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SELECTIVE ATTENTION TOWARD PHYSICAL THREAT IN PATIENTS WITH PANIC DISORDER

by

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A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfillment of the Requirements
for the Degree of

MASTER OF ARTS

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ABSTRACT

Previous research has demonstrated that patients with generalized anxiety disorder and panic disorder show an attentional bias toward threat cues related to their respective disorders. Specifically, it has been shown that generalized anxiety patients are characterized by an attentional bias for social threat cues; whereas, panic patients selectively attend to physical threat cues. The present study is an attempt to advance the state of the literature regarding panic disorder by employing a paradigm which allows for the direct assessment of visual attention relative to threat cues. In total, 18 panic patients and 12 controls were tested. Subjects were required to make a neutral response (button press) to a neutral stimulus (visual probe). The position of the visual probe was altered relative to visually displayed words, which were either threatening (physically or socially) or neutral in content. Probe detection latency was used as a measure of attentive bias toward threat cues. Patients, but not controls, had reduced detection latencies when actively reading physical threat cues compared to social threat cues. The results are consistent with an attentional bias toward physical threat cues in panic patients.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS..... iv

ABSTRACT..... v

LIST OF TABLES..... vii

Page

INTRODUCTION..... 1

PHYSIOLOGICAL MODELS OF PANIC..... 3

Neurotransmitter Abnormalities..... 3
Receptor Dysfunction..... 6
Central Nervous System Dysfunction..... 8
Metabolic Panicogenic Theory..... 12
Separation Anxiety Theory..... 17
General Considerations..... 18

COGNITIVE/PSYCHOPHYSIOLOGICAL MODELS OF PANIC..... 20

SUPPORT FOR A PSYCHOPHYSIOLOGICAL MODEL..... 24

Ideational Content of Panickers..... 24
Perception of Events During Attacks..... 25
Selective Attention Research..... 25

IMPLICATIONS AND HYPOTHESES..... 29

METHOD..... 31

Subjects..... 31
Apparatus and Materials..... 32
Questionnaires..... 32
Threat Cues, Computer, and Visual Probe..... 33
Procedure..... 35
Data Analysis..... 36

RESULTS..... 37

Questionnaire Data..... 37
Probe Detection Latencies..... 39
Effect of Age and Depression Differences..... 42

DISCUSSION..... 43

REFERENCES..... 49

APPENDICES..... 58

LIST OF TABLES

TABLE		PAGE
1.	Table of Means and ANOVAs for Questionnaires by Group.....	38
2.	Mean Probe Detection Latencies in Milliseconds.....	40

INTRODUCTION

Recently panic attacks have been given a central role in the classification of anxiety disorders by the Diagnostic and Statistical Manual of the American Psychiatric Association, Third Edition-Revised (American Psychiatric Association, 1987). A panic attack involves feelings of apprehension and impending doom. These feelings are of sudden onset and associated with an array of somatic and cognitive symptoms. In order to meet the DSM-III-R criteria for panic disorder an individual must have experienced at least 3 unexpected panic episodes within a 3 week period, or 3 episodes that were associated with a minimum of one month of persistent fear of having another attack. In addition, a minimum of 4 recognized symptoms must have developed during one of the attacks. Finally, attacks sustained by known organic factors such as amphetamines, caffeine intoxication, or hyperthyroidism are not considered to be clinical cases (American Psychiatric Association, 1987).

The etiology of panic disorder is controversial, with little agreement among researchers regarding the factors that may be involved. Both physiological and psychophysiological models have been posited. Physiological models contend that some physiological dysfunction operates to produce episodes of panic (e.g., Klein, 1980, 1981; Carr & Sheehan, 1984). Psychophysiological models, on the other hand, are

characterized by the assumption that attacks result from a positive feedback loop between internal cues (e.g., bodily sensations or cognitive events) that a person associates with immediate threat to their well-being (Clark, 1986; Ehlers, Margraf, & Roth, 1988).

PHYSIOLOGICAL MODELS OF PANIC

Over the last 10 years interest and research regarding the physiological correlates of panic disorder has exploded. Consequently, a number of physiological models of panic disorder have been posited. It has been proposed that panic may be the result of a number of physiological dysfunctions: These include abnormalities in neurotransmitter and receptor function, autonomic nervous system regulation, central nervous system function, basic metabolism, and biological thresholds of activation for innate alarm mechanisms. The basic tenents of each of these theoretical positions have been supported to some extent in the literature. Each model is discussed below.

Neurotransmitter Abnormalities

Disturbances of sympathetic nervous system (SNS) function have been implicated in the pathogenesis of anxiety disorders (Redmond, 1979). Activation of the SNS is associated with elevations in plasma catecholamines; therefore increased SNS activity in persons with anxiety disorders should be associated with elevated plasma epinephrine (E) and norepenephrine (NE). Evidence for this hypothesis is mixed.

Rubin (1984), in reviewing the neuroendocrine aspects of panic disorder, draws the conclusion that there are elevated levels of both E and NE in panic patients compared to controls. Although this conclusion is

unwarrented on the basis of empirical evidence cited by Rubin (no studies cited refer to the relationship between catecholamine levels and panic disorder) it is tenable on other grounds.

For example, Nesse, Cameron, Curtis, McCann, and Huber-Smith (1984) reported elevated levels of E and NE in patients with panic disorder. On the other hand, Ballinger et al. (1984) found that in a sample of agoraphobics only NE levels were elevated in comparison to controls; there were no significant differences in E levels between the groups. Similarly, Nesse et al. (1985) found a significant difference only in the levels of NE excreted by panickers compared to controls.

There are several studies that have failed to replicate findings of elevated catecholamine levels in panic patients. Studies by Carr et al. (1986), Gaffney, Fenton, Lane, and Lake (1988), and Liebowitz et al. (1984b) have all failed to find differences in plasma catecholamine levels between groups of panickers and controls.

There are a number of explanations for the discrepant results. For example, lack of control for degree of obesity, food consumption prior to investigation, and site of catecholamine measurement all can influence measured catecholamine levels. These factors were controlled for in a study by Enrique, Hollifield, Katon, Wilkinson, and Veith (1987). All subjects were age

and weight matched, and had fasted for 24 hours prior to the onset of the study. Catecholamines were sampled from arterialized venous blood, a site which is thought to be most representative of levels of E and NE released from the adrenal medulla and postganglionic sympathetic nerves, respectively. The major finding was that plasma E levels were significantly higher in panic patients than controls at rest, whereas there were no differences in NE levels. Thus, the results of this well controlled study are in the opposite direction of studies with less control; namely E levels were found to be elevated in panickers rather than NE or both E and NE.

In view of the inconsistencies in the literature, no firm conclusions can be drawn regarding the role of catecholamines as etiologic agents in panic disorder. Replication of studies is required. In addition, future researchers will need to control for all factors that may affect catecholamine levels. Gorman, Liebowitz, and Klein (1984) note that increases in catecholamine levels may merely reflect levels of anticipatory anxiety. In other words, the increased concentrations of catecholamines evident in some studies may not necessarily precede panic episodes, but may be a response to the episode. Therefore, until research can demonstrate which of these scenarios is correct, and until well controlled replications are conducted, no conclusions regarding the role of catecholamines in panic disorder

can be made.

Receptor Dysfunction

Another model of panic disorder maintains that the problem is the result of abnormally sensitive or hyperactive beta-adrenergic receptors. Symptoms commonly experienced by panickers, such as tachychardia, sweating, and palpitations, suggest the possibility that autonomic-sympathetic discharge may be a critical underlying substrate of panic disorder. Specifically, these manifestations suggest hyperactivity at beta receptor sites of the adrenergic neuronal system. 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 before 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 selectively stimulate and/or block beta receptor sites via administration of various pharacological agents. For example, Schmidt and Elizabeth (cited in Gorman et al., 1984) infused isoproterenol, a beta agonist (i.e., stimulant), into a group of patients with panic disorder and a group of controls. It was found that the infusion

induced panic in the patients but not the controls. Specifically, there was evidence of tachycardia in the patients but not the controls.

