Panic Attacks , Anxiety Sensitivity and Cardiac Awareness

by

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Abstract

Psychophysiological models of panic attacks postulate a positive feedback loop between internal anxiety cues and cognitive reactions to these cues. It is well documented that panic patients respond anxiously to arousal symptoms, attend to physically threatening cues such as somatic sensations, and interpret those cues as indicative of impending personal danger. Several studies have also indicated that panickers have heightened autonomic arousal. This profile suggests that panic patients may possess an enhanced awareness of visceral sensations. Preliminary investigations have provided mixed support for such speculation. The purpose of this study was to investigate the relationship between panic history, anxiety sensitivity, and cardiac awareness. Forty panickers and 40 non-panickers, subdivided into equal groups of high and low anxiety sensitive males and females, were given 60 trials of the Whitehead heartbeat discrimination procedure. Thirty trials were given during rest and 30 following a biological challenge (i.e., hyperventilation) designed to increase physiological arousal. Analysis of variance results indicated that cardiac awareness during rest, while not significantly greater than chance (i.e., > 64% correct discriminations), was superior to awareness following hyperventilation. During resting discriminations, low anxiety sensitive male nonpanickers and high anxiety sensitive male panickers were more accurate at perceiving heartbeats than any other group; however, only the former performed significantly better than chance. Discriminant function analyses revealed that

panic attacks and other indices of anxiety were not good predictors of accurate and inaccurate heartbeat perceivers. These results are discussed in terms of their implications for psychophysiological models of panic. Also, relevance to the general area of cardiac awareness is addressed.

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Recently there has been an increased interest in the phenomenology and psychophysiology of panic attacks. According to the Diagnostic and Statistical Manual of the American Psychiatric Association (APA), Third Edition-Revised (APA, 1987), a panic attack involves feelings of apprehension and impending doom which are of sudden onset and associated with an array of symptoms. In order to meet DSM-III-R criteria for panic disorder an individual: a) must have experienced one or more discrete episodes of unexpected panic that were not triggered solely by exposure to phobic situations, b) must have experienced a minimum of four attacks in a 4-week period or one attack followed by one month of persistent fear of having another attack, and c) must have experienced a minimum of four of the recognized symptoms during one of the attacks (APA, 1987)[1]. Simply stated, the panic attack is a distinct psychophysiological event involving distressful somatic symptoms in the cardiovascular, respiratory, and musculoskeletal systems, which are usually accompanied by catastrophic cognitions and a high level of fear.

Panic disorder is relatively common within the general population. Recent estimates of prevalence suggest that approximately one to eight percent of the population experience panic at a clinical level of severity (Karno et al., 1987; Robins et al., 1984; Salge, Beck, & Logan, 1988; Wittchen, 1986).

Recent evidence suggests, however, that panic attacks are not limited to those suffering from panic disorder. In fact, panic appears to be common among all anxiety disorders and to several other psychiatric disorders (Barlow et al., 1985). Moreover, it has been demonstrated that a large proportion of apparently healthy adults appear to experience panic at a frequency less than required to comprise clinical panic. Specifically, Norton and colleagues (Norton, Dorward, & Cox, 1986; Norton, Harrison, Hauch, & Rhodes, 1985) have indicated that 35% of university students reported having experienced at least one DSM-III panic attack in the year prior to the survey, and 25% reported experiencing one or more DSM-III attacks in the three weeks prior to the survey. More recently, Brown and Cash (1990) have found that 26.6% of college students reported having experienced at least one DSM-III-R panic attack in the previous year, and 13.8% reported one or more DSM-III-R attacks in the last month. Telch, Lucas, and Nelson (1989) reported that approximately 12% of their sample of college students reported at least one unexpected panic attack. Wilson, Sandler, Asmundson, Larsen, and Ediger (1991) reported that discriptions of a typical panic attack on DSM-III panic attack self-reports affect reported prevalence rates of situationally-bound panic attacks (i.e., attacks that occur in response to particular situations). With a written vignette describing a typical panic attack, the prevalence of reported situationally-bound panic attacks was 33.4%; without, the prevalence increased to 51.3%. On the other

hand, the proportion of subjects reporting unexpected (i.e., spontaneous or "out of the blue") attacks (6.7%) was comparable in both conditions. Collectively, these results suggest that panic attacks are a common occurrence in the general population.

Aside from frequency of attacks, the DSM-III-R includes persistent worrying over subsequent attacks as a criterion of panic disorder. To date no empirical investigations have compared panic patients to the general population with regard to this criterion.

Several studies suggest that nonclinical panickers are similar in many ways to panic disorder patients. For example, nonclinical panickers report many of the same symptoms as panic disorder patients, with an average of 8 of the 12 DSM-III symptoms (Norton et al., 1986). Panic disorder patients, however, report more symptoms and a greater severity of symptoms (Norton et al., 1986). Further, both nonclinical panickers and panic disorder patients are characterized by a tendency to interpret arousal as threatening (Cox, Endler, Norton, & Swinson, in press; Donnell & McNally, 1989). Consequently, a strategy that may be of value to the investigation of panic disorder is assessment of individuals who suffer from occasional panic attacks, but who have not developed the complete syndrome of panic disorder. Indeed, Telch et al. (1989) have stated that "study of the [occasional] panicker is likely to yield valuable information on the etiology of panic disorder" (p. 305). Presumably, such individuals would show evidence of any

biological and/or psychological vulnerability that precedes the onset of panic attacks, without the confounding influences of secondary symptoms.

Biological Factors Associated to Panic

Over the past decade it has been well documented that agoraphobia and panic disorder are characterized by a familial incidence suggestive of a genetic contribution (Cloninger, Martin, Clayton, & Guze, 1981; Crowe, Noyes, Pauls, & Slymen, 1983; Crowe, Pauls, Slymen, & Noyes, 1980; Harris, Noyes, Crowe, Dewal, & Chaudry, 1983; Moran & Andrews, 1985; Torgerson, 1983). In general, the theme emerging from these studies is that vulnerability may be inherited; however, expression of the disorder seems to be dependent on personal and environmental factors (Barlow, 1988). What remains undetermined is the exact nature of this vulnerability.

There is evidence to suggest that a diffuse biological reactivity may comprise the inherited vulnerability. Studies indicate that the reactivity may result from a labile sympathetic nervous system (SNS), or a hyperactive noradrenergic system.

Sympathetic Hyperactivity

Disturbances of SNS function have been implicated in the pathogenesis of anxiety disorders (Redmond, 1979). However, direct assessment of SNS functioning is not possible in humans. One approach that has been developed to indirectly measure functioning of this system is the measurement of plasma catecholamine levels. Investigations employing this technique

have demonstrated that elevations in plasma catecholamines (particularly norepinephrine [NE]) are associated with activation of the SNS (Goldstein & Kopin, 1988; Lake, Ziegler, & Kopin, 1981). Additionally, some of the symptoms of panic suggest sympathetic activation. Therefore, it seems reasonable to assume that catecholamine levels will be chronically higher in panic sufferers than controls. Evidence for this assumption is mixed.

Rubin (1984), in reviewing the neuroendocrine aspects of panic disorder, has concluded that panickers possess elevated levels of both epinephrine (E) and NE compared to controls. Although Rubin (1984) cites no empirical evidence to support this conclusion, it is tenable on other grounds. For example, Nesse, Cameron, Curtis, McCann, and Huber-Smith (1984) have reported elevated levels of E and NE in patients with panic disorder. Ballenger et al. (1984), however, found only that NE levels were elevated in their sample of agoraphobics when compared to controls: There were no significant differences between the groups in E levels. Similarly, Nesse et al. (1985) found a significant difference in only the levels of NE excreted by panickers compared to controls. Although these results are mixed, they do suggest the presence of a labile neurotransmitter system within the patients.

However, uncritical acceptance of neurotransmitter lability in panic disorder is unwarranted. Several studies have failed to replicate findings of elevated catecholamine levels in panic patients (Carr et al., 1986; Gaffney, Fenton, Lane, & Lake, 1988;

Liebowitz et al., 1984; Woods, Charney, McPherson, Gradman, & Heninger, 1987). In short, there has only been partial support for the role of a labile neurotransmitter system in the pathogenesis of panic (for a thorough review the reader is referred to Cameron & Nesse, 1988). Alternately, abnormally sensitive beta-adrenergic receptors have been hypothesized to be important in panic responses. Symptoms commonly experienced by panickers, such as tachycardia, palpitations, and dyspnea, suggest the possibility of SNS arousal and discharge being a critical underlying substrate of panic disorder. Ahlquist (1948) was the first to demonstrate that stimulation of the beta-adrenergic receptors resulted in increased heart rate, stronger cardiac contractions, vasodilation, and a number of other physiological changes. It was not long thereafter that researchers interested in panic disorder noticed the similarity between responses elicited in Ahlquist's experiment and panic attacks.

In order to investigate the role of beta-adrenergic receptors in panic disorder, researchers have attempted to stimulate beta receptor sites via the administration of pharmacological agents. It has been demonstrated that isoproterenol, a beta-agonist, causes episodes of panic in panic disorder patients, but not controls (Pohl et al., 1988; Rainey et al., 1984). Nevertheless, there remains some question as to the significance of hypersensitive beta-receptors to naturally occurring panic attacks.

Hyperactivity of the Noradrenergic System

Many of the symptoms of panic disorder appear to reflect central nervous system arousal. As a result there has been considerable conjecture as to the site of arousal. The most prominent candidate is the locus ceruleus (LC), a major nucleus of the noradrenergic system. The basis for considering a relationship between the LC and panic disorder came from a series of studies conducted by Redmond (1979). Redmond electrically and pharmacologically stimulated the LC of stump-tailed monkeys, finding that their response was very similar to that displayed by monkeys in situations of natural threat. The implication drawn was that the reaction of the stimulated monkeys may be analogous to panic episodes in humans.

Physiological investigation into the LC connection to human panic has occurred in the context of pharmacological infusions that increase LC activity. According to Andrade and Aghajanian (1984), drugs that have such an effect interact with the alpha2-adrenergic receptors of the LC, inhibit their function, and thereby increase LC activity (i.e., react via a negative feedback cycle). Yohimbine, an alpha2-receptor antagonist, has been found to provoke panic in panic patients but not controls (Charney, Heninger, & Brier, 1984; Uhde, Roy-Byrne, Vittone, Boulenger, & Post, 1985). These results suggest that increased LC activity may be related to panic disorder.

However, several investigators (Grunhaus, Gloger, Birmacher, Palmer, & Ben-David, 1983; Woods et al., 1987) suggest that more

research is needed before any firm conclusions regarding the role of noradrenergic hypersensitivity in panic disorder can be made.

Lactate Infusion and Panic

Infusion of sodium lactate, a beta agonist and the end metabolic product of glucose metabolism, is considered a useful tool in exploring possible etiological agents of panic. In fact, sodium lactate has been considered the best biological marker of panic (Carr & Sheehan, 1984). Yet, the mechanism by which lactate induces panic is not known (Insel et al., 1984; Margraf, Ehlers, & Roth, 1986; Rainey et al., 1984; Pitts & Allen, 1982). Even though changes in acid-base status (Gorman et al., 1989; Papp et al., 1989), serum-ionized calcium or phosphate (Fryer et al., 1984; Gorman et al., 1986), intravascular blood volume and cerebral blood flow (Mathew, Wilson, & Tant, 1989; Reiman et al., 1989) have been postulated as mechanisms for lactate-induced panic, available evidence suggests that none of these are requisite to its panicogenic properties.

