UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
4301 JONES BRIDGE ROAD
BETHESDA, MARYLAND 20814-4799

APPROVAL SHEET

Title of Dissertation: "Effects of Attentional Focus on Emotional Responding to a Biological Challenge in Panic Disorder"

Name of Candidate: MAJ John Trakowski
Doctor of Philosophy Degree
26 August 1996

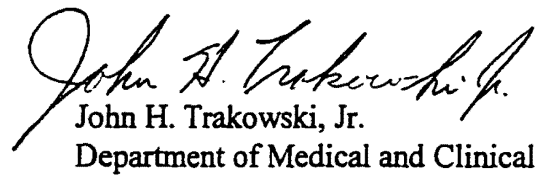
Dissertation and Abstract Approved:

Committee Chairperson	Date: 26 Aug. 1996
Committee Member	Date: 26 Aug 1996
Committee Member	Date: 26 Aug 1996
Committee Member	Date: 26 Aug 1996

The author hereby certifies that the use of any copyrighted material in the thesis manuscript entitled:

"Effects of Attentional Focus on Emotional Responding
to a Biological Challenge in Panic Disorder"

beyond brief excerpts is with the permission of the copyright owner, and will save and hold harmless the Uniformed Services University of the Health Sciences from any damage which may arise from such copyright violations.



John H. Trakowski, Jr.
Department of Medical and Clinical Psychology
Uniformed Services University of the Health Sciences

Abstract

Title of Dissertation: Effects of Attentional Focus on Emotional Responding to a Biological Challenge in Panic Disorder

MAJ John H. Trakowski, Jr., Ph.D., 1996

Thesis directed by: Norman B. Schmidt, Ph.D., Assistant Professor, Department of Medical and Clinical Psychology

The principal aim of this study was to assess whether attentional focus affects fearful responding. Specifically, the present study tested a cognitive model of panic (Clark, 1986) to determine whether attentional focus affects fearful responding to heightened somatic cues. The study compared panic disorder subjects and nonclinical controls at baseline during inhalation of 35% CO_2 gas on measures of subjective and psychophysiological responding. A 2 (Panic Disorder versus Normal Controls) X 3 (Internal Focus versus External Focus versus No Focus) factorial design was employed to test for main effects of group status, instructional set, and their interaction. All subjects were assessed before and after the experimental manipulation on measures of anxiety, panic, cognition, attentional focus, physical symptoms and psychophysiological measures. Subjects were diagnosed for current and past DSM-IV diagnoses for Axis I disorders through a structured diagnostic interview. Subjects meeting DSM-IV criteria for panic disorder with or without agoraphobia (n=45) were matched to nonclinical controls (n=45) in terms of age and gender. Consistent with prediction, PD subjects were more internally focused before the experimental manipulation and were more internally focused during the biological challenge compared to nonclinical controls. PD subjects

also showed more subjective but not physiological distress during the biological challenge. In addition, reliance on emotion focused coping skills increased fearful responding to the biological challenge.

Several hypotheses were not supported. Contrary to prediction, the focus manipulation did not have any substantial impact on fearful responding. There was also no group by condition interactions indicating greater fearful responding for PD subjects in the internal focus condition relative to PD subjects in the external focus condition. Data were reanalyzed according to anxiety sensitivity and level of internal focus. Findings were consistent with the original hypotheses indicating that internal focus predicts fearful responding for individuals with heightened fear of autonomic arousal.

EFFECTS OF ATTENTIONAL FOCUS ON EMOTIONAL RESPONDING TO A BIOLOGICAL CHALLENGE IN PANIC DISORDER

by

MAJ JOHN H. TRAKOWSKI, JR., M.S.

Dissertation submitted to the faculty of the Department of Medical and Clinical Psychology of the Uniformed Services University of the Health Sciences in partial fulfillment of the requirements for the degree of Doctor of Philosophy 1996

Dedication

I dedicate my years at USUHS and my dissertation to my best friend and wife Giannina and my sons Alex and Tony.