Also, Rainey et al. (1984) compared the effects of infused isoproterenol, sodium lactate, and placebo in patients suffering from panic disorder. Sodium lactate, like isoproterenol, is a beta agonist. Ninety-one percent of panickers and 30 percent of controls had a panic attack during lactate infusion. Similarly, 73 percent of panickers and 20 percent of controls experienced panic during isoproterenol infusion. Only 36 percent of panickers and no controls experienced attacks during placebo infusion. In addition, patients reported the attacks experienced during both sodium lactate and isoproterenol infusion to be very similar to their naturally occurring panic episodes. The results of this study support the thesis that beta-adrenergic hypersensitivity may be linked to panic disorder.

Despite these positive results, beta agonist infusion studies can be criticized for several reasons. Gorman et al. (1984) note that these studies are not typically conducted under double-blind conditions. Therefore, subjects are aware that they are receiving a pharmacological agent. It may be that such knowledge differentially affects panickers relative to controls, thus leading to increased arousal and subsequent panic. Furthermore, there is a body of literature which contradicts

the results of the studies mentioned above.

Nesse et al. (cited in Gorman et al., 1984) designed an experiment that measured the ability of isoproterenol to bind to beta-adrenergic receptors. It was found that in both panic disorder patients and controls there was no evidence of hypersensitive beta-adrenergic receptors. In other words, isoproterenol bound to receptors equally in both panickers and controls. Also, Gorman et al. (1983) administered propranolol, a beta antagonist, in an attempt to block panic episodes induced by sodium lactate infusion in panic patients and controls. Although there was significant beta receptor blockage, the patient group experienced panic related symptoms and panic attacks during lactate infusion.

Thus, the literature appears inconclusive with respect to the role of beta-adrenergic hypersensitivity in panic disorder. It would appear from the above discussion that beta-adrenergic hypersensitivity is neither necessary nor sufficient in panic disorder.

Central Nervous System Dysfunction

Many of the symptoms of panic disorder appear to reflect central nervous system arousal. As a result there has been considerable conjecture and speculation about the site of the arousal. The most prominant candidate is a major nucleus of the noradrenergic system, the locus ceruleus (LC). The LC is located in the pons of the brain stem and contains approximately 50 percent

of all neurons in the central nervous system that utilize NE as a neurotransmitter (Grant & Redmond, 1981). Also, the LC has numerous afferent fibres which project to many areas of the brain (e.g., cerebral cortex, brain stem, reticular system, the pain sensitive neurons in the dorsal horn of the spinal cord, and the limbic system) via numerous pathways (Grant & Redmond, 1981).

The basis for theories that link the LC to panic disorder comes from a series of experiments conducted by Redmond (1979). Specifically, Redmond stimulated the LC of stump-tailed monkeys with either electrical currents or pharmacological agents (i.e., opiates). It was found that both forms of stimulation led to a reaction that was very similar to that displayed by monkeys in situations of natural threat. The implication is that the reaction exhibited by the monkeys in the experimental situation may be analogous to panic episodes suffered by humans.

Physiological investigation into the LC connection in humans has occurred mainly in the context of drug infusions that increase LC activity. Drugs that have such an effect interact with the $alpha_2$-adrenergic receptor of the LC. More specifically, drugs that stimulate this receptor decrease LC activity and NE secretion through a negative feedback mechanism (Andrade & Aghajanian, 1984). Inhibition of the $alpha_2$ receptor leads to increased LC firing.

Yohimbine is one of few biochemical agents able to pass through the blood-brain barrier to act on the alpha$_2$ receptor of the LC. Thus, Charney, Heninger, and Breier (1984) investigated the reactions of patients with panic disorder to injections of yohimbine and placebo. Results indicated that during yohimbine injections panic patients rated themselves as having high anxiety and nervousness which was qualitatively similar to that experienced during naturally occurring attacks. The panic patients also experienced significant increases in somatic activity such as palpitations, hot and cold flashes, tremors, blood pressure, and pulse rate. Control subjects experienced only minor changes in somatic activity which were not accompanied by subjective nervousness or anxiety. Thus, agents that stimulate LC activity, such as yohimbine, appear to increase reports of anxiety and related symptoms in those with panic disorder.

Other strategies have been used to investigate the connection between the LC and panic disorder. For example, Charney and Heninger (1986) investigated the effect of chlonidine hydrochloride, an alpha$_2$-adrenergic receptor agonist that decreases noradrenergic function, on plasma 3-methoxy-4-hydroxyphenylglycol (MHPG), cortisol, growth hormone levels, blood pressure, heart rate, and behavior in panic patients and controls. The major finding was that chlonidine induced greater

declines in plasma MHPG levels and diastolic blood pressure in panic patients compared to controls. Also, patients exhibited smaller increases in growth hormone and behavioral indices (e.g., drowsiness) than did controls. When these results are considered in conjunction with the results of Charney et al. (1984) it seems plausible that panic disorder patients have regulatory problems with alpha$_2$-adrenergic receptors and noradrenergic cardiovascular mechanisms. The authors argue that in a sub-group of panickers, those with decreased levels of MHPG, aberrant regulation of alpha$_2$-adrenergic receptors may lead to increased lability of the noradrenergic system. The result is greater sensitivity to provocation of panic.

There is at least one study that directly suggests alpha$_2$-adrenergic functioning is probably not increased in patients with panic disorder. Grunhaus, Gloger, Birmacher, Palmer, and Ben-David (1983) measured prolactin levels and blood pressure in panic patients and controls following a cold pressor test. There were no significant differences between patients and controls in prolactin or blood pressure responses to the cold pressor test. The cold pressor response is thought to be mediated by stimulation of alpha receptors which, in turn, leads to elevation of blood pressure. Since no differences were found between patients and controls in resting or stimulated blood pressure, the

authors conclude that alpha$_2$-adrenergic function is not increased in patients with panic disorder.

Barlow (1988) provides a comprehensive review of the literature regarding the role of the LC in panic disorder. The studies discussed above are representative of that literature. In general, it appears that the LC: a) may be the primary generator of panic attacks, b) may cause panic by overstimulation from other physiological systems, c) may be only one of several systems responsible for panic, and d) may not be involved in panic disorder. It can be concluded that the existence of conflicting data make uncritical acceptance of the LC theory premature. In addition, it is apparent that there is a need for further research in the area.

Metabolic Panicogenic Theory

Carr and Sheehan (1984) argue that panic attacks occur spontaneously and are an entity distinct from other anxiety states. Further, they argue the the mechanism through which panic occurs is purely metabolic. It is lactate, the end-metabolic product of glucose metabolism, that appears to be the best metabolic marker of panic disorder.

In one of the first studies with lactate, Pitts and McClure (1967) found that lactate infusion produced panic attacks in a majority of anxiety neurotics but not controls. Using current DSM-III-R criteria, these patients would be diagnosed with panic disorder.

Recently, many studies have been published that confirm Pitts and McClure's (1967) findings (Appleby, Klein, Sacher, & Levit, 1981; Liebowitz et al., 1984a, 1984b; Rifkin, Klein, Dillon, & Levit, 1981)

Liebowitz et al., (1984b), for example, found that approximately 75 percent of panic disorder patients experienced a panic attack during lactate infusion. No control subjects experienced panic during infusion. In addition, patients rated the attacks experienced during lactate infusion to be very similar to naturally occurring attacks.