Nonetheless, there is considerable evidence to suggest that the incidence of lactate-induced panic attack is higher in panic-prone patients (50-70 %) than healthy controls (10 %: Pitts & McLure, 1967; Appleby, Klein, Sacher, & Levitt, 1981; Gorman et al., 1989; Liebowitz et al., 1984; Pohl et al., 1988; Rainey et al., 1984; Rifkin, Klein, Dillon, & Levitt, 1981).

It is now questionable, however, whether it is the degree of responsiveness to lactate infusion or differences in baseline levels of anxiety and heart rate that explain differences in

outcome of lactate infusion in panickers and controls (see Margraf et al., 1986). Specifically, several studies have reported that panic patients exhibited higher levels of anxiety and heart rate than controls prior to infusion of lactate (Freedman, Ianni, Ettedgui, Pohl, & Rainey, 1984; Liebowitz et al., 1984; Rainey et al., 1984). Therefore, it is possible that the lactate infusion simply elevates heart rate and anxiety equally within each group, and that the initially elevated levels within panickers aid induced increases to result in an anxiety attack. This thesis has been supported empirically by Ehlers et al. (1986).

In addition, there are several physiological studies that have indicated cognitive factors as being important in the mediation of lactate-induced panic attacks. For example, Gaffney et al. (1988) and Freedman et al. (1984) have indicated that there are no differences between patients and controls in autonomic nervous system or cardiopulmonary functioning in response to lactate infusion. However, these researchers found that panickers were emotionally overresponsive toward the physiological changes induced by the infusions. It may be, therefore, that cognitions mediate the occurrence of panic attacks. This thesis will be further elaborated in subsequent sections.

A Psychophysiological Approach to Panic

Models of panic proposed by Clark (1986) and Beck and Emery (1985) maintain that panic is the result of a positive feedback

loop in which the perception of internal cues (e.g., somatic sensations or cognitions) leads to catastrophic interpretations of immediate threat to one's well-being. Similarly, Ehlers, Margraf, and Roth (1988) have proposed that panic results from a cumulative cycle of anxiety-based appraisals of cognitive and physiological changes that result from various situational variables (e.g., physical effort, caffeine intake, stress, emotionality).

In general, these models posit that panic is the result of a positive feedback loop between physiological changes in the body and an individuals cognitive response to these changes (Barlow, 1988). Consequently, these models can accommodate and explain a wide variety of agents as potential triggers of panic episodes. Triggers may include: a) internal or external stressors that increase the likelihood of threatening cognitive or physiological events, b) individual biological (e.g., beta-adrenergic hypersensitivity) or psychological (e.g., anxiety sensitivity, selective attention) predispositions, and c) situational variables that influence whether somatic sensations are associated with fear and danger (e.g., coping resources, availability of explanations for somatic sensations).

The Psychophysiology of Panic

Psychophysiological investigations have documented evidence consistent with heightened biological reactivity in panic patients. Agoraphobics and panickers have been found to exhibit higher resting heart rates than controls (Holden & Barlow, 1986;

Michelson & Mavissakalian, 1985; Woods et al., 1987). It has also been demonstrated that resting anxiety patients show higher levels of palmer skin conductance and a greater frequency of spontaneous skin conductance fluctuations (Chattopadhyay, Cooke, Toone, & Lader, 1980; Lader & Wing, 1964; Raskin, 1975). Furthermore, under conditions of passive stimulation, these patients exhibit a decreased degree of habituation of skin conductance orienting responses relative to controls (Lader, 1980; Lader & Wing, 1964; Raskin, 1975). According to Hugdahl, Fredrickson, and Ohman (1977), heightened physiological arousal in conjunction with habituation deficits may be a prerequisite for the development of panic. Moreover, it has been advanced that these same physiological characteristics may reflect the underlying substrate upon which both emotionality and clinical anxiety are based (McGuffin & Reich, 1984).

Contrary to the aforementioned evidence, the general finding of a heightened biological reactivity in panickers has not been established without question. For instance, Hoehn-Saric and McLeod (1985, 1988) maintain that the precipitating factors of several anxiety disorders, including panic disorder, remain unclear. Nevertheless, the majority of investigations of the psychophysiology of panickers suggest that they are characterized by a state of heightened autonomic arousal. The mechanism by which this heightened autonomic arousal is translated into panic may, however, involve psychological elaboration (Beck & Emery, 1985; Clark, 1986; Ehlers, Margraf, & Roth, 1988).

The cognitive predisposition to interpret arousal (i.e., heightened physiological activity) as threatening has been referred to as anxiety sensitivity (Reiss & McNally, 1985). Donnell and McNally (1989) have indicated that "anxiety sensitivity denotes the tendency to respond anxiously to arousal symptoms" (pp. 325-326). Thus, persons with high anxiety sensitivity may be more likely than others to panic when experiencing physiological arousal because they believe that the anxiety may have harmful consequences. That is, they may be more likely to catastrophically misinterpret bodily sensations (e.g., rapid heart beat may be misinterpreted as an impending heart attack). It has been reported that panickers do indeed score high on measures of anxiety sensitivity (McNally & Lorenz, 1987; Reiss, Peterson, Gursky, & McNally, 1986). Recently it has been reported that 50 % of high anxiety sensitive individuals report having experienced panic whereas only 11.1 % of low anxiety sensitive persons do so (Cox et al., in press).

In a similar vein, recent investigations have indicated that panickers are characterized by an attentional bias toward physical threat cues. Specifically, panic patients and infrequent panickers have been found to be slower than controls at color-naming words related to physical threat during a modified Stroop color-naming task (Ehlers, Margraf, Davies, & Roth, 1988). Group responses to socially threatening and neutral words did not differ. Recently, other investigators have reported similar findings (Asmundson, Sandler, & Wilson, 1990;

Hope, Rapee, Heimberg, & Dombeck, 1990; McNally, Riemann, & Kim, 1990). Generalized anxiety patients have also been found to selectively attend to threat cues, but to exhibit a bias toward socially threatening cues (MacLeod, Mathews & Tata, 1986; Mathews & MacLeod, 1985, 1986). Together, these results provide evidence suggestive of a predisposition within panickers to attend and respond anxiously to physical threat cues.

Theoretically, the combination of a biological predisposition (e.g., heightened autonomic arousal) and a psychological predisposition (e.g., anxiety sensitivity, attentional bias toward physical threat) may provide the factors necessary and sufficient to promote the occurrence of panic. This proposition is consistent with the assertions of Barlow (1988), Clark (1986), and Ehlers, Margraf, and Roth (1988).

Visceral Self-Perception and its Measurement

During the previous century there have been several theories postulated regarding the role of arousal in emotion (e.g., Cannon, 1929; Duffy, 1934, 1951, 1957; Schachter & Singer, 1962). Both Cannon (1929) and Duffy (1951, 1957) have proposed that there are no <u>specific</u> patterns of autonomic arousal associated with the experience of distinct emotions. Instead, these theorists suggested the notion of a generalized visceral arousal that underlies all emotion. Schachter and Singer (1962) revised this line of reason, postulating that when individuals perceive generalized visceral arousal they explain it by labelling the experience as an emotion that is consistent with environmental

cues. While a number of criticisms have been raised regarding the notion of a generalized visceral arousal (Neiss, 1988, 1990) and the importance of the cognitive response to unexplained arousal (Reisenzein, 1983), none have questioned the assumption that cognitive labelling, and ultimately emotion, are secondary to the perception of visceral arousal. Despite the importance that these theories place on visceral self-perception, there has been little empirical investigation into this topic.

Although research on visceral self-perception with respect to theories of emotion has been scant, there has been considerable investigation in other areas. For instance, interest in the assessment of visceral self-perception has been influenced by research into biofeedback. Brener (1977; Brener & Jones, 1974) hypothesized that biofeedback enhances the ability of individuals to discriminate between visceral sensations and, hence, provides them with the ability to voluntarily control visceral responses. In order to test this hypothesis valid methods for assessing visceral perception have been developed.

The first of these required subjects to tap in rhythm with their perception of their heartbeat (McFarland, 1975). A number of variations of this subjective estimation method have been employed by other researchers (e.g., Tyrer, Lee, & Alexander, 1980; Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988). The second approach required the subject to discriminate between two sets of signals, one of which was a veridical analogue of the heartbeat, and one which was not (Brener & Jones, 1974;

Whitehead, Drescher, Heiman, & Blackwell, 1977).

Of these two general approaches to the measurement of visceral perception, the latter more objective technique has proven to be more acceptable. Signal discrimination techniques have been developed by Brener and Jones (1974), Katkin and colleagues (Katkin, Morell, Goldband, Bernstein, & Wise, 1982), and Whitehead et al. (1977). It should be noted, however, that these techniques have been applied in fundamentally different ways. Specifically, the Brener-Jones and Katkin procedures involve the provision of performance feedback to the subjects; no such feedback is given in the Whitehead procedure. Obviously, then, the former procedures involve learning the skill to discriminate visceral sensations (akin to biofeedback), whereas the latter deals with the measurement of naturally existing levels of visceral perception. In support of this distinction, Jones et al. (1984) have suggested that the Whitehead procedure is a stronger test of awareness of visceral afference. Furthermore, Ross and Brener (1981) have noted that the Whitehead procedure elicits passive strategies that involve introspection to detect interoceptive visceral sensations, whereas the Brener-Jones procedure elicits active solution strategies based on learning. Katkin (1985) has indicated that his discrimination procedure is also based on challenge and learning.

Empirical investigation also supports the distinction between these procedures. Jones et al. (1984) have reported that the correlations between the Brener-Jones and Whitehead

procedures are weak and negative (-.09 to -0.11).

Aside from procedural distinctions, a number of individual differences that appear to systematically affect an individual's ability to perceive visceral sensations, particularly the beating of the heart, have been identified. These include gender (males superior to females; Whitehead et al, 1977), hemispheric laterality (right hemisphere superior to left; Hantas, Katkin, & Reed, 1982; Montgomery & Jones, 1984), body position (sitting superior to standing; Jones, Jones, Rouse, Scott, & Caldwell, 1987), exertion and physical activity (exertion superior to rest; Jones & Hollandsworth, 1984) and body composition (lean superior to non-lean; Jones et al., 1987; Montgomery & Jones, 1984; Rouse, Jones, & Jones, 1988).

Katkin (1985) has reported that males are better able to learn heartbeat perception when performance feedback is provided. It is interesting to note, however, that the gender effects reported by Whitehead et al. (1977) are generally not reported for the Brener-Jones (Brener & Jones, 1974; Ross & Brener, 1981) or Katkin (Katkin, 1985; Katkin et al., 1982) procedures when they withhold performance feedback. Recent investigation suggests that the reported gender effects evident in the Whitehead procedure may be an artifact of body composition. Rouse et al. (1988) found that lean subjects were better heartbeat discriminators than non-lean subjects. Also, when body fat was manipulated through pre-selecting subjects for body composition, there were no significant differences between gender for

heartbeat detection. It remains undetermined whether these results generalize to the procedures involving performance feedback.

From this brief review it becomes apparent that the procedures and results of visceral perception research are complex - the various objective procedures are uncorrelated and there are many individual differences in the ability to detect visceral sensations. Nevertheless, this avenue of investigation, still in its early stages, may prove valuable to areas of psychophysiology to which it has not yet been applied, including that of panic attacks.