Acknowledgment

I want to first acknowledge that without my faith in the Lord these past four years at USUHS would have never been possible. Next, I would like to thank the old and new faculty of the Department of Medical and Clinical Psychology (Dr. Feuerstein, Dr. Krantz, Dr. Baum, Dr. Grunberg, Dr. Law, Dr. Sbrocco, and Dr. Schmidt) for their support and encouragement. Especially, I would like to thank Dr. Singer for his steadfast and unwavering support. I would also like to thank the military faculty from Walter Reed and Malcom Grow for providing me my practicum experiences.

Next, I would like to thank my dissertation committee (Dr. Singer, Dr. Schmidt, Dr. Sbrocco and Dr. Stabb) for their support and guidance throughout the dissertation process. To the Schmidt group (Darin, Margy, Lavent, Loretta, and Helen) thank you for reviewing versions of my manuscript and helping me run my protocol. To Helen, Matt, Pete, and Martha thank you for your support and camaraderie in helping me to prepare for my dissertation defense. To Brad Schmidt, my primary advisor thank you for your guidance and encouragement through the process. It was a growing experience that I will never forget. We made it Brad! I would also like to thank all the students and staff, especially Lorelei, Randy and Julie for their support and encouragement. To "the group" thanks for the special experience. To my father-in-law "Papi", thank you for being a second father to my sons when I was unable to be with them. To my sons Alex and Tony, I thank you for reminding me that there is always time to play. Finally, I want to thank my best friend and wife for all her love, support and encouragement through the years.

Table of Contents

List of Tables

List of Figures

CHAPTER 1

INTRODUCTION

1.1. Diagnostic Criteria for Panic Disorder With and Without Agoraphobia

A panic attack is a period of intense fear and discomfort accompanied by somatic and/or cognitive symptoms. The sudden onset of symptoms builds to a peak coinciding with a sense of imminent danger and an urge to escape. Some examples of the somatic and cognitive symptoms include palpitations, shortness of breath, fear of dying and a fear of losing control (American Psychiatric Association, 1994).

Panic disorder is the presence of recurrent, unexpected panic attacks followed by at least 1 month of worry about having additional attacks, the possible consequences of additional panic attacks, or having a significant behavioral change related to the attacks. Panic disorder with agoraphobia includes significant avoidance of or distress in places or situations from which escape may be difficult or embarrassing (APA, 1994).

1.1.1. Epidemiological Aspects of Panic

The Epidemiologic Catchment Area (ECA) survey of 1980 reported that the prevalence for panic disorder under the DSM-III criteria was 1.4% of the general population (Robins & Regier, 1991). Women were diagnosed with panic disorder approximately twice the rate of men. The 1-month, 1-year, and lifetime prevalence rates for women were 0.7%, 1.2%, and 2.1%, respectively, whereas the corresponding rates for men were 0.4%, 0.6% and 1.0%. The diagnosis of panic disorder was most common among subjects aged 30 to 44 years, and least common among subjects who were 65 years or older.

Katerndahl and Realini (1993) disputed the ECA survey findings reporting a lifetime prevalence of DSM-III-R panic disorder nearly twice that of the ECA. They evaluated a community sample of 1,306 residents of San Antonio, Texas using the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1992) and found a lifetime rate of panic disorder was 4.1% for women, 1.5% for men with a combined rate of 3.8%. The authors conclude that the elevated rates of panic in their study were due to the fact that the SCID is likely to detect more true cases of panic disorder than the methods used in the ECA survey.

Agoraphobia was diagnosed in the ECA survey if the subject had a fear of at least one of the following: being alone; going out of the house alone; being in a crowd; tunnels, or bridges; or being on any kind of public transportation like airplanes, buses or elevators (Bourdon, Boyd, Rae, Burns, Thompson, & Locke, 1988). The 1-month, 1-year, and lifetime prevalence rates for agoraphobia among women were 4.4%, 5.9%, and 7.7% (Bourdon et al., 1988). The corresponding rates among men were 1.6%, 2.1%, and 2.9% (Bourdon et al., 1988). One year after the original ECA interviews, investigators reinterviewed 80% of the subjects to determine the number of new cases of each disorder. The study yielded an incidence of panic disorder at 2.4 new cases per 1,000 of the population per year (Keyl & Eaton, 1990). The estimated annual incidence of severe, spontaneous panic was nine per 1,000 of the population per year. Risk factors for panic attack included female sex, history of depression or grief, substance abuse or dependence, and seizures.