The results of Liebowitz et al. (1984) appear to support the metabolic theory of panic. However, the results can be criticized. First, panic patients had higher resting heart rates than did controls. In addition, those 75 percent of patients who experienced panic attacks during lactate infusion had higher heart rate and diastolic blood pressure at baseline than those who did not have attacks. Therefore, it is possible that the lactate infusion did not differentially affect those that did and did not experience panic attacks, but simply caused increases of equal magnitude from baseline. Since the baseline of panickers were higher initially, the increase caused by lactate infusion may have promoted the anxiety attack. Second, the resting partial pressure of carbon dioxide (pCO_2) was lower in patients than controls, suggesting that the panickers

may be in a chronic state of respiratory alkalosis. This indicates that those who experienced panic attacks were aroused prior to infusion, thus increasing the probability of having an attack. Finally, it is not known whether the infusions specifically increased anxiety or a more global discomfort of which anxiety is only a part. Thus, the specific effects of lactate infusion apear to be unclear.

Margraf, Ehlers, and Roth (1986b) reviewed the methods and results of 13 lactate studies, making criticisms similar to those stated above. In general they noted that the studies were characterized by serious methodological flaws, lack of specificity and sensitivity, and failure to consider cognitive processes. Thus, conclusions that panic patients respond differentially to lactate infusions because of physiological differences may be premature.

In fact, a number of researchers suggest that cognitive factors may be more important in the mediation of panic than physiological ones. Gaffney et al. (1988) found no difference between patients and controls in autonomic nervous system or cardiopulminary functioning in response to lactate infusion. However, panic patients were emotionally overresponsive towards the physiological changes induced by the lactate infusion. The authors conclude that cognitions may be more relevant than physiological factors to the occurrence of panic.

Similarly, Freedman, Ianni, Ettedgui, Pohl, and Rainey (1984) found that cognitions were central to lactate induced panic. Panic patients and controls were given double-blind infusions of lactate, isoproterenol, and placebo. Lactate and isoproterenol induced attacks occurred with greater frequency in patients than controls. Patients also reported greater subjective anxiety, but only during lactate infusion. In addition, panic patients exhibited higher levels of sympathetic activity; however, no physiological measure reliably differentiated panic attacks from non-attack periods. As a result, Freedman et al. (1984) suggest that peripheral physiological responses are neither necessary nor sufficient for the occurence of panic. Rather, it seems that patients who report symptoms do so in order to have the researcher terminate the infusion and reduce their fear of further arousal. Based on this premise, panic disorder could be classified as a syndrome of phobic responses to intense anxiety rather than a response to irregular lactate metabolism.

van der Molen, van den Hout, Vroemen, Lousberg, and Griez (1986) provide the strongest evidence for the notion that cognitive factors are key to the responses associated with lactate infusion. These researchers were interested in delineating the effects of lactate infusion in two differentially instructed groups of normal subjects. A double-blind, placebo controlled,

cross-over procedure was employed. Some subjects were told that infusions would produce anxiety, whereas others were told that they would produce a pleasant excitement. In the former condition subjects reported anxiety following lactate but not placebo infusion. There was no change in mood in the later condition after either infusion. Since anxiety did not occur to placebo, physical symptoms induced by lactate infusion are important in the occurence of anxiety. However, situational cues, expectancy, and cognitions can be said to be the more important mediators of anxiety. Furhtermore, the results of this study bring into question the effects of instructions, not only on normal controls, but also on panic patients. Different experiments have used different instructions, and often an experiment will have different instructions for the panic patients and controls. Therefore, the results of these experiments may be determined by the expectancies and cognitions arising from instructions rather than the infusion of lactate.

The metabolic panicogenic theory is popular. Nevertheless, it is limited by numerous flaws and its supporting literature can be criticized on several grounds. Apparently, cognitions are more important to the occurrence or nonoccurrence of panic than metabolic factors.

Separation Anxiety Theory

A biologically based theory of the mechanism of panic disorder has been formulated by Klein (1980). Simply stated, Klein proposed that panic results from a dysfunction of the mechanism that underlies normal human separation anxiety. Consonant with Bowlby (1973), the mechanism is thought to be genetically programmed and biologically determined rather than learned.

There are several lines of evidence that link separation anxiety to panic disorder. First, Klein (1964) has indicated that as many as 50 percent of all patients with panic disorder or agoraphobia report having had pathological separation anxiety as children. Second, drugs that are effective in the treatment of panic disorder have also proven to be useful in reducing separation anxiety (Gittelman & Klein, 1984; Klein, 1980).

The key argument advanced by Klein for the validity of his model is that of drug specificity. This argument is based on evidence showing that certain drugs (e.g., tricyclic antidepressants, imipramine, monoamine oxidase inhibitors) show clear benefits in the treatment of panic (Garakani, Zitrin, & Klein, 1984; Klein, 1984), but do not affect anticipatory anxiety On the other hand, drugs that are helpful in reducing anticipatory anxiety (e.g., benzodiazepines) do not effectively block panic attacks (Klein, 1984).

This evidence, coupled with the finding that drugs effective in the treatment of panic are also effective in treating separation anxiety, form the basis of Klein's model. Since panic and anticipatory anxiety are affected differentially by these drugs, they are considered to be qualitatively different disorders.

However, several studies have yielded results that are inconsistent with the drug specificity argument. For example, several researchers have found that certain benzodiazepines (e.g., alprazolam, diazepam) can be effective in the treatment of panic (Shader, Goodman, & Gever, 1982; Sheehan, 1982; Noyes et al., 1984). These results render the drug specificity argument inconclusive.

General Considerations

Although each physiological model of panic disorder is unique, they do overlap to some extent. The one characteristic common to all of these models is that panic attacks are assumed to occur spontaneously. That is, attacks are believed to occur in the absence of specific triggering cues or events. Such an assumption is necessary in order for attacks to be attributed solely to a physiological dysfunction.

The literature, however, does not seem to support the thesis that panic attacks are solely the result of a physiological dysfunction. Cognitive variables which are deemed unimportant by most physiological

researchers seem to be the most important factors mediating the probability of a panic attack occurring. This is evident in studies of beta-adrenergic hypersensitivity, $alpha_2$-adrenergic receptor functioning, and metabolic activity. Further, there is a body of literature which suggests that the assumption that attacks occur spontaneously is unfounded. This literature and the models upon which it is based are presented in the following section.

COGNITIVE/PSYCHOPHYSIOLOGICAL MODELS OF PANIC

Recently, a cognitive model of panic disorder has been proposed by Clark (1986). The major premise of the model is that catastrophic misinterpretation of bodily sensations, mainly those involved in normal anxiety responses, leads to panic. Initially, external or internal stimuli which are perceived as threatening provoke a mild state of apprehension. This state is typically accompanied by a wide range of bodily sensations. Catastrophic misinterpretation of these anxiety-produced sensations leads to a subsequent increase in apprehension which, in turn, augments bodily sensations. If this cycle continues, it will culminate in a full-blown episode of panic.

In recognition of the interaction between psychological and physiological factors it may be preferable to label Clark's (1986) model a psychophysiological model. Clearly, it is the interaction of cognitions and bodily sensations that are proposed to result in panic.

Ehlers, Margraf, and Roth (1988) offer another psychophysiological model of panic that is more general than that of Clark (1986). According to the model, cognitive or physiological changes occur as a result of various situations (e.g., physical effort, caffeine intake, stress, emotionality). The person perceives these changes. As suggested by Clark (1986), the changes are associated with threat and danger. The person responds to the

perceived threat by becoming anxious. The anxiety leads to physiological changes, bodily sensations and/or cognitive symptoms (e.g., apprehension). If appraisal of the symptoms leads to further increases in anxiety, then panic may result.

Both models posit that a positive feedback between cognitive and physiological variables leads to panic. Thus it is possible for these models to accomodate a wide variety of agents as potential triggers of a panic episode. Triggers of panic may be: a) internal or external stressors that increase the likelihood of threatening cognitive or physiological events, b) individual biological (e.g., noradrenergic dysfunction) or psychological (selective attention to threat cues) predispositions, and/or c) situational variables that influence whether bodily sensations are associated with fear and danger (e.g., coping resources, availability of explanations for sensations). Unlike the physiological models which limit the cause of panic to a disturbance of physiology, the psychophysiological models explain panic in terms of both cognitive and physiological events. As a result, psychophysiological models provide a comprehensive account of all characteristics of panic disorder, including those central to the physiological models.