Autonomic Hyperarousal, Anxiety Sensitivity, and Somatic Awareness

From a psychophysiological perspective, there are two salient features that may interact to produce episodes of panic. As discussed, panickers exhibit increased levels of autonomic arousal as evidenced through their elevated resting heart rates (Holden & Barlow, 1986; Michelson & Mavissakalian, 1985; Woods et al., 1987), elevated heart rates prior to lactate infusion (Freedman et al., 1984; Liebowitz et al., 1984; Rainey et al., 1984), and labile palmer skin conductance profile (Chattopadhyay et al., 980; Lader, 1980; Lader & Wing, 1964; Raskin, 1975). Additionally, panickers display an attentional bias for physically threatening stimuli (Asmundson et al., 1990; Ehlers, Margraf, & Roth, 1988; Hope et al., 1990; McNally et al., 1990) and a tendency to respond to such stimuli in an anxious manner

(Donnell & McNally, 1989; McNally & Lorenz, 1987; Reiss et al., 1986).

Research has indicated that a consequence of increased physiological arousal is that the perception of bodily sensations becomes more acute (e.g., Jones & Hollandsworth, 1981; Katkin et al., 1982). At rest, the majority of normal subjects show little ability to accurately perceive the beating of their heart (Katkin, 1985; Katkin et al., 1982; Whitehead et al., 1977). Above chance levels of cardiac awareness on the Whitehead heartbeat discrimination procedure (Whitehead et al., 1977) have been shown to range from 25 to 40% (Jones, O'Leary, & Pipkin, 1984; Montgomery & Jones, 1984). Katkin and colleagues (Katkin, 1985; Katkin et al., 1982) have not indicated the incidence of above chance awareness levels for their discrimination procedure, however they reported that only subjects who show heightened levels of arousal (i.e., electrodermally labile subjects) can be trained to be accurate heartbeat discriminators (Katkin et al., 1982). For several reasons these results are compelling to those investigating panic. First, it is of interest as to what proportion of the 25 to 40% of good heartrate perceivers on the Whitehead procedure are panickers. Since the incidence of panic was not assessed, there is no way of determining this from the results of Jones and colleagues (Jones et al., 1984; Montgomery & Jones, 1984). Nevertheless, the percentage of good heartbeat discriminators is similar to the percentage of individuals in the general population that report infrequent panic attacks (Brown &

Cash, 1990; Norton et al., 1985, 1986; Telch et al., 1989). Second, it is interesting to note that the subjects to whom Katkin et al. (1982) were able to teach accurate heartbeat perception possessed a characteristic common to sufferers of anxiety - labile electrodermal responsivity. Other studies have shown that short-term enhancement of physiological arousal (e.g., via physical exercise) can lead to significant improvements in cardiac awareness (Jones & Hollandsworth, 1981; Montgomery, Jones, & Hollandsworth, 1984). Overall, it seems reasonable to speculate that the chronically heightened autonomic arousal characteristic of panickers may lead to heightened levels of visceral awareness.

It has been documented that panickers report themselves to be acutely aware of physiological changes within their bodies (Chambless, Caputo, Bright, & Gallagher, 1984; Hibbert, 1984; King, Margraf, Ehlers, & Maddock, 1986; McNally & Lorenz, 1987), and that they interpret these changes as indicative of impending danger (Clark et al., 1988; Foa, 1988; van den Hout, 1988). It appears, therefore, that panickers are aware of physiological changes in somatic activity, and that these changes take on special significance. That is, panickers tend to express great concern, and even fear, toward changes that may indicate that something is wrong with their body -- they are highly anxiety sensitive. Thus, they may have learned to monitor somatic activity in order to assess the functional integrity of their body.

Moreover, Hibbert (1984) and Ley (1985) have indicated that the perception of changes in somatic activity precedes the fear associated with panic. Thus, somatic sensations may serve as cues that trigger episodes of panic. Barlow et al. (1985) have suggested that this type of interoceptive conditioning may lay the foundation for a maladaptive biofeedback circuit capable of initiating and sustaining panic attacks. In short, it appears that the cognitive profile of people who suffer from panic attacks is characterized by a high level of concern regarding physical well-being, which may cause them to be vigilant for minor variations in somatic function that they believe signal the onset of an attack. A critical question, then, is whether panickers are actually accurate perceivers of somatic activity.

Unfortunately, the physiological-cognitive feedback cycle has been the focus of little empirical analysis. Using self-reports, King et al. (1986) found panic patients to describe high awareness of their heartbeat and other visceral functions. However, only two studies have directly assessed the accuracy of heartbeat perception among anxiety patients. First, Tyrer et al., (1980) explored the degree of cardiac awareness among a group of psychiatric out-patients suffering from anxiety neurosis, phobic anxiety, or hypochondriasis. Patients subjectively estimated their heart rate during exposure to placid and potentially stressful films. EKG was also recorded during this period. Subsequently, subjective and objective heart rate measures were correlated. Results indicated that both the

anxious and hypochondriacal patients produced significantly higher correlations than did the phobics. Tyrer et al. (1980) proposed that anxious patients develop a heightened sensitivity to normal bodily sensations because of the association between these sensations and feelings of threat and panic. In a more recent study, Ehlers et al., (1988) assessed the accuracy of heart rate perception in panic patients and controls. Heart rate perception was assessed by having subjects match the rate of tone pips to their heart rate. An error score was then determined by taking the absolute difference of estimated and EKG-determined heart rate. It was found that panic patients and controls did not differ in their accuracy of heart rate perception.

The results of each of these studies can be criticized on several grounds. In the Tyrer et al. (1980) study no control group was assessed. Therefore, the extent to which an anxious patient's cardiac awareness is heightened above normal levels cannot be determined. More importantly, the methodology employed in each study was such that subjects simply had to guess their average heart rate without actually discriminating between individual beats. Consequently, the good performance of the Tyrer et al. (1980) subjects may have resulted from good guesses, whereas the poor performance of the Ehlers et al. (1988) subjects may have resulted from poor guesses. Katkin et al. (1982) have made similar criticisms of the procedures used in the aforementioned investigations.

Purpose and Hypothesis

The purpose of the present study was to explore the relationship between panic attacks, anxiety sensitivity and cardiac awareness using a paradigm that overcomes the shortcomings of previous methodologies employed in research addressing this issue (e.g., Ehlers et al., 1988; Tyrer et al., 1980). As previously noted Brener and Jones (1974), Katkin et al. (1982), and Whitehead et al. (1977) have all developed heart rate discrimination procedures that meet this criterion. The focus of this investigation was to explore naturally heightened cardiac awareness (rather than to teach heartbeat discrimination), therefore visceral perception was assessed using the Whitehead procedure. Heart rate discrimination was assessed during a period of rest and following a biological challenge designed to induce a state of physiological arousal. Hyperventilation, a commonly used procedure for the production of panic-like symptoms (see Holt & Andrews, 1989), was chosen as the biological challenge. The major question addressed was whether the combination of an apparent biological and psychological predisposition toward panic would increase an individual's level of visceral awareness. The major hypothesis proffered was that individuals with high anxiety sensitivity (a presumed psychological predisposition toward panic) and a history of panic attacks (evidence of biological influences) would show greater cardiac awareness during rest and following biological challenge than panickers with low anxiety sensitivity and non-panickers

with high and low anxiety sensitivity. Further, it was presumed that anxiety sensitivity and panic history would overshadow gender differences in cardiac awareness.

Method

Subjects and Subject Selection

A total of 463 introductory psychology students at the University of Manitoba, Winnipeg, Canada were mass screened in order to identify their previous experience with panic attacks and their level of anxiety sensitivity. Panickers and non-panickers were identified using the Anxiety Questionnaire (AQ, Telch et al., 1989), a 15-item instrument designed to obtain information relevant for making DSM-III-R diagnoses of panic disorder. Criteria for inclusion in the panic group were based on frequency of attacks and symptom severity. Specifically, subjects included as panickers had to report experiencing at least one panic attack in the last month, at least one other attack during the last year, and four symptoms of at least moderate severity at the time of questioning.

Overall, 36.5 % (N = 169) of the sample reported having experienced panic attacks at some point during their life, 32.61 % (N =151) reported having had at least one attack in the last year, 10.58 % (N= 49) reported having experienced spontaneous panic attacks, and 20.73 % (N = 96) met the aforementioned criteria for inclusion in the panic group. Approximately 63.5 % (N = 294) of the sample had never experienced a panic attack.

Anxiety sensitivity (AS) was assessed for individuals

meeting the panic group criteria and for those having never experienced panic attacks. AS was determined using the Anxiety Sensitivity Index (ASI: McNally & Lorenz, 1987; Reiss et al., 1986). In accord with normative data (Peterson & Reiss, 1987), high AS individuals were designated as those scoring 30 or more on the ASI; low ASI individuals were those scoring 10 or less. Due to the relative scarcity of low AS panickers a more liberal criterion had to be accepted for entrance into this group (i.e., four panickers were accepted with ASIs of 14).

The final experimental sample comprised four groups of 20 subjects, sub-divided on the basis of panic history and level of ASI; high AS panickers (12 females, 8 males), low AS panickers (12 males, 8 females), high AS nonpanickers (10 males, 10 females), and low AS nonpanickers (10 males, 10 females). The mean age of individuals in these groups were 20.0, 19.3, 20.2, and 19.0 years, respectively. Complete subject information is summarized in Table 1.

Table 1

Summary Table of Subject Information

Measure	Mean Score				F	Prob.
	Panicker		Nonpanicker			
	Low As	High As	Low AS	High AS		
Age	19.3	20.0	19.0	20.2	ns	
ASI	10.2	33.4	7.5	32.3	227.4	.0001
STAI-state	35.3	47.7	35.3	45.3	9.2	.0001
Height(cm)	174.4	171.4	170.4	171.8	ns	
Weight(kg)	70.1	68.0	63.2	66.7	ns	
OI	.00229	.00231	.00216	.00225	ns	

Note: ASI = Anxiety Sensitivity Index, STAI = State-Trait Anxiety Inventory, OI = Obesity Index.

degrees of freedom on all tests were 1 and 72.

Apparatus and Materials

Materials

All subjects completed an inventory comprising a number of self-report questionnaires (see Appendix A). The primary instrument used to screen the occurrence of panic was the AQ (Telch et al., 1989). The AQ is a 15-item questionnaire designed to obtain information necessary for making the diagnosis of panic disorder according to DSM-III and DSM-III-R criteria. The ASI (McNally & Lorenz, 1987; Reiss et al., 1986), a 16-item questionnaire that measures the fear of anxiety, was used as an index of anxiety sensitivity.

Additional questionnaires were completed by individuals selected for the experimental session. Indices of state anxiety were obtained from the State-Trait-Anxiety-Inventory (STAI, Spielberger, Gorsuch, & Luschene, 1970). A brief questionnaire was given to probe subjects' anticipated level of cardiac awareness. Finally, a 16-item symptom checklist was administered to probe somatic sensations and cognitions at three points during the experiment. All subjects participating in the experimental session were also interviewed using a shortened version (i.e., Panic Disorder Module) of the Structured Clinical Interview for the DSM-III-R (SCID; Spitzer et al., 1987) in order to verify accuracy of self-reports of panic history. Agreement between the AQ and SCID has been shown to be acceptable (Kappa = .79; Telch et al., 1989).

