it is expressed through self-report (see Whittal et al., 1996, for a discussion of the limitations of this method). Participants in the present study also reported neutral or positive cognitions, which acted as coping mechanisms, either by reassuring or distracting the person. Coping mechanisms have been shown to be associated with negative ideation and elevated anxiety (Stoler & McNally, 1991).

The subjective distress of the patients was generally moderate at the time of most (85%) of the panic attacks. This finding indicates that the patients experienced anxiety and emotional responses at the time of these attacks (cf. Barlow, 1988; Ley, 1992). However, it appears that like the other self-report procedures, SUDS were reported in an inconsistent manner. This was one of the measures, where there was a substantial amount of data missing. It seems that the subjects' major emphasis was on reporting the cognitions and they probably forgot to report the SUDS. Distress levels reported within the moderate range could be due to the fact that the subjects were engrossed

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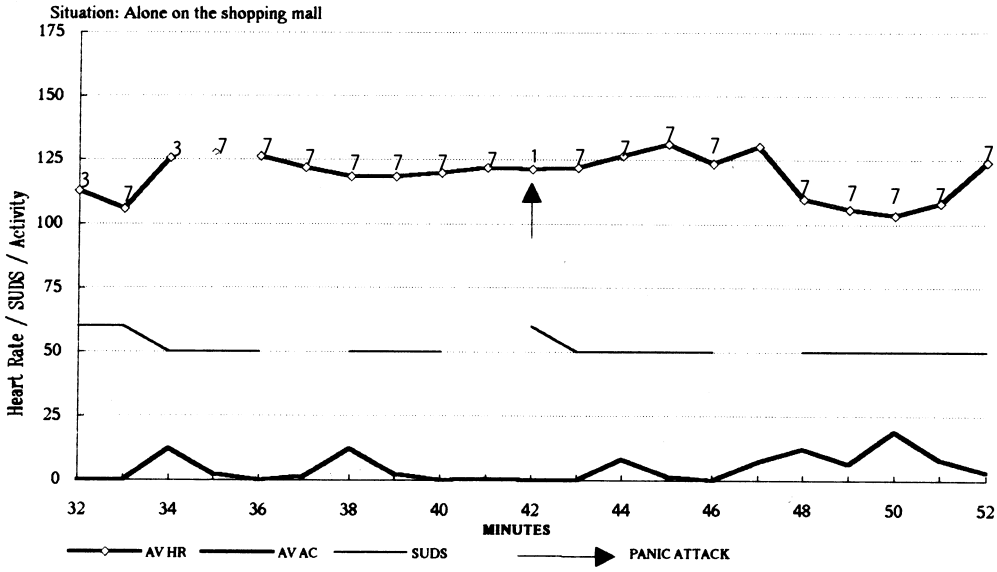

Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 5. Panic attacks of Subject 5

with the task of reporting cognitions and reported the SUDS inattentively. It is also important to note that the ambulatory monitoring procedures do not point out the exact time at which the SUDS were reported. There is no certainty that they reported immediately after the cue. It is possible that they reported them after a delay when they experienced a decrease in their subjective distress.

Limited information about autonomic arousal (in the form of fluctuating heart rate) was provided by the present study. There was little evidence of an elevated heart rate at the time of the panic attacks. Most of the panic attacks (71%) were not characterized by fluctuations in heart rates. The absence of variability in the heart rate could be due to one of several factors. First, it is important to note that not all panic attacks are marked by sudden changes in heart rate. Previous findings have shown that increases in heart rate occurred only in a subgroup (50%) of all panic attacks (Margraf et al., 1987; Taylor et al., 1986). In those studies, not all monitored panic attacks were associated with heart rate changes. Therefore, absence of increased cardio-vascular activity at the time of a panic attack is consistent with previous findings based on panic induction (Ehlers, Margraf, & Roth, 1988; Ehlers, Margraf, Taylor, & Roth, 1988) and physiological ambulatory monitoring procedures (Shear et al., 1992).

Second, fluctuation in heart rate is only one of the physical symptoms of panic attacks. Patients who reported panic attacks may have responded with symptoms other than those of a cardio-vascular nature and these symptoms were not monitored (Light, 1990). A variety of physiological responses could not be measured due to equipment