For instance, it was previously cited that proponents of physiological models assume that panic attacks occur

spontaneously. The term "spontaneous" is used in several ways in the literature. First, "spontaneous" is used to refer to an attack that is unpredictable to the sufferer. In this case empirical evidence can be cited to support such use: Barlow et al. (1985) found that persons experiencing unpredictable (spontaneous) panic attack report a larger number of, and more severe symptoms than those having cued panic. Alternatively, the term is used to refer to a biological dysfunction that is independent of psychological factors (Klein, 1980; Sheehan, 1982). This notion of spontaneity has not been empirically investigated and is based solely on clinical impression; therefore, some authors discourage use of the term in this manner (e.g., Margraf, Ehlers, & Roth, 1986a).

Psychophysiological models propose that attacks that are unpredictable (i.e., spontaneous) are the result of some internal anxiety cue, such as a bodily sensation or cognitive event, that the person associates with personal danger. This results in heightened bodily sensations which are not readily distinguishable from the ensuing attack. Thus, attacks that appear spontaneous and "out of the blue" may be triggered by cues that are not readily apparent.

There is a growing body of evidence suggesting that panic can be precipitated by internal cues. Both somatic sensations and cognitive events have been investigated.

This literature is reviewed below.

SUPPORT FOR A PSYCHOPHYSIOLOGICAL MODEL

Ideational Content of Panickers

Empirical evidence suggests that persons with panic disorder typically interpret bodily sensations as being personally dangerous. Hibbert (1984) compared the ideational consitution of patients with generalized anxiety disorder to that of those with panic disorder. Panic patients were more likely to have thoughts oriented toward illness, death, or losing control than were anxiety patients. Groups did not differ in frequency of thoughts concerned with anticipation of social embarrassment, thus indicating that cogitation about physical concerns was a primary distinction between groups. In discussing the results of his study, Hibbert stated that "the ideational content in those experiencing panic attacks can be understood as a reaction to the somatic symptoms, a connection insisted upon by all but 2 of the patients" (Hibbert, 1984, p. 622). In other words, panic patients systematically misinterpret their somatic experiences as being personally dangerous.

Recent studies support the conclusion of Hibbert (1984). Panickers have been found to report more fear of bodily sensations (Chambless, Caputo, Bright, & Gallagher, 1984; McNally & Lorenz, 1987) and to interpret the bodily sensations as indicative of impending danger (Clark et al., 1988; Foa, 1988; van den Hout, 1988).

Perception of Events During Attacks

Further evidence supporting the psychophysiological models comes from the panic patient's perception of the sequence of events during an attack. Although only two studies have questioned patients about their perception of events (Hibbert, 1984; Ley, 1985), both found that physical symptoms were typically the first thing noticed. Specifically, Hibbert (1984) found that 53 percent of panic patients, as opposed to 0 percent of nonpanic patients, reported physical sensations as the first thing noticed during an attack. Similarly, Ley (1985) reported that the predominance of bodily sensations experienced during a panic attack occurred before feelings of fear in 90 percent of his sample of agoraphobic patients. These findings suggest that physical sensations occur prior to the actual episode of panic and are cognitively misinterpreted as dangerous by panic sufferers. Presumably, it is the misinterpretation of these internal cues (i.e., physical sensations) which leads to an episode of panic.

Selective Attention Research

There is a body of evidence that allows for the hypothesis that internal cues such as thoughts or ideas which are perceived and appraised as threatening may lead to a panic attack. A series of recent studies (MacLeod, Mathews, & Tata, 1986; Mathews & MacLeod, 1985; Mathews & MacLeod, 1986) have demonstrated that

threat cues are more likely to be perceived and appraised as threatening by anxious patients than normal controls. Mathews and MacLeod (1985) found that anxious patients were slower than controls at naming words related to physical and social threat compared to neutral words on a modified Stroop color-naming task. Subsequently, Mathews and MacLeod (1986) attempted to determine whether anxiety patients' threat cue related performance deficit could occur without awareness. A dichotic listening paradigm in which stories were presented in an attended channel and threat-related material in an unattended channel was employed. Results indicated that anxious patients were slower than normal controls at performing a simultaneous reaction time task when unattended words were threatening in content. The authors interpreted this as indicating the existence of a pre-attentive bias toward threat cues in anxious patients. On the basis of these two studies, they concluded that threat cues are processed differently by anxious patients and normal controls and that the difference seems to be dependent on a pre-attentive bias operating prior to awareness.

MacLeod et al. (1986) examined the possibility that the results of Mathews and MacLeod (1986) were not necessarily due to a pre-attentive bias toward threat cues. An alternative explanation is that both neutral and threatening cues are processed equally by the anxious

patients, but the presence of threat cues increases their anxiety state to a level that impairs performance. Likewise, it is plausible that all subjects extract some degree of partial information from the task, and then guess at the identity of a word in a mood-congruent fashion. The effect observed by Mathews and MacLeod (1986) would occur if anxious patients responded to threat material and controls to neutral material. If this was the case, then a mood-dependent response bias would be the correct explanation of the effect, not an attentional bias toward threat.

To control for these alternative explanations MacLeod et al. (1986) used a paradigm which allowed for a direct measurement of the distribution of visual attention. Subjects were required to make a neutral response (i.e, button press) in reaction to a neutral stimulus (i.e., visual probe appearing on a computer monitor). The position of the visual probe was altered relative to visually displayed words, which were either threatening or neutral in content. Probe detection latencies were recorded in order to assess the impact of threat cues on the disribution of visual attention. Results indicated that anxious patients shifted attention to threat words, whereas normal controls shifted away from such material. The authors interpret this result as indicative of the existence of an attentional bias for threat cues.

Recently, Ehlers, Margraf, Davies, And Roth (1988) investigated attentional biases in panic disorder patients and persons suffering infrequent panic episodes. In their first experiment these researchers used a modified Stroop color-naming task similar to that employed by Mathews and MacLeod (1985). The threat words used were related to physical threat, separation, and embarrassment. Panic patients, but not controls, were slower at color naming physical threat words. There were no differences between the groups for either separation or embarrassment conditions. In a second experiment with infrequent panickers, responses to color naming physical threat words were slower in the panickers than controls. Again, the groups responses to neutral words did not differ. These results provide preliminary evidence for an attentional bias for physical threat cues in panic patients and infrequent panickers.

Similarly, MacLeod et al. (1986) have shown that anxiety patients exhibit an attentive bias toward threat cues. In addition, their research has eliminated alternative interpretations of the results. Nevertheless, their results cannot be generalized from their patient population to those suffering from panic disorder. At present, therefore, the results of Ehlers, Margraf, Davies, and Roth (1988) are open to interpretations unrelated to attentive biases.

IMPLICATIONS AND HYPOTHESES

The studies conducted on panic disorder patients' ideational content, their perception of events during attacks, and their apparent attentive bias toward physical threat provide strong support for psychophysiological models of panic. The work of Ehlers, Margraf, Davies, and Roth (1988) holds strong implications for the positive feedback loop suggested in psychophysiological models of panic. It seems reasonable to speculate that the selective processing of physical threat cues begins the cycle leading to a panic attack. That is, one could argue that cues, be they thoughts, ideas, or somatic sensations, which are perceived as threatening by an individual, lead to apprehension which, in turn, leads to internal sensations of anxiety and ultimately to panic. However, before such speculation can be substantiated, it must be demonstrated that the effects discovered by these researchers cannot be accounted for by alternative explanations such as mood-congruent response bias.