Apparatus

EKG was recorded in a sound-attenuated, temperature-controlled environmental chamber containing an armchair for the subjects and a video camera by which subjects' activity was monitored visually. Subjects remained in audio contact with the experimenter via an intercom system.

The EKG was monitored by standard lead II electrode placement from electrodes connected to both ankles and the right arm. A Grass model 7 polygraph was used to record the EKG, and the signal was fed on-line to a laboratory computer (386-2 Mini Tower VGA). Detection of EKG R-waves was facilitated with a hardware device which allowed the computer to detect cardiac-generated voltage peaks above a predetermined level. Interbeat intervals (IBIs), were assessed as maximum peak to peak voltage, and stored on disk.

The laboratory computer was programmed to control the timing, presentation, scoring, and analysis of the various components of the procedure. Discrimination trials were presented according to the standard Whitehead procedure (Whitehead et al., 1977). The computer monitor was located approximately 2 m in front of the subject. Prompts corresponding to each of the experimental phases (i.e., "Rest", "Get Ready", "Respond") were presented by the computer. Feedback stimuli were 100 ms flashes of a computer-generated stimulus (series of Xs) presented during the discrimination trials. Following each trial the subject indicated whether the feedback stimuli were

synchronous or asynchronous with their heartbeat by pressing the appropriate key on a response panel. Responses were detected and scored by the computer.

Procedure

Initially, subjects were mass screened using the AQ and ASI in order to identify their previous experience with panic and their level of anxiety sensitivity. All respondents were divided into groups of those who had and had not experienced panic attacks. Subjects who reported having experienced panic attacks consistent with panic criteria outlined previously became candidates for the panicker groups; those not reporting any experience with panic became candidates for the non-panicker groups. Levels of AS were then assessed, and individuals within each of the panic groups were sub-divided into those with high and low AS. Subsequently, subjects were contacted by phone in order to arrange a laboratory appointment.

Upon arrival at the laboratory, subjects were advised of the general procedures of the experiment and informed consent was obtained (see Appendix B). The primary investigator was blind to the group designation of each subject, and became aware of specific designations only after the subject had gone through the protocol and proceeded to the clinical interview.

Subjects were tested individually. The subject was seated comfortably in the subject room, and asked to complete the state form of the STAI, a brief questionnaire probing anticipated levels of cardiac awareness, and a 16-item symptom checklist.

Following completion of the questionnaires, electrodes were applied, and the discrimination procedure was explained by computer-generated instructions of the task (see Appendix C). Immediately thereafter, 10 practice discriminations were given in order to familiarize the subject with the task. The subject was permitted to ask any questions she/he may have had.

Prior to the actual discrimination task, measures of heart period were recorded on disk for the last 5 minutes of a 10 minute baseline adaptation period.

The Whitehead procedure (Whitehead et al., 1977) was used to assess cardiac awareness. This procedure involves the discrimination of two types of feedback stimuli (100 ms stimulus flashes), S+ and S-, presented in different temporal sequence following ventricular depolarization (denoted by the EKG R-wave). Typically, S+ stimuli are delayed 128 ms after each R-wave, and should be perceived as synchronous with the subjects' heartbeats (see Brener & Kluvitse, 1988). S- stimuli are delayed 384 ms following the R-wave, and should be perceived as out of phase (see Brener & Kluvitse, 1988). These conventions were maintained in this experiment. A total of 60 feedback trials, 10 s in duration, were presented. Three blocks of 10 discrimination trials were given immediately following the baseline measures while the subject was at rest. Following these trials the subject completed the 16-item symptom checklist for the second time.

Each of the remaining 3 blocks of discrimination trials followed a brief period of voluntary hyperventilation.

Hyperventilation was performed for 90 s prior to each of the last three trial blocks. The computer generated prompts (e.g., "Inhale", "Exhale") encouraging the subject to breath at a rate of 30 breaths per minute, a frequency used in the literature to operationalize hyperventilation (Magarian, 1982; Gorman et al., 1984; Holt & Andrews, 1989). Subjects were also instructed to breathe as deeply as possible. No mechanical measures of respiratory rate or volume were taken; however, compliance was checked visually via the video monitor.

For all blocks (i.e., pre- and post-hyperventilation) each discrimination trial was preceded with the presentation of a 4 s "Ready" prompt which allowed the subject to focus attention on cardiac activity. Following this were the presentation of either S+ or S- stimuli, which the subject judged as being either synchronous or asynchronous with their heartbeat. Responses were prompted by a message on the computer screen and were made by pressing the appropriate key on the response panel. The "true" key was activated if the feedback was perceived as synchronous with heartbeat; if not, the "false" key was activated. Each trial and decision was followed by a 7 s rest period.

Over the 60 discrimination trials, the probability of S+ and S- feedback was equal ($p = .5$). Additionally, the probability of S+ and S- feedback within each block of 10 trials was equated.

Following completion of the post-hyperventilation discrimination trials the experimenter administered the 16-item questionnaire to probe somatic sensations and cognitions

experienced during hyperventilation. Subsequently, electrodes were removed and the SCID interview was given. Subjects were then debriefed, and questions were answered.

Data Reduction and Analysis

Perceptual sensitivity

Pre- and post-hyperventilation perceptual sensitivity for heartbeat measures were calculated for each subject based on their accuracy of discriminating S+ and S- feedback. These measures were calculated using a nonparametric approximation of d' (Green & Swets, 1966) that is frequently used with the Whitehead procedure -- 2arcsin P(A) (McNicol, 1972). The 2arcsin P(A) transformation effectively separates discrimination ability from response bias, and is almost perfectly correlated with d' between d' values of 0 and 3. The perceptual sensitivity measure, as used here, was calculated in a manner such that departures from randomness in either direction were taken as indications of discrimination. Thus, situations in which good discriminators consistently mislabelled stimuli and received low scores were avoided (see Jones et al., 1984). Consequently, discrimination values could range from pi/2 (1.571 = chance) to pi (3.1415 = perfect discrimination).

Repeated measures analysis of variance (ANOVA) techniques were employed to compare high and low AS panickers and non-panickers on heartbeat perceptual sensitivity during rest and biological challenge. The between subjects variables were panic history, AS, and gender. The within subjects variable was

experimental phase (i.e., pre-, post-hyperventilation).

Physiological indices

Heart period, recorded by computer scoring of IBIs, was averaged over the final 5 minutes of the 10 minute baseline period. Heart period during the discrimination trials was obtained by determining the period of complete IBIs occurring during the 4 s ready signal prior to every trial.

ANOVA techniques were used in the comparison of high and low AS panickers and non-panickers on the physiological indices taken during baseline and the pre- and post-hyperventilation phases of discrimination trial presentation.

Results

Overview

Several interesting aspects of the relationship between panic attacks and cardiac awareness emerged in the analysis of the data. The results indicated that: a) cardiac awareness during rest was superior to awareness following hyperventilation; b) low AS male nonpanickers and high AS male panickers were more accurate at perceiving heartbeats at rest than any other group, but only the former performed significantly better than chance; and, c) panic attacks and other indices of anxiety were not good predictors of accurate heartbeat perceivers. These results are covered in detail below, as are those pertaining to physiological measures and symptom ratings.

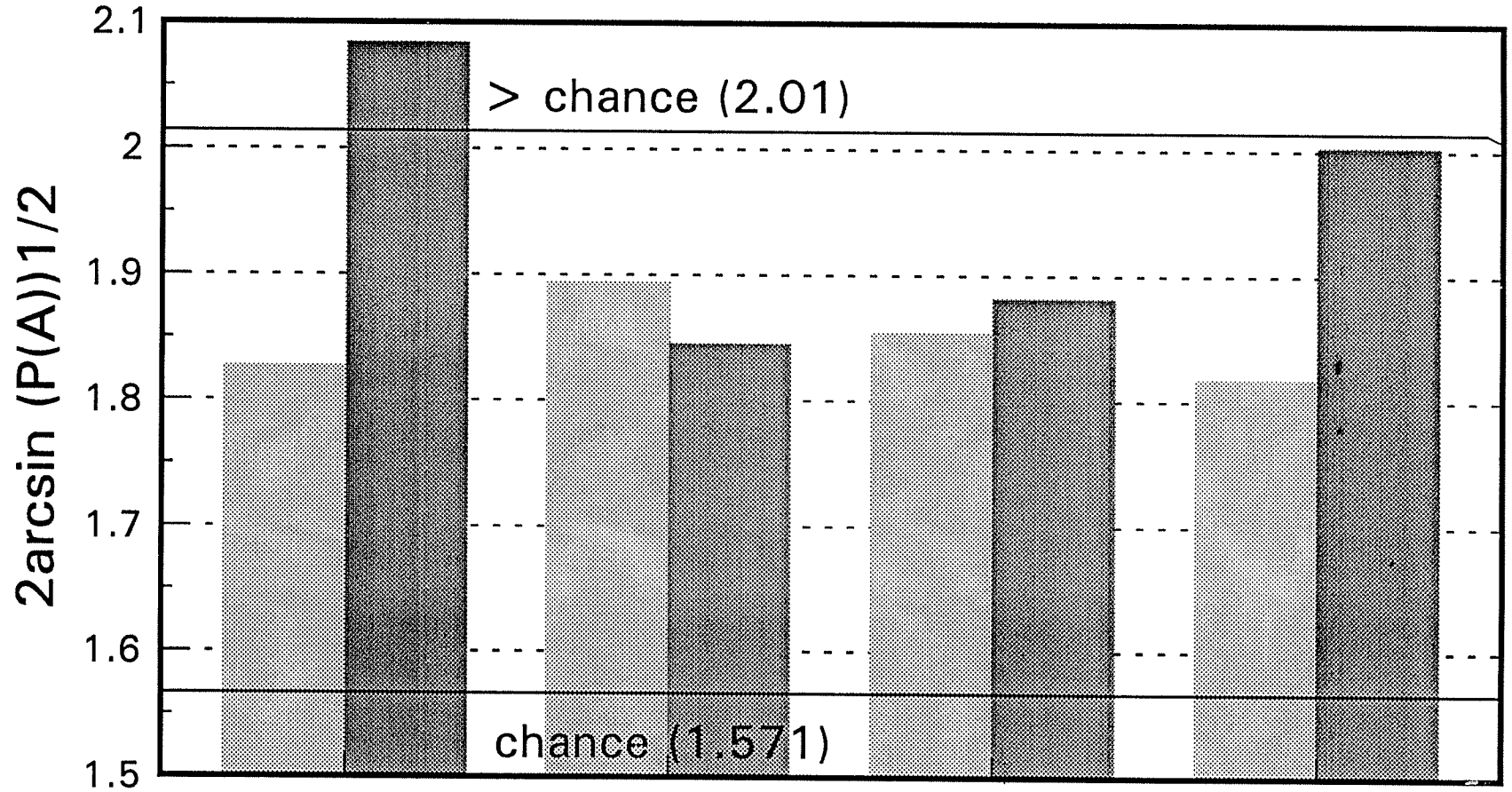
Discrimination Accuracy

The primary focus of this experiment was an evaluation of

the nature of the relationship between panic history, AS, and cardiac awareness. This inquiry was evaluated in a 2 (Panic History: Positive, Negative) x 2 (Anxiety Sensitivity: High, Low) x 2 (Gender) x 2 (Phase: Pre-, Post-hyperventilation) repeated measures ANOVA of perceptual sensitivity scores. The analysis of between subjects variables revealed no significant main effects or interactions (all $Fs(1,72) < 2$). The analysis for the within subjects variable yielded a main effect for Phase ($F(1,72) = 5.18$, $p < .05$), indicating that perceptions of cardiac activity were more accurate during Phase 1 (2arcsin p(A) = 1.892) than Phase 2 (2arcsin P(A) = 1.825). A significant Phase x Panic History x Anxiety Sensitivity x Gender interaction ($F(1,72) = 5.63$, $p < .02$) was also observed.