1.2. Biological Models of Panic

Biological challenge research paradigms have been used to examine the biological models of panic disorder. Challenge-induced panic has been proposed as a biological marker for panic disorder (Dager, Cowley, & Dunner,1987). Accordingly, biological challenges provoke panic attacks by directly activating a biochemical abnormality. These challenges are designed to test certain neurobiological systems, and if panic results, dysfunction in the system is then believed to be a vulnerability to naturally occurring panic attacks. Studies have investigated respiratory function (e.g., hyperventilation), cardiovascular difficulties (e.g., mitral valve prolapse), and neuroendocrine aspects of the disorder but no single biological dysfunction appears to underlie all panic attacks.

Researchers has also examined whether there is a connection between the noradrenergic system (i.e., the locus ceruleus) and panic. Infusion of yohimbine is a biological test of the noradreneric system. Yohimbine is a specific alpha-2-adrenergic receptor agonist that increases locus ceruleus activity (Charney, Heniger, & Brier, 1984; Uhde, Boulenger, Post, Siever, Vittone, Jimerson, & Post, 1984; Uhde, Roy-Byrne, Vittone, Boulenger, & Post, 1985). This biological challenge reliably produces panic but there are many ways of provoking panic that do not involve the noradrenergic system. Other biological challenges (e.g., carbon dioxide, caffeine, lactate infusion) have been used to provoke somatic symptoms that are similar to those reported during panic attacks. However, all of the studies using biological challenges lead to different explanations for the cause of panic.

Klein (1993) has suggested that spontaneous panics occur when the brain's suffocation monitor signals a lack of useful air and triggers a suffocation alarm. This alarm is activated not only by rising levels of brain carbon dioxide. As the suffocation monitor misfires, it produces sudden respiratory distress followed swiftly by a brief hyperventilation, panic, and the urge to flee.

Klein cites a wide range of evidence to support his theory. For example, he reports that the fear of suffocation is common in the general population (Rachman, 1990) and suggesting a common adaptive mechanism. Dyspnea is a characteristic of spontaneous panic, but not of normal fear. In addition, infants with congenital central hypoventilation syndrome (also known as Ondine's curse) breathe normally while awake, but cease breathing once asleep, and may die in the absence of ventilatory support procedures. These children show no signs of respiratory that suggests that they lack a suffocation detection monitor (Mc Nally, 1994). Klein also states that PD is the most common anxiety disorder among individuals with pulmonary disease (Karajgi, Rifkin, Doddi, & Kolli, 1990).

1.3. Psychological Models of Panic

1.3.1. Cognitive Models of Panic

Cognitive models of panic (Beck & Emery, 1985, Clark, 1986) propose that panic attacks can result from a wide range of stimuli. These stimuli can be external (such as a mall for an agoraphobic who has previously had an attack in the mall) but more often are internal (body sensation, thought or image). A state of apprehension occurs when either the external or internal stimuli are perceived as a threat. Catastrophic misinterpretation of

bodily cues necessarily lead to an increase in anxiety. Catastrophic misinterpretation typically involves the perception that the sensations are a threat to the individual's physical and/or mental well being. The interaction between the catastrophic interpretation of sensations and the intensification of anxiety-related symptoms experienced creates a vicious cycle that may culminate in a panic attack.