Thus, the present study was designed to investigate the relationship between panic disorder and the existence of an attentional bias toward physical threat using a paradigm that allows for a direct measurement of how visual attention is distributed. This paradigm, similar to that used by MacLeod et al. (1986), controls for interpretative problems by requiring subjects to

make a neutral response (i.e., key press) to a neutral stimulus (i.e., visual probe). Specifically, on trails of interest, word pairs consisting of a threatening and neutral word are presented on a visual display monitor, Distribution of visual attention is measured by a secondary task involving detection of a visual probe. This probe may appear in the spatial location of either the threatening or neutral word, following word offset. Probe detection latency, which has been shown to be a sensitive measure of visual attention (e.g., Navon & Margalit, 1983), is recorded by having subjects press a button upon perception of the probe. Latency measures on trials of interest allow for determination of the impact of threatening material on visual attention.

The following hypotheses were proferred;

1. Panic patients, but not controls, will shift attention toward threat cues resulting in reduced detection latencies for visual probes that follow threat cues compared to those following neutral cues.

2. Panic patients will have significantly shorter probe detection latencies for physical threat cues compared to social threat cues, whereas controls will not.

METHOD

Subjects

Patients were recruited from the anxiety clinic of the St. Boniface Hospital, Winnipeg, Manitoba. Appendix A outlines the request for participation that was circulated to potential patients. Eighteen patients (13 females, 5 males) in total were tested. All patients were diagnosed by a qualified clinician and met the DSM-III-R criteria for panic disorder. Any patients exhibiting marked phobic avoidance, obsessional behavior, or medical problems were excluded from the study.

An additional 12 subjects (9 females, 3 males) were selected from an introductory psychology subject pool at the University of Manitoba to serve as controls. Any subjects reporting infrequent panic attacks (i.e., at a frequency less than required to meet DSM-III-R criteria for panic disorder) or having medical ailments were excluded from the study.

Due to the different nature of the institutions from which the two subject groups were selected, it was difficult to match for age, although the attempt was made. The mean ages for the patient and control groups were 35.9 (SD=8.9) and 26.8 (SD=7.7) respectively. The age difference between groups was significant, $F(1,28)=8.39$, $p<.01$. The age range for the patient group was 24 to 55 years; for the control group it was 19 to 45 years.

Apparatus and Materials

Questionnaires

All patients and subjects completed an inventory comprised of a number of self report questionnaires. The primary instrument used to screen the occurrence of panic in the nonpatient group was a questionnaire derived from the Panic Attack Questionnaire (Sandler, Asmundson, Wilson, Ashton, & Ramsum, 1987). This questionnaire probes the occurrence, severity, frequency, and symptoms associated with panic attacks (see Appendix B). The Beck Depression Inventory (BDI, Beck & Beck, 1972) was given in order to obtain an indication of the level of depression within each group. Indices of trait anxiety and state anxiety were obtained from the State-Trait-Anxiety-Inventory (STAI, Spielberger, Gorsuch, & Luschene, 1970). Finally, the Agoraphobic Cognitions Questionnaire (ACQ) and the Body Sensations Questionnaire (BSQ, Chambless et al., 1984) were given in order to assess differences between groups in ideational constitution and experience of bodily sensations during periods of anxiety.

Threat Cues, Computer, and Visual Probe

Forty-eight words, 24 related to physical threat and 24 related to social threat, were extracted from previous research by MacLeod et al. (1986). These particular words were selected because of their threatening content. Appendix C lists the words used. All threat words were paired with a length and frequency matched neutral (i.e., nonthreatening) word in order to create 48 key word pairs. Filler material was comprised of 240 neutral word pairs, randomly created and length matched.

An Epson micro-computer and visual display monitor were used to present word pairs. Words pairs were presented in the center of the monitor separated vertically by 3-cm. Subjects were permitted to adjust the contrast and brightness of the monitor to a level that was comfortable. Each word pair was displayed for a duration of 500-ms. A visual dot probe (i.e., a small circular point created by the computer) appeared on 96 of 288 trials, approximately 25-ms following word display offset. Fifty percent of probed trials involved one of the 48 key word pairs. The probability of the threat word appearing in either the upper or lower half of the visual monitor was equated. Similarly, the probe was presented with equal probability in either the same spatial position as the threat word or the neutral word. Therefore, on critical trials, two factors were independently varied; the position of the threat

word and the position of the subsequent visual probe. By varying both threat word position and probe position four possible conditions for each subject were created with 12 of the 48 key trials appearing under each condition. These conditions were: a) threat word and probe in the upper area of the monitor, b) threat word in the upper area and probe in the lower area, c) threat word and probe in the lower area, and d) threat word in the lower area and probe in the upper area. Word pair presentation was balanced across conditions.

On trials in which the probe appeared, the subject was required to respond in order to offset the probe. Each trial without the probe was followed by another word pair 1-s after offset of the previous word pair.

Procedure

All subjects were tested individually. Informed consent was obtained from each subject after the nature of the procedures had been explained. Appendix D outlines the consent form provided to the subjects.

The computer task and questionnaires were completed in one session. During the first half of the session, subjects were invited into the laboratory, seated in front of a visual display monitor and keyboard, and given task instructions (Appendix E). Subjects were told that word pairs would appear on the monitor, and were asked to read aloud the top word of each pair as soon as it appeared. Subjects were also advised that on some trials a visual dot probe would appear immediately following word offset in the position of either the top or bottom word. In response to this probe, subjects were asked to press the spacing bar of the computer keyboard as quickly as possible. A practice session of 6 trials (all with probes) followed, and any questions regarding procedure were addressed prior to beginning the actual experiment. The subjects then received 288 test trials. Subsequently, the computer keyboard was removed and subjects completed all questionnaires. Upon completion, the subjects were debriefed and all questions were answered.

Data Analysis

One-way analysis of variance was used to analyze questionnaire data. A mixed factor analysis of variance with repeated measures was used to analyze probe detection latency data gathered from the critical conditions. The between-subjects variable was disorder classification (panic patient vs control), and the within-subjects variables were threat type (physical vs social), threat position (upper vs lower area), and probe position (upper vs lower area).

RESULTS

Questionnaire Data

In order to investigate differences between groups in depression, state anxiety, trait anxiety, agoraphobic cognitions, and bodily sensations during anxiety analyses of variance were performed on the relevant questionnaire items. Table 1 contains univariate statistics and group means for each questionnaire.

As expected, the panic patients scored higher than controls on both state, $F(1,28)=14.67$, $p<.001$, and trait anxiety, $F(1,28)=26.14$, $p<.001$. As well, patients reported more agoraphobic cognitions, $F(1,28)=10.63$, $p<.005$. and bodily sensations, $F(1,28)=12.47$, $p<.005$, during periods of anxiety. Although patients scored higher than controls on depression (BDI), $F(1,28)=11.36$, $p<.005$, it is important to note that the primary diagnosis for all patients was panic disorder.

Table 1

Table of Means and ANOVAs for Questionnaires by Group

Questionnaire Measure	Mean Score		F	Prob.
	Patient	Control		
STAI state anxiety score	53.9	36.1	14.67	.0007*
STAI trait anxiety score	52.6	34.3	26.14	.0000*
ACQ score	2.35	1.52	10.63	.0029**
BSQ score	2.65	1.80	12.47	.0015**
BDI score	14.1	3.6	11.36	.0022**

Note: STAI = State-Trait-Anxiety-Inventory; ACQ = Agoraphobic Cognitions Questionnaire; BSQ = Body Sensations Questionnaire; BDI = Beck Depression Inventory.

degrees of freedom for all tests were 1 and 28.

* $p < .001$, ** $p < .005$.