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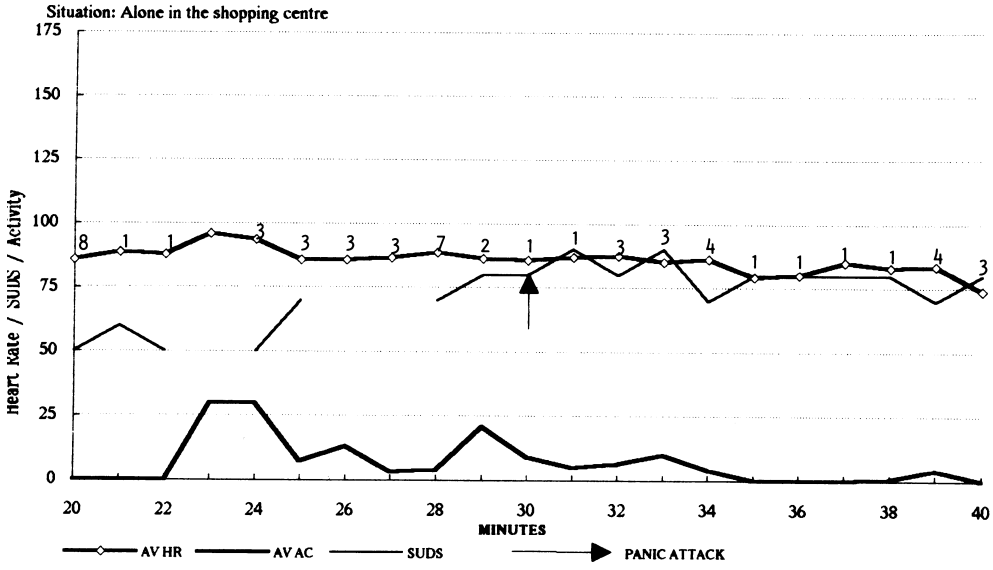

Note: AV HR = average bpm; AV AC = average activity level per minute; SUDS = subjective distress level per minute; activity and SUDS are multiplied by 10; cognitions: 1 = catastrophic, 2 = escape, 3 = negative affect, 4 = coping, 5 = approach, 6 = positive affect, 7 = description, 8 = irrelevant; Gaps in cognitions and curves showing AV HR, AV AC and SUDS = missing data/ no response

Figure 6. Panic attacks of Subject 6

limitations. Vitalog stores data regarding heart rate, physical activity, and event markers. However, it does not have the capacity to store more than three modalities. Therefore, information about other physiological correlates could not be obtained using the Vitalog equipment.

Third, it is possible that the crucial feature of panic attacks is the perception of sensations, which need not be highly correlated with actual physiological changes (Margraf & Ehlers, 1989). Patients might have experienced subtle physical changes that were not detected by the cardiovascular measure. Furthermore, there is substantial empirical evidence that patients allocate attentional resources to their internal milieu (Cloitre, Heimberg, Holt, & Liebowitz, 1992; McNally, Riemann, Louro, Lukach, & Kim, 1992), suggesting they might have noticed the bodily changes even when these were subtle (Ehlers, 1993; Rapee, 1994). Thus, perception and catastrophic misinterpretation of subtle bodily changes (i.e., not detected by the cardiovascular measure) may be a significant feature of panic attacks.

Fourth, it is important to note that heart rates were recorded after 5 seconds. In order to manage the data the heart rates recorded during one minute were averaged to obtain one value for each minute. This value was then compared with the other responses occurring during that minute. It is possible that within each minute there were minor fluctuations in the heart rate, which were lost as a result of converting a number of recordings into one average value.

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Concurrent relationships among cognitions, affect, and physiology were not often observed in the present data. A moderate association between cognitions and SUDS was found. This is in line with previous findings (Michelson, 1988). However, only marginal links of heart rates with SUDS and cognitions were apparent. It is important to note that the monitoring procedures may not be sensitive enough to evaluate the relationships among the different variables because the timing intervals used may be too coarse to reveal such associations. For example, it is difficult to know if the cognitions were reported at the time indicated or if they were concurrent with the subjective or physiological responses recorded at that time. The exact time at which a cognition of SUD is reported is not evident, making it difficult to link them to their concurrent heart rate. It is also difficult to determine which cognition or SUD is affecting which heart rate and vice versa. Furthermore, it is difficult to determine the extent to which heart rates are confounded by other factors such as physical activity or metabolism. In view of these issues, it appears that the relationships among cognitions, affect, and physiology cannot be effectively investigated by employing ambulatory monitoring and cognitive sampling in their current forms. However, the present research has demonstrated the utility of these methods in the assessment of ongoing cognitions, subjective distress, and heart rate responses in anxiety or panic as they occur during *in vivo* exposure.

In summary, the present study demonstrates that catastrophic cognitions are the most important attribute of panic attacks. This finding indicates the importance of catastrophic cognitions in PD and PDA. The present results also suggest that SUDS may play a secondary role in panic attacks and that there is some overlap of SUDS with cognitions. The influence of physiology, in the form of heart rate fluctuations, in the panic attacks was minor. However, this may have been due to the limitations of the monitoring equipment. The Vitalog is confined to data that can be evaluated separately and it fails to provide information about the synchronization among different types of responses. In conclusion, catastrophic cognitions appear to be the primary feature of a panic attack. The use of *in vivo* cognitive assessment in the present research showed it is a valuable procedure for investigating these ongoing cognitions. Furthermore, involving the patient reporting the cognitions in the process of classifying the cognitions enhanced the methodology accuracy. Nevertheless, the results of the present study should be considered with some caution as they are based on a relatively small sample of panic attacks.

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