1.3.2. Barlow's Biopsychosocial Model of Panic

Barlow (1988) expands upon earlier cognitive models by positing a biological disposition to panic disorder in addition to emphasizing the interoceptive conditioned linkage between bodily sensations and the panic response. In Barlow's model, individuals with panic disorder are believed to possess a biological predisposition to overreact to stress. These individuals react to stress with heightened physiological arousal leading to an "alarm reaction." The alarm reaction is described as a false alarm when there is no true danger. In the absence of true danger, individuals frequently attend to internal bodily sensations as the locus of the threat. Similar to earlier cognitive models, the misinterpretation of bodily sensations will create fear.

In addition, a conditioned linkage often occurs between interoceptive cues and the "false alarm". Particular sensations become conditioned cues for reactivation of the alarm response. In each additional attack the alarm response becomes more firmly linked with these sensations. Learning that interoceptive cues are threatening, coupled with a lack of predictability and controllability of future attacks, leads to the development of anxious apprehension and phobic avoidance.

The similarity in these psychological models is that panic disorder is conceptualized as a disorder of thinking in which the patient catastrophically misinterprets benign cues. The "alarm reaction" or panic episode itself is a normal emergency response to perceived danger. Barlow's model (1988) also emphasizes the conditioned link that occurs between interoceptive cues and the alarm response. In sum, the principal mechanisms involved in the generation and maintenance of pathological fear are: (a) the perception and catastrophic misinterpretation of bodily sensations, and (b) the interoceptive conditioning of bodily sensations to the fear response.

1.4. Biological Challenges

There have been a variety of biological challenges utilized in the study of panic disorder. Examples of biological challenge agents include carbon dioxide, sodium lactate, yohimbine, caffeine, hyperventilation, cholecystokinin, and isopreterenol. Researchers have used these biological challenges (i.e., the introduction of biological substances) to provoke somatic symptoms that are similar to those reported during panic attacks (Sanderson, Rapee, & Barlow, 1989; Clark, Salkovskis, & Anastiasiades, 1990). Challenge studies have demonstrated that patients with panic disorder show higher levels of anxiety and panic compared to both psychiatric and normal controls (Rapee, 1986; Rapee, Mattick, & Murrell, 1986).

1.4.1. Carbon Dioxide

Inhalation of a single vital capacity breath of a 35% CO_2/65% O_2 mixture immediately reproduces the physical symptoms of panic in both patients and healthy control subjects (van den Hout, 1988), but triggers fear in panic disorder patients (Griez,

Lousberg, van den Hout, & van den Molen, 1987). A single vital capacity inhalation of 35% CO_2/65% O_2 produces immediate alveolar hypercapnia that stimulates the carbon dioxide receptors. Their stimulation triggers an intense ventilatory response that results in a hypocapnia overshoot (van den Hout, & Griez 1985). The response to 35% CO_2 is an immediate hypercapnic acidosis that is later followed by a hypocapnic alkalosis specific to hyperventilation. Papp, Klein, Martinez, Schneier, Cole, Liebowitz, Hollander, Fyer, Jordan, and Gorman (1993b) reported that 35% CO_2 produced panic in 72% of panic patients, 30% of social phobics, and 4% of healthy control subjects. Carbon dioxide challenges are a potent means of provoking intense cardiorespiratory sensations that produce fear in anxiety-sensitive individuals.

1.4.2. Sodium Lactate

Pitts and McClure (1967) were the first to demonstrate the panicogenic effects of sodium lactate infusions. They found that anxiety symptoms were evoked by exercise and appeared to be concomitant with an extreme rapid rise of blood lactic acid. The mean rates of lactate-induced attacks in panic patients and in nonpsychiatric control subjects are approximately 67% and 13%, respectively (Cowley & Arana, 1990) indicating that, lactate challenges successfully discriminate between panic patients and normal controls.

1.4.3. Yohimbine

Yohimbine is an alpha-adrenergic antagonist that has been used to provoke panic. This biochemical agent is one of the few of its type that can cross the blood-brain barrier and act centrally. Yohimbine produces high rates of panic in panic disorder patients. For example, Uhde et al. (1985) reported that five out of seven patients reported panic attacks

after oral ingestion of a low dosage of yohimbine. Charney, Heninger, and Breier (1984) found that panic disorder patients became significantly more fearful and indicated their experience was highly similar to naturally occurring panic attacks.