Probe Detection Latencies

The repeated measures analysis of variance revealed a main effect for probe position, $F(1,28)=9.81$, $p<.005$. Probe detection latencies were shorter when the probe appeared in the upper area of the display than when it appeared in the lower area (489-ms vs 523-ms). In addition, there was a main effect of threat type, $F(1,28)=15.75$, $p<.001$. Probes were responded to faster when they followed a word pair consisting of a physical threat and neutral word than when they followed a pair comprised of a social threat and neutral word (488-ms vs 524-ms). The threat type main effect was modified by a significant three-way interaction of Group x Threat type x Threat position, $F(1,28)=4.95$, $p<.05$. The mean probe detection latencies for the factors involved in this interaction are shown in Table 2. Summary analysis of variance statistics are provided in Appendix F.

Baron and Treimann (1980) have indicated circumstances under which the interpretation of interactions involving a between-groups factor may pose problems; specifically, if groups operate at different levels of the independent variable. In the current study, groups did not differ in detection latencies, $F(1,28)=0.04$, thus there should be no interpretive problems with these data. Therefore, post-hoc pairwise comparisons of the means involved in the Group x Threat type x Threat position interaction were carried out using the Tukey Honestly Significant

Table 2

Mean Probe Detection Latencies in Milliseconds

Group/threat type	Threat in upper area[a]	Threat in lower area[b]
Panic patients		
Physical threat	481	505
Social threat	540	510
Control subjects		
Physical threat	481	482
Social threat	507	534

[a]Subjects are actively reading the threat cue when it appears in the upper area.

[b]Subjects are actively reading a neutral word when threat cues appear in the lower area.

Difference (HSD) procedure (Kirk, 1982). When the threat word appeared in the upper area (where subjects were actively reading), panic patients responded to probes more quickly if the threat term was physical as opposed to social (481-ms vs 540-ms), HSD=52.59, $p < .05$. Control subjects' responses to probes following physical threat presented in the upper area did not differ significantly from their response to probes following social threat presented in the upper area. Neither group exhibited differences in response to probes following physical or social threat presented in the lower area (which was not attended to). Furthermore, there were no between-group differences in response to probes following physical threat or social threat presented in either location. Thus, panic patients appear to have an attentional bias for physical threat cues. Controls, on the other hand, respond in similar fashion regardless of the nature of the threat.

Effect of Age and Depression Differences

As previously stated, patients and controls did not only differ in the experience of panic, but also in age and level of depression. It is possible that these differences affected the observed results. However, when age and level of depression were used as covariates in an analysis of covariance of probe detection latencies, the Group x Threat type x Threat position interaction remained significant. Thus, group differences in age and depression do not appear to be related to the observed effects.

DISCUSSION

The panic disorder literature indicates clearly that panic patients differ from others in their experience of anxiety. It has been suggested that panickers differ from others in terms of predisposing physiological factors. For example, neurotransmitter abnormalities (Enrique et al., 1987; Nesse et al., 1984), receptor dysfunction (Rainey et al., 1984), hyperactivity of the LC (Charney & Heninger, 1986; Charney et al., 1984), and abnormal metabolism (Carr & Sheehan, 1984) have all been suggested as predisposing mechanisms of panic. However, it appears that physiological differences are not sufficient for panic: Psychological factors seem to be necessary for the occurrence of panic.

Psychologically, panickers differ from others in the way they interpret bodily sensations as indicative of impending danger (Clark et al., 1988; Foa, 1988; van den Hout, 1988). In addition, panickers exhibit greater amounts of fear of bodily sensations (Chambless et al., 1984; McNally & Lorenz, 1987). Overall, their ideational constitution and perception of bodily events is quite different from others (Hibbert, 1984).

Preliminary investigation has shown that cognitive events, specifically selective attention toward physical threat, differentiate panickers from controls (Ehlers, Margraf, Davies, & Roth, 1988). However, the nature of the paradigm used in the study allows for interpretations

of the results that are not related to attentional biases.

The current study clearly demonstrates the existence of an attentional bias toward physical threat in panic patients. The visual attention paradigm adopted from MacLeod et al., (1986) has a distinct advantage over the paradigm used by Ehlers, Margraf, Davies, and Roth (1988) in that it eliminates the possibility of response bias explanations of the effect by requiring a neutral response (bar press) to a neutral stimulus (dot probe). For the same reason, it eliminates explanations based on impaired performance efficiency in panickers resulting from increased arousal associated with responding to threatening material, as in a Stroop color-naming task. Specifically, the results of the current study show that panic disorder patients, but not controls, have significantly shorter probe detection latencies for physical threat cues compared to social threat cues. These results are indicative of an attentional bias toward physical threat in panic patients.

The results of this study are consistent with psychophysiological models of panic (Clark, 1986; Ehlers, Margraf, & Roth, 1988). These models maintain that internal cues, such as bodily sensations or cognitive events that are associated with personal threat, can act as triggers of panic episodes. The current study demonstrates that panickers have an attentional bias toward physical threat cues. That is, they selectively attend to cues

of a physically threatening nature. It seems reasonable to speculate that this selective attention may trigger episodes of panic.

The results also reflect patterns observed in studies investigating panickers responses to somatic sensations. The results reveal physical cues as being the most relevant to panickers, just as it has been shown that panickers primary concerns are physical in nature (Hibbert, 1984) and that they report more fear of physical sensations than others (Chambless et al., 1984; McNally & Lorenz, 1987). In fact, panickers did report significantly greater fear of physical sensations than controls on the questionnaire given in this study.

Unlike the results of MacLeod et el. (1986) with generalized anxiety patients, the interaction of probe position with threat position and group was not significant. Thus it cannot be said, based on the current results, whether or not panickers actually shift their attention toward threat cues. The results indicate that when the patient is actively reading a physical threat word, they detect a probe quicker, regardless of its position, than if attending to a social threat word. Similar results were not obtained when the threat term appeared in the lower area of the monitor, which was not attended to by subjects. It is possible that the subjects who participated in this study did not have a chance to shift attention from the

area of active reading to the lower area of the monitor. Likewise, it is possible that they never even attempted to attend to the lower area. These possibilities would explain why probe position did not interact with threat position and group. However, they do not explain the differences between the results of this study and that of MacLeod et al. (1986). To resolve this discrepency it will be necessary to conduct a study of similar nature, with both patient groups, in which eye movements are directly measured.

Many investigators have proposed theoretical mechanisms which account for the relationship between anxiety states and attentional processes (e.g., Beck & Clark, 1988; Eysenck, 1988; Sarason, 1988). The general notion espoused is that anxiety patients are characterized by an attentional set toward physical and psychological threat. Furthermore, the threat is perceived as being dangerous to ones personal domain. Similarly, it has been posited that anxiety patients are characterized by danger schemata (i.e., cognitive structures that facilitate processing of danger related information) that exist in a relatively permanent state of activation, or have a decreased threshold of activation (Foa & Kozak, 1986; Ingram & Kendal, 1987; Mathews & MacLeod, 1985). The results of the present study are consistent with these postulations: Persons with panic disorder have an attentional set toward, and an active danger schemata

for, physical threat cues. The existence of such a set and schemata provides an explanation for why panic patients, but not controls, have a marked bias for physical threat cues compared to social threat cues.

Future investigation with panic patients needs to address the question of whether these attentional biases toward physical threat occur prior to awareness. If it could be shown that these biases operate without the patients awareness, and that they are present when the patient is not currently anxious, then it would be possible to shed some light on the occurrence of "spontaneous" panic attacks. Specifically, it may be that attentional biases occur prior to awareness and act to trigger panic episodes. This would explain why many panickers cannot identify the triggers of their panic episodes.

There are other important issues that need to be addressed by future research. The psychophysiological models of panic emphasize bodily sensations as triggers of anxiety which, in turn, can lead to panic. Therefore, it would be of benefit to study attentional biases to somatic sensations directly. In addition, it may be desireable to investigate attentional biases in infrequent panickers, especially those experiencing "spontaneous" attacks. Positive results from this group may help explain one of the predisposing factors involved in the development of panic disorder. Finally, studies

of a longitudinal nature are needed in order to determine if attentional biases are responsible for the maintenance of panic disorder.