In order to investigate the nature of this complex interaction, separate ANOVAs were performed for Phase 1 and Phase 2 perceptual sensitivity data. There were no significant main effects or interactions for Phase 2 (all Fs $(1,72) < 1$). For Phase 1, however, there was a marginally significant main effect of Gender ($F(1,72) = 3.65$, $p < .06$) revealing that males were more accurate cardiac perceivers than females (2arcsin p(A): 1.942 vs 1.844). A significant Panic History x Anxiety Sensitivity x Gender interaction ($F(1,72) = 4.42$, $p < .05$) was also observed. The means for this interaction are shown in Figure 1).

Figure 1 Mean Perceptual Sensitivity for Phase 1 Discriminations



Group	Low AS/Nonpanickers	Low AS/Panickers	High AS/Nonpanickers	High AS/Panickers
female	1.826	1.894	1.854	1.817
male	2.084	1.845	1.882	2.003

Note: AS = Anxiety Sensitivity

Apriori planned pairwise comparisons (Kirk, 1968) of the Panic History x Anxiety Sensitivity x Gender interaction ($F(1,72) = 4.42$, $p < .05$) showed that male nonpanickers with low AS were significantly more accurate heart beat perceivers than male panickers with low AS, $t(36) = 3.175$, $p < .025$. High AS male nonpanickers and panickers did not differ in accuracy. In addition, male nonpanickers with low AS were significantly more accurate than those with high AS, $t(36) = 2.539$, $p< .05$; whereas, male panickers with high AS were significantly more accurate than those with low AS, $t(36) = 2.048$, $p < .05$. All females were similar in their ability to detect cardiac activity, regardless of their panic history or level of AS. Finally, the comparison of gender indicated that male nonpanickers with low AS were more accurate heart beat perceivers than low AS females nonpanickers, $t(36) = 3.331$, $p < .05$, and that male panickers with high AS were more accurate that females with a similar profile, $t(36) = 2.411$, $p < .05$. There were no differences between low AS male and female panickers, nor between high AS male and female nonpanickers.

One group t-tests were also performed in order to determine which of the 2arcsin P(A) group scores were significantly better than chance. The only group to demonstrate accuracy significantly greater than chance was the low AS male nonpanickers, $t(8) = 1.935$, $p < .05$.

The aforementioned analyses were performed on the original questionnaire-derived groups. The SCID data, however, indicated

that individuals in the low AS panicker group failed to meet the specified criteria for inclusion at the time of testing. In fact, of the 20 subjects originally included in this group, 30 % (N = 6) reported, during interview, that they had never experienced a panic attack. An additional 50 % (N = 10) indicated that they had not experienced a panic episode during the previous month. Only 20 % (N = 4) of the original group continued to meet the criteria at the time of interview. Norton (personal communication, April, 1991) has indicated that low anxiety sensitive panickers have proven to be a problematic group for other researchers as well. Consequently, it was decided that elimination of this group was most appropriate for subsequent tests of panic effects. The data were, therefore, reanalysed using only the high AS panickers and nonpanickers in order to control for AS and probe for specific effects of panic. All of the high anxiety sensitive panickers (N = 20) continued to meet the specified criteria upon interview, and 70 % (N = 14) met DSM-III-R criteria for panic disorder. A 2 (Group) x 2 (Gender) x 2 (Phase) repeated measures ANOVA failed to reveal any significant effects or interactions. Therefore, panic history (as determined by clinical interview) does not appear to be important to cardiac awareness.

Accurate vs Nonaccurate Discriminators

Individual performance within all groups was examined more closely by dividing subjects into groups of those who exhibited high and low levels of visceral awareness. Subjects were

considered to have high awareness (HA) if they exhibited discrimination scores at a level significantly greater than chance (> 64 % correct). According to Jones et al. (1984), 64 % correct is the point of significance for any single subject as derived from expansion of the binomial. Low awareness (LA) subjects were defined as those not included in the HA group.

For Phase 1, the HA group (N = 26) consisted of 16 males and 10 females. The Phase 2 HA group (N = 16) consisted of 6 males and 10 females. Three subjects (1 male, 2 females) were accurate in each phase. Overall, 26 of 80 (32.5 %) subjects demonstrated above chance performance on the pre-hyperventilation phase of the heartbeat discrimination task. Only 16 of the 80 (20 %) subjects, three of whom had been accurate prior to hyperventilation, were accurate following hyperventilation.

Stepwise discriminant function analyses were performed for each gender in an attempt to determine which variables were most important in predicting membership in the HA and LA groups. Predictor variables included the number of panic attacks in the previous month, ASI, OI, relevant phase heart period, irrelevant phase sensitivity index, state anxiety, age and the subject's anticipated level of accuracy. Of all the variables assessed, only OI and anticipated accuracy were significant predictors - OI for females during resting discriminations ($F(1,39) = 4.69$, $p < .05$; lower OI associated with membership in the HA group) and anticipated accuracy for males during post-hyperventilation discriminations ($F(1,37) = 5.32$, $p < .05$; higher anticipated

accuracy associated with HA members). The OI variable yielded an R-square of .11, while the R-square for anticipated accuracy was .13.

Physiological Indices

A 2 (Panic History) x 2 (Anxiety Sensitivity) x 2 (Gender) ANOVA for average interbeat intervals obtained during the baseline period indicated a significant main effect for Gender ($F(1,72) = 6.89$, $p < .01$, with females exhibiting shorter heart periods (IBI = 693.63 ms; 86.5 bpm) than males (IBI = 762.67; 78.7 bpm). Similar gender differences have been reported elsewhere (Rouse et al., 1988; Tortara & Anagnastakas, 1984; Sandler et al., in press). In addition, a 2 (Panic History) x 2 (Anxiety Sensitivity) x 2 (Gender) x 2 (Phase: Pre-, Post-hyperventilation) repeated measures ANOVA of interbeat intervals sampled prior to every discrimination trial revealed a significant main effect for Gender ($F(1,72) = 6.49$, $p < .01$). Male subjects exhibited longer IBIs than female subjects (764.60 ms vs 695.98 ms; 78.5 bpm vs 86.2 bpm). A significant Phase x Anxiety Sensitivity interaction ($F(1,72) = 8.85$, $p < .005$) was also observed. Post-hoc Newmann-Keuls comparisons of the means involved in this interaction indicated that the interbeat intervals of high AS individuals were significantly longer than those of low anxiety sensitive individuals during Phase 2 (740.57 vs 717.67, $p < .05$; 81.0 bpm vs 83.6 bpm), whereas there were no significant differences between groups during Phase 1 (726. 55 vs 732.95; 82.6 bpm vs 81.9 bpm).

Means for IBIs observed during baseline, Phase 1 and Phase 2 discriminations are shown in Table 2.

Table 2 Means of Interbeat Intervals for Each Group During Baseline, Phase 1 and Phase 2 Discriminations

	Means					
	Baseline		Phase 1		Phase 2	
Groups	Males	Females	Males	Females	Males	Females
NP/LAS	747.22	703.82	741.22	684.09	772.44	694.63
NP/HAS	748.30	622.00	749.70	627.60	741.20	624.90
P/LAS	761.58	725.37	758.75	720.12	766.50	729.00
P/HAS	799.62	722.83	825.00	745.42	775.50	736.83

Note: NP = Nonpanicker, P = Panicker, LAS = Low Anxiety Sensitivity, HAS = High Anxiety Sensitivity.

Symptom Ratings

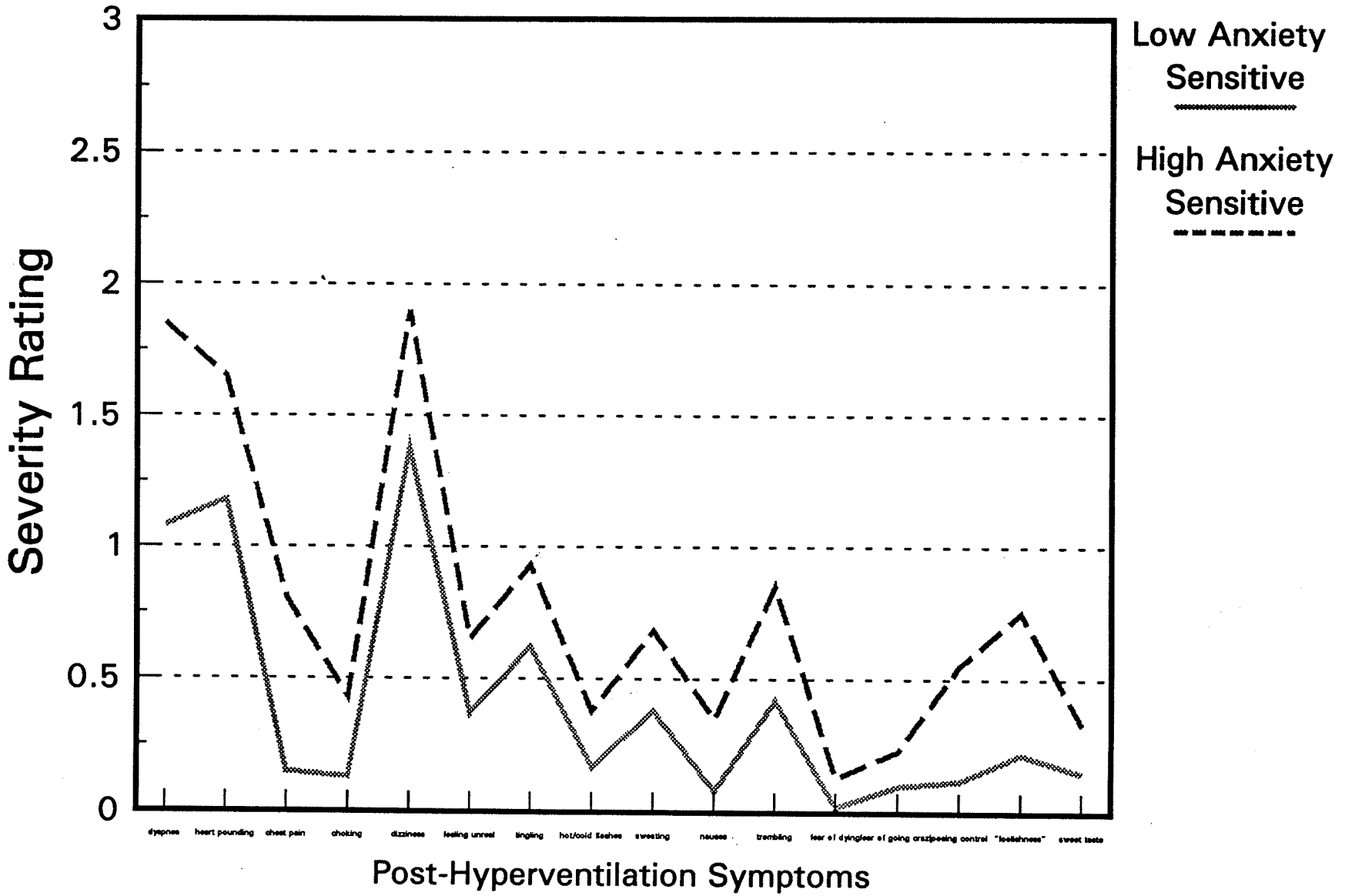
Severity ratings for each of the 16 items of the symptom questionnaire were obtained on 4-point scales (0 = Not present, 1 = Mild, 2 = Moderate, 3 = Severe) prior to baseline heart period measurement and Phase 1 discrimination, and following Phase 2 discrimination. Global severity scores were calculated for each rating period by summing the subjects ratings across all 16 symptoms. A 2 (Panic History) x 2 (Anxiety Sensitivity) x 2 (Gender) x 3 (Phase) repeated measures ANOVA revealed significant main effects for Anxiety Sensitivity ($F(1,72) = 14.67$, $p < .001$), and Phase ($F(2, 144) = 50.34$, $p < .0001$). High AS was associated with greater symptom reporting and more symptoms were reported following hyperventilation than before. However, these main effects were modified by a significant Phase x Anxiety Sensitivity interaction ($F(2,144) = 4.41$, $p < .05$).