While the results of the present study are encouraging, there are several reasons for accepting them with caution. First, the results were obtained from patient and control groups that differed in age and level of depression. However, when the effects of these differences were statistically removed, the results remained unchanged. It is, therefore, unlikely that group differences in age and depression were responsible for the findings. Second, the effects observed in this study are small. Nevertheless, the results complement those of Ehlers, Margraf, Davies, & Roth (1988). Indeed, panic patients are characterized by an attentional bias toward physical threat.

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APPENDICES

APPENDIX A

Request for Participation

The Department of Psychology is currently engaged in a research program studying the role of cognitive variables in panic. We are asking patients of the anxiety clinic to contribute approximately 60 to 80 minutes of their time to participate in a simple, non-stressful research project. If you would consider taking part, we would like to contact you and tell you more about what would be involved.

If you leave your name and phone number below, we will contact you shortly. Thank you for your consideration.

Gordon J. G. Asmundson
Dept. of Psychology

Acknowledgement

I have read the Request for Participation, and I would consider taking part in the study. I can be reached at: _____ (telephone number).

At this point, I am only agreeing to be contacted. I may or may not agree to participate in the study once I have had a chance to discuss the procedures.

Signed: _____

Date: _____

APPENDIX B

Panic Attack Inventory

NAME

AGE

SEX

A panic attack is the sudden onset of intense apprehension, fear, or terror, often associated with feelings of impending doom. Some of the most common symptoms experienced during an attack are: dizziness, shortness of breath, chest pain or discomfort and trembling or shaking.

If you have never experienced a panic attack, please ignore questions 1-11 inclusive but complete the rest of the questionnaire. If you have experienced panic attacks, please answer all the questions on the following pages.

1. In the past year approximately how many panic attacks have you had?

   1-2 3-4 5-6 7-8 9-10 11 or more

2. In the past three weeks how many panic attacks have you had?

   0 1 2 3 4 5 6 7 8 9 10 (or more)

3. For how many months or years (approximately) have you been experiencing panic attacks?

_____ months _____ years

4. Have the panic attacks become more frequent recently?

Yes No

5. Do you avoid the following situations because of fear of having a panic attack? (circle one)

	NEVER				ALWAYS
large crowds	0	1	2	3	4
social functions	0	1	2	3	4
shopping	0	1	2	3	4
transportation	0	1	2	3	4
walking alone (at night)	0	1	2	3	4
other (specify)	0	1	2	3	4

6. In which of the following situations have panic attacks occurred?
(you may check more than one).

a) _____ in a life threatening situation (describe below)

_____

b) _____ when receiving injections or minor surgery

c) _____ eating or drinking with other people

d) _____ hospitals or visits to doctor

e) _____ travelling alone by bus or coach

f) _____ walking alone in busy streets

g) _____ being watched or stared at

h) _____ going into crowded shops

i) _____ talking to people in authority

j) _____ sight of blood

k) _____ being criticized

l) _____ going alone far from home

m) _____ thought of injury or illness

n) _____ speaking or acting to an audience

o) _____ large open spaces

p) _____ going to the dentist

q) _____ attacks occurred unexpectedly, "out of the blue"

r) _____ during or following relaxation

s) _____ during or following exercise

t) _____ sleeping

u) _____ while under the influence of alcohol or drugs

v) _____ prior to or during tests or exams

w) _____ while driving a car

x) _____ walking alone at night

y) _____ while experiencing high levels of stress

z) _____ during sexually intimate situations

aa) _____ during a family crisis

bb) _____ during or following interpersonal conflict with an intimate partner (e.g., spouse)

cc) _____ during or following interpersonal conflict with a non-intimate person (e.g., boss)

dd) _____ while meeting stranger(s)

ee) _____ being in an enclosed area

ff) _____ loss or separation from significant other (e.g., death of friend, leaving home).

gg) _____ other (please explain)

7. When a panic attack occurs, generally what is the time period between the onset of the attack and when the panic is most intense?

   a. almost immediate

   b. very rapid (less than 10 minutes)

   c. moderately rapid (10-30 minutes)

   d. moderately slow (30 minutes-1 hour)

   e. slowly (more than one hour)

8. Please indicate how severely you experience each of the following symptoms when you are having a panic attack.

	Does not Occur	Mild	Moderate	Severe	Very Severe
a. dyspnea (difficulty breathing)	0	1	2	3	4
b. palpitations (heart pounding)	0	1	2	3	4
c. chest pain or discomfort	0	1	2	3	4
d. choking or smothering sensations	0	1	2	3	4
e. dizziness, vertigo, or unsteady feelings	0	1	2	3	4
f. feelings of unreality	0	1	2	3	4
g. paresthesias (tingling in hands or feet)	0	1	2	3	4
h. hot and cold flashes	0	1	2	3	4

i. sweating	0	1	2	3	4
j. faintness	0	1	2	3	4
k. trembling or shaking	0	1	2	3	4
l. fear of dying (or being seriously ill)	0	1	2	3	4
m. fear of going crazy	0	1	2	3	4
n. fear of doing something uncontrolled	0	1	2	3	4

9. How long, on the average, does a panic attack last (from start to finish)?

   a. just a few minutes (0 - 10 minutes)

   b. 10 - 30 minutes

   c. 30 minutes to one hour

   d. several hours

   e. more than one day

10. (a) Have you ever been treated for panic attacks?

    YES _____ NO _____

    If yes, please explain:

    _____

(b) Have you ever used alcohol or non prescribed drugs for preventing or reducing panic attacks?

YES _____ NO _____

11. Were you experiencing any of the following stressful events at the time you had your first panic attack?

YES ___ NO ___ difficulties at work

YES ___ NO ___ loss of a loved one

YES ___ NO ___ birth of a child

YES ___ NO ___ marital/family problems

YES ___ NO ___ life-threatening situation

YES ___ NO ___ other stressful event (please specify)

In this section are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and I can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve

3. 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I can see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.

1 I feel guilty a good part of the time.

2 I feel quite guilty most of the time.

3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.

1 I feel I may be punished.

2 I expect to be punished.

3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.

1 I am disappointed in myself.

2 I am disgusted with myself.

3 I hate myself.

8. 0 I don't feel I am any worse than anybody else.

1 I am critical of myself for my weaknesses or mistakes.

2 I blame myself all the time for my faults.

3 I blame myself for everything bad that happens.

9. 0 I don't have any thoughts of killing myself.

1 I have thoughts of killing myself, but I would not carry them out.

2 I would like to kill myself.

3 I would kill myself if I had the chance.

10. 0 I don't cry anymore than usual.

    1 I cry more now than I used to.

    2 I cry all the time now.

    3 I used to be able to cry, but now I can't cry even though I want to.

11. 0 I am no more irritated now than I ever am.

    1 I get annoyed or irritated more easily than I used to.

    2 I feel irritated all the time now.

    3 I don't get irritated at all by the things that used to irritate me.

12. 0 I have not lost interest in other people.

    1 I am less interested in other people than I used to be.

    2 I have lost most of my interest in other people.

    3 I have lost all of my interest in other people.

13. 0 I make decisions about as well as I ever could.

    1 I put off making decisions more than I used to.

    2 I have greater difficulty in making decisions than before.

    3 I can't make decisions at all anymore.

14. 0 I don't feel I look any worse than I used to.

    1 I am worried that I am looking old or unattractive.

    2 I feel that there are permanent changes in my appearance that make me look unattractive.

    3 I believe that I look ugly.

15. 0 I can work about as well as before.

    1 It takes an extra effort to get started at doing something.

    2 I have to push myself very hard to do anything.

    3 I can't do any work at all.

16. 0 I can sleep as well as usual.

    1 I don't sleep as well as I used to.

    2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.

    3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. 0 I don't get more tired than usual.

    1 I get tired more easily than I used to.

    2 I get tired from doing almost anything.

    3 I am too tired to do anything.

18. 0 My appetite is no worse than usual.

    1 My appetite is not as good as it used to be.

    2 My appetite is much worse now.

    3 I have no appetite at all anymore.

19. 0 I haven't lost much weight, if any, lately.

1 I have lost more than 5 pounds.

2 I have lost more than 10 pounds.

3 I have lost more than 15 pounds.

I am purposely trying to lose weight by eating less.

Yes ___ No ___

20. 0 I am no more worried about my health than usual.

1 I am worried about physical problems such as aches and pains; or upset stomach, or constipation.

2 I am very worried about physical problems and it's hard to think of much else.

3 I am so worried about my physical problems, that I cannot think about anything else.

21. 0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

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 that indicates how you GENERALLY feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	almost never	sometimes	often	almost always
1. I feel pleasant	1	2	3	4
2. I tire quickly	1	2	3	4
3. I feel like crying	1	2	3	4
4. I wish I could be as happy as others seem to be	1	2	3	4
5. I am losing out on things because I can't make up my mind soon enough	1	2	3	4
6. I feel rested	1	2	3	4
7. I am "calm, cool, and collected"	1	2	3	4
8. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9. I worry too much over something that really doesn't matter	1	2	3	4

10. I am happy	1	2	3	4
11. I am inclined to take things hard	1	2	3	4
12. I lack self confidence	1	2	3	4
13. I feel secure	1	2	3	4
14. I try to avoid facing a crisis or difficulty	1	2	3	4
15. I feel blue	1	2	3	4
16. I am content	1	2	3	4
17. Some unimportant thoughts runs through my mind and bothers me	1	2	3	4
18. I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
19. I am a steady person	1	2	3	4
20. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

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

DIRECTIONS: In this section are some thoughts or ideas that may pass through your mind when you are nervous or frightened. Please read each statement carefully and then circle the number to the right of the statement that indicates how often the thought occurs when you are nervous.

	Thought never occurs				Thought always occurs
1. I am going to throw up	1	2	3	4	5
2. I am going to pass out	1	2	3	4	5
3. I must have a brain tumor	1	2	3	4	5
4. I will have a heart attack	1	2	3	4	5
5. I will choke to death	1	2	3	4	5
6. I am going to act foolish	1	2	3	4	5
7. I am going blind	1	2	3	4	5
8. I will not be able to control myself	1	2	3	4	5

9. I will hurt someone	1	2	3	4	5
10. I am going to have a stroke	1	2	3	4	5
11. I am going to go crazy	1	2	3	4	5
12. I am going to scream	1	2	3	4	5
13. I am going to babble or talk funny	1	2	3	4	5
14. I will be paralyzed by fear	1	2	3	4	5

DIRECTIONS: Below is a list of specific body sensations that may occur when you are nervous or in a feared situation. Please read each item carefully and circle the number to the right of the item that indicates how frightened or worried you are by the feeling.

	Not frightened or worried by this sensation				Extremely frightened by this sensation
1. Heart palpitations	1	2	3	4	5
2. Pressure in chest	1	2	3	4	5
3. Numbness in arms or legs	1	2	3	4	5
4. Tingling in fingertips	1	2	3	4	5
5. Numbness in another part of your body	1	2	3	4	5
6. Feeling short of breath	1	2	3	4	5
7. Dizziness	1	2	3	4	5
8. Blurred or distorted vision	1	2	3	4	5
9. Nausea	1	2	3	4	5

10. Butterflies in stomach	1	2	3	4	5
11. Knot in stomach	1	2	3	4	5
12. Lump in throat	1	2	3	4	5
13. Wobbly or rubber legs	1	2	3	4	5
14. Sweating	1	2	3	4	5
15. Dry throat	1	2	3	4	5
16. Feeling disoriented	1	2	3	4	5
17. Feeling disconnected from your body	1	2	3	4	5

APPENDIX C

Threat Words Presented in Experimental Task

Physically Threatening		Socially Threatening	
injury	agony	criticized	humiliated
ambulance	harm	embarrassed	opposed
emergency	violence	inadequate	incompetent
disease	collapse	failure	worthless
cancer	disabled	stupid	ridiculed
fatal	assault	pathetic	ignored
mutilated	casualty	foolish	insecure
coffin	hearse	inferior	despised
deathbed	unwell	indecisive	ashamed
paralyzed	fracture	inept	mocked
hazard	inquest	lonely	scorned
coronary	corpse	hated	disgraced

APPENDIX D

Consent Form

Information to Participants

The experiment which you are about to take part in is a research project conducted by Mr. Gordon Asmundson through the Anxiety Disorders Clinic in the Department of Psychiatry at St. Boniface General Hospital. 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 relationship between cognitive variables and panic. Initially you will be comfortably seated in front of a computer monitor where you will receive a series of simple word tests. Word pairs will appear on the screen of the computer monitor. You will read the top word of each pair aloud. Following some word pairs, a small dot will appear in the position of either the top or bottom word. When this occurs, you will press a button on a hand-held control in order to turn the dot off as quickly as possible. Following completion of the word tests you will be asked to complete a questionnaire.

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 cognitive variables involved in panic disorder as described above.

Voluntary Participation

There are no risks associated with participation in this experiment. 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. Your care from the hospital will not be affected in any way by your agreeing or declining to participate in the study.

Confidentiality

Personal information obtained on questionnaires and from word tests is confidential and will only be shared with research collaborators of Mr. Asmundson at the University of Manitoba and St. Boniface Hospital. 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 or treatment purposes.

Signature: _____

Witness: _____

Date: _____

APPENDIX E

Instructions to Subject

Part 1

Hello, my name is Gord Asmundson. I would like to thank you for agreeing to participate in this study. By doing so, you are providing an opportunity to help advance research in the area of anxiety. The first thing I am going to ask you to do is have a seat in front of the computer monitor. I am going to have you complete a series of simple word tests. During these tests, word pairs will appear on the monitor. Whenever a word pair appears, I want you to read the top word aloud. On several trials a small dot will appear in either the position of the top or the bottom word immediately after the words disappear from the screen. When this happens, I want you to press this button as quickly as possible. Now we will try a few practice trials. Do you have any questions? There will be a fairly large number of trials. However, the entire procedure should not take more than 20 minutes. I will let you know when we are finished.

Part 2

Now I am going to ask you to complete this questionnaire. Please read the appended instructions

carefully before you begin. It should take you approximately 30 minutes to finish the entire questionnaire; however, do not feel pressured if you need more time.

APPENDIX F

Summary Repeated Measures ANOVA Table for Probe Detection Latencies

Source	SS	df	MS	F	Tail Prob.
Mean	58799379.37	1	58799379.37	585.94	0.0000
G	3844.87	1	3844.87	0.04	0.8462
error	2809814.25	28	100350.51		
R	73230.81	1	73230.81	15.75	0.0005
RG	668.31	1	668.31	0.14	0.7075
error	130206.28	28	4650.22		
S	1718.28	1	1718.28	0.76	0.3914
SG	3936.92	1	3936.92	1.74	0.1982
error	63475.50	28	2266.98		
RS	2616.31	1	2616.31	0.55	0.4635
RSG	23449.81	1	23449.81	4.95	0.0343
error	132605.28	28	4735.90		
T	60152.95	1	60152.95	9.81	0.0040
TG	1564.58	1	1564.58	0.26	0.6174
error	171665.34	28	6130.90		
RT	53.28	1	53.28	0.03	0.8596
RTG	2143.78	1	2143.78	1.28	0.2671
error	46814.34	28	1671.94		
ST	546.37	1	546.37	0.24	0.6276
STG	1502.67	1	1502.67	0.66	0.4228
error	63569.25	28	2270.33		
RST	1842.81	1	1842.81	1.03	0.3181
RSTG	174.31	1	174.31	0.10	0.7569
error	49940.61	28	1783.59		

Note: G = group; R = threat type; S = threat position; T = probe position.