Post-hoc pairwise comparisons were conducted on the means involved in this interaction using the Tukey Honestly Significant Difference (HSD) procedure (Kirk, 1969). Symptoms were rated significantly higher across all phases by high AS individuals compared to low AS individuals. Additionally, within each group symptom totals following hyperventilation were significantly higher than either the baseline or pre-hyperventilation phases, $HSD = 2.595$, $p < .01$. There were no significant within-group differences between baseline and pre-hyperventilation symptom ratings.

Individual symptoms rated post-hyperventilation were

analyzed using MANOVA techniques. The MANOVA revealed a marginally significant Symptom Rating x Anxiety Sensitivity interaction, $F(15,58) = 1.71$, $p = .0734$ (using Wilk's lambda). As evident in Figure 2, the symptom profiles for the high and low AS groups exhibit a highly consistent pattern.

Figure 2 Individual Symptom Ratings Following Hyperventilation



However, the results of subsequent univariate ANOVAs revealed that symptom severity ratings differed significantly on nine of the 16 symptoms. Subjects with high AS gave higher ratings on the following symptoms - difficulty breathing ($F(1,72) = 15.88$, $p < .0005$), heart racing or pounding ($F(1,72) = 6.24$, $p < .02$), chest pain or pressure ($F(1,72) = 13.88$, $p < .0005$), choking or smoothering ($F(1,72) = 6.72$, $p < .05$), dizziness/vertigo/unsteadiness ($F(1,72) = 6.31$, $p < .02$), nausea ($F(1,72) = 5.61$, $p < .05$), trembling or shaking ($F(1,72) = 4.80$, $p < .05$), feelings of loosing control ($F(1,72) = 10.70$, $p < .005$), and feeling afraid of making a fool of themselves ($F(1,72) = 12.60$, $p < .001$).

Discussion

The results of the present investigation contribute empirical evidence relevant to panic attacks and cardiac awareness, as well as to the scantly investigated relationship between the two. In the following paragraphs consideration will be given to: a) the relationship between panic attacks and cardiac awareness, b) hyperventilation and cardiac awareness, c) gender and cardiac awareness, and d) the predictors of accurate cardiac perceptions. Furthermore, the implications for psychophysiological models of panic, shortcomings of the present investigation, future directions and practical suggestions will be discussed.

The Relationship Between Panic Attacks and Cardiac Awareness

The results of the present investigation indicate that panic

history is not related to the ability to perceive cardiac activity. Based on the assessment of high anxiety sensitive panickers (many of whom met DSM-III-R criteria for panic disorder) and nonpanickers, for whom interview data supported group designation, there were no differences in observed levels of cardiac awareness. Further, neither of these groups exhibited awareness levels significantly greater than chance. Similar conclusions were yielded via assessment of the groups derived from self-reported panic history: Low anxiety sensitive male nonpanickers and high anxiety sensitive male panickers both exhibited relatively high levels of cardiac awareness at rest; however, only the low anxiety sensitive male nonpanickers displayed awareness levels significantly greater than chance.

Assessment of cardiac awareness using the Whitehead procedure ensures that in order to receive high awareness scores an individual must discriminate between individual beats of their heart. That is, the individual must be able to distinguish S+ from S- stimuli. If a subject has no perceptual sensitivity for their heartbeat, the two sets of stimuli will be indistinguishable. The possibility of good guesses yielding high scores, as is possible with subjective estimation techniques (Katkin et al., 1982), is virtually eliminated. Previous investigation into the relationship between panic anxiety and cardiac awareness has been limited to subjective estimation techniques, and has provided mixed results. Tryer et al. (1980), in the first study of this nature, found that anxiety neurotics

(whom by current DSM-III-R criteria would likely be diagnosed with panic disorder) and hypochondriacal patients had a significantly greater accuracy in estimating their heart rate than did phobic patients. Recently, however, Ehlers et al. (1988) failed to find any differences in the ability of panic patients and healthy controls to subjectively estimate their cardiac activity. As previously mentioned, the results of the former investigation may be an artifact of good guesses on the part of the anxiety patients, while those of the latter may be attributable to panic patients' poor guesses. The results of the present study shed some light on this quandary, supporting the conclusion of Ehlers et al. (1988). In general, panickers appear to be no better or worse at detecting the beating of their heart than healthy control subjects.

Hyperventilation and Cardiac Awareness

Jones and Hollandsworth (1984) have indicated that cardiac awareness increases following exertion and physical activity. In the present investigation, however, the manipulation of physiological arousal via increased respiration rate and depth (i.e., hyperventilation) did not increase levels of cardiac awareness. In fact, resting levels of awareness, while not significantly greater than chance, were superior to those following hyperventilation. Thus, it appears that the process of hyperventilation acted in some way to reduce general perceptual accuracy for visceral activity.

It is important to note that hyperventilation did not appear

to affect (i.e., increase) the level of physiological arousal experienced by subjects. Close scrutiny of the resting post-hyperventilation heart rates, however, provides a clue for the understanding of this anomaly. Heart rates during both of these periods were in the mid-80s. It may be, therefore, that a ceiling for heart rate increases was reached for the particular hyperventilation paradigm used -- 30 breaths per minute, beyond which further increases were unlikely. Since the resting heart rates were so high, the physiological effects of the biological challenge may have been masked. Why were resting heart rates so high? There are several possible answers to this question, the most probable being that all subjects were highly reactive toward the physiological monitoring equipment and chamber. Indeed, it was the first time most of them had been exposed to such a situation. Consequently, a longer baseline period may have been in order.

Although hyperventilation did not produce significant changes in heart period during the discrimination trials, it did increase the number and severity of self-reported panic-related symptoms experienced by all individuals. As expected, post-hyperventilation severity ratings were greater for individuals with high anxiety sensitivity than those without. This finding is consistent with that of Donnell and McNally (1989), suggesting that high anxiety sensitive individuals respond to hyperventilation with more anxiety than do low anxiety sensitive individuals. Nevertheless, for both high and low

anxiety sensitive individuals, post-hyperventilation ratings were much more severe than either the baseline or rest ratings. Thus, while apparently unarousing by standards of objective physiological measurement, hyperventilation was subjectively experienced as arousing.

Why then were there no increases in cardiac awareness following hyperventilation? Several investigators (Montgomery & Jones, 1984; Jones et al., 1984) have reported that subjects who show departures from normal respiration pattern during heartbeat discriminations tend to pause their respiration at the beginning of the discrimination trials. Jones et al. (1984) stated that "subjects' 'pausing' during trials tended to say that the pause let them reduce respiratory interference (noise) and 'feel beats' more clearly" (p. 147). Furthermore, Montgomery and Jones (1984) indicted that their "good heartbeat perceivers... tended to remain quiet and attend to internal sensations by simply slowing their respiration rate, thereby attenuating that source of somatic 'noise' and accentuating more cardiac specific feedback" (p. 464). It is likely that the hyperventilation manipulation prevented subjects in the present study from slowing their respiration at the beginning of discrimination trials, thereby hampering their ability to reduce internal noise and decreasing their ability to accurately detect cardiac activity. In fact, all of the participants in the current investigation reported a mild to moderate difficulty with breathing following hyperventilation. Additionally, 20 % indicated that hyperventilation reduced their

ability to concentrate on the signal produced by their heart.

Gender and Cardiac Awareness

Gender differences in cardiac awareness have been reported in several studies utilizing the Whitehead task. Whitehead et al. (1977) first noted male superiority of visceral perception in a report combining data from several experiments that assessed resting cardiac awareness. Subsequently, Pennebaker and Hoover (1984) replicated these reported gender differences. William and Jones (1982) have also indicated that the incidence of above-chance performance is greater in males than females. The results of the present experiment support both of these conclusions in that males displayed generally higher levels of cardiac awareness than females, and the only group to display an above-chance level of awareness was the low anxiety sensitive male nonpanickers.

It is interesting to note that all previously reported gender differences in investigations using the Whitehead procedure were based on the assessment of resting levels of cardiac awareness. In the current investigation, assessment of perceptual accuracy following hyperventilation failed to reveal a gender difference. In fact, the percentage of males discriminating at above-chance levels fell from 40 % at rest to 15 % following hyperventilation, while the percentage of accurate females remained about the same (25 % at rest versus 27.5 % post-hyperventilation). At present the reasons for this are unknown. Katkin (1985) has proposed that gender differences in

cardiac awareness may be due to a greater beta-adrenergic response to arousal producing situations by men than women, thereby increasing the mens' cardiovascular functions and strength of the signal for discrimination. As previously mentioned, the hyperventilation manipulation used in the present study was experienced as very arousing by the subjects; yet, no gender differences were observed following hyperventilation. Since a difference is expected, but not observed, gender-based differences in the activity of the beta-adrenergic system may not be the best explanation of gender differences in cardiac awareness. However, it is quite possible that hyperventilation, because of its acute nature in the present investigation, did not serve to increase beta-adrenergic functioning (Stein, personal communication, May, 1991). Indeed, the one physiological index of arousal used in this experiment, heart period, showed no significant change following hyperventilation.

Predictors of Accurate Perceptions of Cardiac Activity

In an attempt to comprehensively identify variables that could account for the variability seen in discrimination levels, high and low awareness individuals were assessed using discriminant function analysis. The variables that best predicted membership to high and low accuracy groups were obesity index and anticipated accuracy for women and men, respectively. In some respects these results are consistent with previous research findings. It has been reported by several investigators (Montgomery & Jones, 1984; Jones et al., 1987) that leaner male

subjects tend to be better discriminators of cardiac activity. Rouse et al. (1988) have reported that both lean males and females are better cardiac discriminators. While body composition was assessed in the present experiment only as an incidental variable (it would have been impractical to preselect subjects on the basis of a lean/non-lean dimension) it was found that leanness was characteristic of females who discriminated heartbeats at a level significantly greater than chance. This finding complicates the body composition issue in that, unlike previous reports, body composition was not related to cardiac awareness in males. This issue is further complicated by the results of Gardner, Morrell, Watson, and Sandoval (1990) who found that obese and normal-weight subjects were equally adept at performing heartbeat discriminations.

Rouse et al. (1988) have also reported that perceptions of high accuracy were related to heightened levels of cardiac awareness. The results of the present study indicate that high levels of anticipated accuracy are related to accurate perceptions, but only for male subjects.

Importantly, those variables that were hypothesized to be strong predictors of high awareness of cardiac activity - panic attacks and anxiety sensitivity, were not. This result strengthens the argument that panic attacks are not related to heightened cardiac awareness.

Assuming, however, that beta-adrenergic influences on the heart are a major contributor to increased cardiac awareness, it

is interesting that panic history was not associated with accurate perceptions of cardiac activity. According to many investigators panic is the result of a hypersensitive beta-adrenergic system. Thus, on this basis alone, it seems a reasonable speculation that panickers should exhibit heightened levels of cardiac awareness. However, they do not. There are several implications of this result. On one hand, it may be that panickers do not have a hypersensitive beta-adrenergic system. Indeed, this notion has been challenged on many other grounds (see Margraf et al., 1986). On the other hand, beta-adrenergic activity may not be related to cardiac awareness, at least for the Whitehead procedure. An alternative hypothesis suggests that the somatosensory system may be predominant in accurate perceptions of cardiac activity (Jones, 1982; Jones et al., 1987). Regardless of the specific theoretical orientation adopted to explain cardiac awareness, the present results suggest that panic attacks are not related to accurate perceptions of cardiac activity.

Implications for Psychophysiological Models of Panic

What implications does such a result hold for psychophysiological models of panic? The basic assumption of these models is that misinterpretation of somatic sensations leads to increases in anxiety and physiological arousal, which in turn culminate in panic. That is, changes in somatic activity are perceived and interpreted, in some cases, as threatening. As a result, physiological arousal and somatic activity continues to

increase and the cycle escalates to the point of panic. The results of the present study suggest that panic-prone individuals are not accurate perceivers of cardiac activity. Accurate heartbeat perception, however, is not an essential assumption of the psychophysiological models. Rather, importance is placed on misinterpretation and overresponsiveness toward general changes in cardiac activity, or other somatic function, such as might occur following caffeine intake, postural change, or increased respiration. Thus, reports by panickers of acute awareness for physiological changes in their bodies (Chambless et al., 1984; Hibbert, 1984; King et al., 1986) may reflect their subjective perception of somatic activity, more so than actual somatic activity. In essence, the finding that panickers are not accurate heartbeat perceivers emphasizes the relevance of cognitive factors in the experience of panic.

Recently, empirical evidence has emerged in support of this speculation. Pauli et al. (1991) evaluated the psychophysiological model in a field study in which panickers and healthy control subjects were asked to report cardiac perceptions over a 24 hour period. Ambulatory EKG was also measured during this period. The incidence of reported cardiac perceptions was the same for each group - panickers did not report any more cardiac activity than did control subjects. Interestingly, it was found that heart rate accelerations preceded cardiac perceptions for both groups. However, following cardiac perceptions control subjects experienced cardiac deceleration, whereas panickers had

cardiac acceleration associated with increased anxiety. Thus, as proposed in the psychophysiological models, it seems that a "misinterpretation" of initial cardiac acceleration leads to increased anxiety, augmented accelerations, and possibly panic.

Based on the similar number of cardiac perceptions reported over the monitoring period, Pauli et al. (1991) concluded that panickers are no better at perceiving cardiac activity than healthy control subjects. The results of the present investigation unequivocally support this supposition. While accurate perceptions of cardiac activity would enhance the likelihood of the positive feedback loop being triggered, it is not necessary for the process.

Shortcomings, Future Directions, and Practical Suggestions

The goal of this study was to assess naturally occurring levels of cardiac awareness in panickers. Inherently, persual of this objective introduced a short-coming to the study. The Whitehead procedure, which withholds knowledge of performance results from subjects, does not allow for, and is not designed to investigate learning the skill of accurately discriminating visceral sensations. A future direction may be to see if panickers and nonpanickers differ in their ability to learn how to discriminate heartbeats. The Katkin procedure, which does provide knowledge of results, is an ideal procedure for such a pursuit. Interestingly, Katkin et al. (1982) have reported that subjects who can learn to be accurate heartbeat discriminators exhibit electrodermal lability, a characteristic of panickers.

If it could be shown that panickers can learn to be accurate heartbeat discriminators, there may be some important practical implications.

The strength of conclusions that could be based on initial analyses of groups selected on the basis of self-report information regarding panic history were obviously weakened by failure to correctly identify some panickers. Specifically, 30 % of low anxiety sensitive panickers failed to report ever having experienced a panic attack when given a structured interview. Additionally, for these individuals, the frequency of attacks, general nature of the attacks (i.e., situational vs spontaneous), and number of reported symptoms also varied widely between the self-report and structured interview. These discrepancies underscore problems that investigators may encounter when relying on identification of panickers through self-reports alone: A substantial portion of the sample may represent false positives. Incidentally, when interviewed, 100 % of high anxiety sensitive panickers continued to report having experienced panic attacks with a frequency and nature similar to that given on the self-report. Thus, in the absence of a structured interview, a practical strategy may be to screen panickers for anxiety sensitivity, and to include in the experimental sample only those who exhibit high levels of this characteristic.

Overall, the apparent lack of visceral acuity in panickers underscores the role of cognitive factors in the experience of panic. While there may be a biological basis for panic attacks,

the results of the present investigation suggest that accurate perceptions of a biological pathology, whatever its nature or its physiological repercussions, are not necessary for the experience of panic. Instead, it seems that inaccurate cognitions (e.g., perceptions, interpretations) regarding somatic activity may be a requisite for panic.

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Footnote

[1] According to previous diagnostic criteria of the DSM-III (APA, 1980) panic disorder was diagnosed if an individual had experienced a minimum of three panic attacks in the preceding three weeks in association with a minimum of four of the associated symptoms occurring during one of those attacks.

Appendix A

Dear Introductory Psychology Student:

We would like to thank you for agreeing to participate in our research project. We are continuing to investigate the incidence of panic attack in the university population. We want to learn more about the experience of anxiety and whether you have had a panic attack. A definition of panic is given in the following pages.

We would like to assure you that all of your responses will be strictly confidential. No one other than the principal investigators will have access to the questionnaire data. Further, the data will be coded for computer entery and thus your anonymity will be maintained. The questionnaire forms will be kept under lock and key until coded, and then destroyed.

We ask for your cooperation in completing this questionnaire as truthfully as possible. You will be given 1 hour credit for this task.

We are intending to do some additional investigation of panic attacks in the next few weeks. If you would be willing to help us in this subsequent phase of our research program, please indicate your willingness to be contacted by indicating your name and telephone number in the appropriate spaces on the following page. Participation in this subsequent phase will provide you with additional research credit hours.

Thank you for your cooperation.

Sincerely,

Gordon Asmundson, M.A. &

Lorna Sandler, Ph.D

If you are willing to participate in an additional investigation of panic attacks, please fill in your name and telephone number. You need not have experienced a panic attack to participate. If you have any questions regarding this subsequent investigation please feel free to ask for details. Providing your name and number only indicates your willingness to participate, and does not commit you to taking part in further study.

Name:_____

Telephone Number:_____

Please proceed to the next page.

Name:_____ Age:_____

Date:_____ Sex:_____

Below is a series of questions relating to stress, anxiety, and panic. Read each question carefully, then circle the response that best fits your own experience. There are no right or wrong answers.

PART I

1. How much of the time do you experience stress or anxiety? (circle one number.)

0 Never

1 Rarely

2 Sometimes

3 Much of the time

4 Almost all of the time

5 All the time

2. Would you say that you have a problem coping with strees or anxiety?

a. Yes b. No

3. During the last month, how much has stress or anxiety interfered with your life (e.g., school, job, social life)? (Circle one number.)

0 No interference/impairment

1 Mild interference/impairment

2 Moderate interference/impairment

3 Severe interference/impairment

4 Very-Severe interference/impairment

4. Are you currently taking any prescription medications for stress or anxiety?

a. Yes b. No

5. Have you ever taken any prescription medications for stress or anxiety?

a. Yes b. No

6. Are you currently seeing a doctor or counselor because of stress or anxiety?

a. Yes b. No

7. Have you ever visited a doctor or counselor because of stress or anxiety?

a. Yes b. No

PART II

INSTRUCTIONS: Listed below are several questions concerning your experiences with panic. Before you proceed, it is extremely important that you read carefully the definition of panic given below. Only count your experience as panic if it meets this definition

Definition of Panic: A panic attack is the experience of a sudden surge or spike of intense fear, terror, or feeling of impending doom accompanied by several of the following symptoms: heart racing or pounding; shortness of breath; sweating; dizziness or lightheadedness; feelings of unreality; tingling or numbness; choking; chest pain; trembling or shaking; hot flashes or chills; fear of dying, going crazy, or losing control. Although it is rare to have all of these symptoms during a panic attack, it is common to have several of these symptoms.

A panic attack differs from the feelings of nervousness, tension, or mild anxiety that most of us have when we worry about life circumstances such as school, work, or family. Unlike these milder forms of anxiety or tension, the feelings associated with a panic attack are more intense and come on very abruptly, similar to the rapid onset of feelings that would occur should you find yourself in a situation where you were in immediate danger (e.g., robbery). For this survey, do not count feelings of nervousness, tension, or mild anxiety as a panic attack. However, if these feelings of tension or mild anxiety are followed by a sudden surge of exrtreme fear, terror, or apprehension, then consider this a panic attack.

1. Have you ever felt a sudden rush of intense fear or anxiety or feeling of impending doom (panic attack)? (Note: Answer "Yes" only if your experience meets the above definition of panic.)

a. Yes b. No

IF NO, STOP HERE

1a. Have the attacks been limited to stressful situations, such as

applying for a new job? (Note: Answer "No" if you have had a panic attack at least once in a situation that doesn't usually make you anxious)

a. Yes b. No

1b. Have the attacks been limited to situations where you are the focus of others' attention (such as having to speak in front of a group of people)? (Note: Answer "No" if you have had a panic attack at least once when you were not the focus of others' attention.)

a. Yes b. No

1c. Have the attacks been limited to times when you were taking drugs or medicines such as caffeine, alcohol, cocaine, marijuana, cold medicines, etc.? (Note: Answer "No" if you have had at least one panic attack when you were not taking drugs or medicines.)

a. Yes b. No

1d. Have the attacks been limited to times when you were physically ill? (Note: Answer "No" if you have had at least one panic attack when you were not physically ill.)

a. Yes b. No

2. Have you ever had a panic attack occur totally "out of the blue"? (Note: Answer "Yes" if one or more of your panic attacks occurred in a situation
where you were not expecting it, such as watching TV or sitting

at home.)

a. Yes b. No

3. At what age did you first begin to have panic attacks?

_____ Years Old

4. What were the feelings (symptoms) during your worst attack? (Record a number from the scale below next to each feeling or symptom. For example, if you had mild chest pain during your worst attack you would record a "1" next to that symptom.)

None	Mild	Moderate	Severe
0	1	2	3

_____ shortness of breath

_____ dizziness, unsteadiness, or feeling faint

_____ heart racing or pounding

_____ trembling or shaking

_____ sweating

_____ feeling like you were choking or smothering

_____ nausea, stomach upset, or diarrhea

_____ feeling things around you were unreal, or feeling detached from your body

_____ tingling or numbness in parts of your body

_____ hot flashes or chills

_____ pain or pressure in your chest

_____ feeling afraid that you might die

_____ feeling afraid that you might go crazy

_____ feeling afraid that you might lose control

_____ feeling afraid that you might make a fool of yourself

_____ feeling a sweet taste in your mouth

5. What are the situations in which you have experienced a panic attack? (Note: Check all that apply, even situations in which you have had only one attack.)

___public speaking	___driving	___airplane
___using drugs	___arguement	___after drinking coffee
___riding a bus or train	___sleeping	___shopping
___relaxing	___eating out	___during exercise
___worrying about school	___closed-in place	___large store or mall
___watching TV	___while ill	___working at a job
___waiting in line	___being left alone	___other (Please list)
___during or after sex	___taking a test	

_____

_____

6. Has there ever been a time where you have had four or more panic attacks all within a four-week period?

a. Yes b. No

7. Since your first panic attack has there ever been a period, lasting at least one month, when you were worried a lot about having another attack?

a. Yes b. No

7a. During the past month how much have you worried about having a panic attack? (Note: Circle one number from list below.)

0 Not worried at all during the last month

1 Rarely worried (i.e., less than 10% of the days)

2 Occasionally worried (between 10% and 50% of the days)

3 Frequently worried (between 50% and 90% of the days)

4 Constantly worried (every day or almost every day)

7b. Some people are more frightened by a panic attack than others. If you knew that you were going to have a panic attack tomorrow, how much fear would you have anticipating tha attack? (Circle one number from the list below.)

0 No fear

1 Mild fear

2 Moderate fear

3 Severe fear

4 Extreme (very severe) fear

8. How many panic attacks have you had in the last 30 days? (Record the number in the space below. If you are unsure of the exact number, list one number that is your best estimate.)

_____ panic attacks in the last 30 days

8a. How many panic attacks have you had in the last 6 months? (record the number in the space below)

------ panic attacks in the last 6 months

8b. How many panic attacks have you had in the last year? (record the number in the space below.)

----- panic attacks in the last year

9. During the past month (30 days), how much have the panic attacks (or fear of panic) interfered with your life (e.g., school, job, social life, traveling)? (Circle one number from the list below.)

0 No interference/impairment

1 Mild interference/impairment

2 Moderate interference/impairment

3 Severe interference/impairment

4 Very Severe interference/impairment

10. Are there now situations, places, or activities that you avoid because you are afraid you might have a panic attack?

a. Yes b. No

10a. How much do you avoid each of the activities/situations listed below? (Place a number next to each activity.)

Never Avoid	Rarely Avoid	Sometimes Avoid	Usually Avoid	Always Avoid
0	1	2	3	4

___high places	___shopping malls	___eating out
___alcohol	___open spaces	___being alone

___caffeine ___waiting in lines ___driving on a busy road

___exercise ___enclosed spaces ___movie theaters

___buses or trains ___bridges ---Walking on a busy street

___other (please list)_____

11. Listed below are a number of ways that people attempt to cope or manage panic attacks. Place a check next to each coping method that you have used.

___medication ___prayer/church ___changing my thinking

___alcohol ___exercise ___talk to family/friends

___distraction ___seeing a therapist

___cut down/stop caffeine ___reading about panic/anxiety

___relaxation/yoga/meditation

___avoid situations or activities that bring on panic or anxiety

___other (please list) _____

11a. Which of the above methods have worked best in helping you deal with panic attacks? (Check only one method.)

___medication ___prayer/church ___changing my thinking

___alcohol ___exercise ___talk to family/friends

___distraction ___seeing a therapist

___cut down/stop caffeine ___reading about panic/anxiety

___relaxation/yoga/meditation

___avoid situations or activities that bring on panic or anxiety

___other (please list) _____

11b. What is the overall level of effectiveness of the method listed

above? (Note: Circle a number on the scale below.)

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
Not at all Effective		Slightly Effective		Moderately Effective			Very Effective			Totally Effective

Circle the one phrase that best represents the extent to which you agree with the item. If any of the items concern something that is not part of your experience (e.g., "It scares me when I feel shaky" for someone who has never trembled or had the "shakes"), answer on the basis of how you think you might feel <u>if you had</u> such an experience. Otherwise, answer all items on the basis of your own experience.

1. It is important to me not to appear nervous.

   Very Little A Little Some Much Very Much

2. When I cannot keep my mind on a task, I worry that I might be going crazy.

   Very Little A Little Some Much Very Much

3. It scares me when I feel "shaky" (trembling).

   Very Little A Little Some Much Very Much

4. It scares me when I feel faint.

   Very Little A Little Some Much Very Much

5. It is important for me to stay in control of my emotions.

   Very Little A Little Some Much Very Much

6. It scares me when my heart beats rapidly.

   Very Little A Little Some Much Very Much

7. It embarrasses me when my stomach growls.

Very Little A Little Some Much Very Much

8. It scares me when I am nauseous.

Very Little A Little Some Much Very Much

9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack.

Very Little A Little Some Much Very Much

10. It scares me when I become short of breath.

Very Little A Little Some Much Very Much

11. When my stomach is upset, I worry that I might be seriously ill.

Very Little A Little Some Much Very Much

12. It scares me when I am unable to keep my mind on a task.

Very Little A Little Some Much Very Much

13. Other people notice when I feel shaky.

Very Little A Little Some Much Very Much

14. Unusual body sensations scare me.

Very Little A Little Some Much Very Much

15. When I am nervous, I worry that I might be mentally ill.

Very Little A Little Some Much Very Much

16. It scares me when I am nervous.

Very Little A Little Some Much Very Much

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the number to the right of the statement to indicate how you feel RIGHT NOW, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement.

	Almost never	sometimes	often	almost always
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I am regretful	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am worrying over possible misfortune	1	2	3	4
8. I feel rested	1	2	3	4
9. I feel anxious	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4

12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel high strung	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel confident	1	2	3	4
17. I am worried	1	2	3	4
18. I feel over-excited and rattled	1	2	3	4
19. I feel joyful	1	2	3	4
20. I feel pleasant	1	2	3	4

	Not at all		moderately		Extremely
Please indicate how accurate you think you will be in detecting your heart beats.	1	2	3	4	5

1. Please indicate on the line below the rate at which you think your heart is beating RIGHT NOW.

very slow | slow | average | fast | very fast

|_____|_____|_____|_____|

	Not at all		moderately		Extremely
2. How excited do you feel right now?	1	2	3	4	5
3. How anxious do you feel right now?	1	2	3	4	5

4. Please indicate how strongly you are experiencing each feeling.

None	Mild	Moderate	Severe
0	1	2	3

_____ shortness of breath

_____ dizziness, unsteadiness, or feeling faint

_____ heart racing or pounding

_____ trembling or shaking

_____ sweating

_____ feeling like you were choking or smothering

_____ nausea, stomach upset, or diarrhea

_____ feeling things around you were unreal, or feeling detached from your body

_____ tingling or numbness in parts of your body

_____ hot flashes or chills

_____ pain or pressure in your chest

_____ feeling afraid that you might die

_____ feeling afraid that you might go crazy

_____ feeling afraid that you might lose control

_____ feeling afraid that you might make a fool of yourself

_____ feeling a sweet taste in your mouth

Appendix B

Consent Form

Information to Participants

The experiment which you are being requested to take part in is a research project conducted by Mr. Gordon Asmundson through the Department of Psychology at the University of Manitoba. Mr. Asmundson, a graduate student in psychology at the University of Manitoba, is supervised by Dr. L. S. Sandler from the Department of Psychology at the University of Manitoba.

The purpose of this experiment is to examine the ability of individuals to perceive the beating of their heart. You will be comfortably seated in an armchair for the duration of the experiment. Three electrodes will be applied to your body, one on your right wrist, one on your right ankle, and one on your left ankle. After a brief resting period (approximately 10 minutes) you will be given instructions, on the computer monitor, of the details of the heartrate discrimination task. Basically, this task will require you to indicate if a series of stimulus flashes are the same as, or different from, your current heart rate. This task will be performed during a period of inactivity and following several short periods of rapid breathing. Each of these periods will be indicated and explained in full in the instructions. Following the completion of the instructions you will receive 60 heartbeat discrimination trials, 30 during inactivity and 30 following rapid breathing.

Participant Consent

I,....., have been informed of the nature of the research project by ..... and consent to participate in it. I understand that the project is designed to investigate the ability to perceive the beating of the heart.

Voluntary Participation

There are no risks associated with participation in this experiment. Rapid breathing may produce feelings of anxiety. Otherwise, no stressful, painful, or invasive procedures will be employed. However, if you would like to stop the experiment at any point, please feel free to do so.

Confidentiality

Personal information obtained on questionnaires is confidential and will only be shared with research collaborators of Mr. Asmundson at the University of Manitoba. The evaluation information gathered during the study will be used for research purposes, however any details that may reveal your identity will be excluded from any research reports. Furthermore, personal information will not be used for any assessment purposes.

Signature:_____

Witness: _____

Date: _____

Appendix C

Subject Instructions

Instruction 1

Hello and welcome to the psychophysiology lab of the University of Manitoba. Over the next 45 to 50 minutes you will be presented with a series of blinking character strings ("XXX"'s). You will be asked if these strings are blinking in time with your heartbeat. The following instructions will explain the task required of you.

The computer will provide all instructions during the course of the experiment. When you see the word "RELAX", all that is required is that you rest comfortably. When the feedback period is about to begin, you will be prompted with a "GET READY!" prompt. A series of "XXX"'s will then begin to blink on and off in the middle of the screen. The "XXX"'s will either be blinking in time with your heartbeat, or out of time with your heartbeat. You are to decide if the feedback is a true representation of your heartbeat. When you are asked this question, answer with a "T" for true, or a "F" for false. Following 30 trials of this, you will be required to go through three periods of rapid breathing (hyperventilation), each followed by 10 more trials of the heartbeat feedback, for a total of 30 more trials.

You will complete a total of 60 heartbeat discriminations. Sometimes the blinking of the "XXX"'s will be <u>true</u> representations of your heartbeat, and sometimes they will be <u>false</u>. Please try to determine whether they are true or false without actually taking your pulse.

If you have any questions concerning the procedure, please feel free to ask for clarification; if not, then prepare to begin. Remember, you will be prompted throughout the experiment by the computer.

To familiarize you with the task you are to perform, you will receive a block of practice trials. This consists of 10 trials of the following:

1) a "GET READY!" prompt
2) ten displays of the string of "XXX"'s
3) a question asking if the sequence was flashing in time with your heartbeat, to which you will answer either "T" for true, or "F" for false, and
4) a "RELAX" message.

Please concentrate on your heartbeat when watching the flashing "XXX"'s. The actual experiment will follow the practice trials. Relax, and remember to concentrate on your heartbeat.

Instruction 2

At this point in the experiment you will perform the first of three rapid breathing sessions. During each session of rapid breathing, you will be required to breath at a rate of 30 breaths per minute. The computer will provide a visual and auditory cue to aid you in timing your breathing. Please breath as deeply as possible.

In a few instances the rapid breathing may produce feelings of lightheadedness, tingling, and/or anxiety. THESE FEELINGS ARE ONLY TEMPORARY AND DISAPPEAR RAPIDLY.

Instruction 3

The experiment is now over. Please remain seated and wait for the experimenter to enter the roon and remove the electrodes from you. Any further questions regarding the experiment will be answered at this time.

Thank you for your participation. Good-bye!