1.4.4. Caffeine

Caffeine ingestion produces dose-dependent increases in anxiety for healthy as well as for anxious subjects (Uhde, 1990), through the antagonism of adenosine, a neuromodulator that inhibits release of norepinephrine (Charney, Heninger, and Jatlow, 1985). Boulenger, Uhde, Wolff, & Post (1984) found that caffeine consumption was highly correlated with self reported anxiety in patients with panic disorder. Challenge studies with caffeine have found higher rates of panic in patients than in healthy controls. For example, Charney et al. (1985) found that 71% of panic disorder patients reported significant increase in anxiety and fear following the oral administration of caffeine.

1.4.5. Hyperventilation

Hyperventilation occurs any time an individual overbreathes or blows off an excess of CO_2 from the lungs faster than it can be manufactured by the body. This action decreases the pCO_2 in the lungs and blood that raises the blood pH (respiratory alkalosis) giving rise to a variety of physiological symptoms including chronic sighing, dizziness, paresthesias, palpitations, and dyspnea. Garssen, Van Veenendaal, and Bloemink (1983) found that hyperventilation in agoraphobic patients with panic produced somatic symptoms that were highly similar to the sensations they felt during a panic attack. Rapee (1986) compared panic disorder patients subjective responses to generalized anxiety disorder patients after 90 seconds of voluntary hyperventilation. Panic disorder

patients reported markedly greater distress and a greater symptoms in response to the voluntary hyperventilation.

1.4.6. Cholecystokinin

Cholecystokinin (CCK) is a neuropeptide present in high concentrations in the cerebral cortex, the amygdala, and the hippocampus that functions as a neurotransmitter (Bradwejn, Koszycki, Couetoux du Tertre, Bourin, Palmour & Ervin, 1992). Recent human research indicates that injected CCK is anxiogenic (Bradwejn et al., 1992). Preclinical studies suggest that benzodiazepine receptor agonists antagonize the peripheral and central effects of CCK, and lorazepam prevents CCK-induced fear (de Montigny, 1989). All of these findings led Bradwejn et al. (1992) to suggest that panic disorder may be characterized by dysregulation in the CCK system.

1.4.7. Isoproterenol

Isoproterenol is a biological agent that stimulates only beta-adrenergic receptor sites. Rainey, Pohl, Williams, Knitter, Freedman, & Ettedgui (1984) compared the effects of isoproterenol with those of sodium lactate in panic disorder patients. The authors found that 10 out of 11 panic disorder patients and 3 out of 10 control subjects experienced a panic attack during the lactate infusion. During the isoproterenol infusion, 8 out of 11 panic disorder patients and 2 of the 10 control subjects experienced panic. Similarly, Pohl, Yeragani, Balon, Ortiz, & Aleem (1990) found that isoproterenol produced higher rates of panic in panic disordered patients than in normal controls.

1.4.8. Relaxation

Another "challenge" that produces panic is relaxation. Adler, Craske, and Barlow (1987a, 1987b) examined the panicogenic properties of relaxation with a group of 15 panic disorder patients with and without agoraphobia. In this study the patients were instructed to listen to one of three tapes: a relaxation tape, a tape with instructions on muscle tension, and a neutral tape containing a passage from a popular novel. The authors found that the patient's response to the relaxation tape was associated with a significantly greater similarity to natural panic and with less self-control than the responses to the other tapes.

1.5. Psychological Factors in Provocation Studies

Biological challenges represent the most widely employed experimental paradigm for investigating the neurobiological basis of panic disorder. It is hypothesized that, specific agents (e.g., sodium lactate, CO_2, caffeine) precipitate panic by triggering some neurobiochemical dysregulation mechanism. The previous review of biological challenges indicates that panic disorder subjects show significant increases in anxiety following relaxation, infusions of sodium lactate, inhalation of carbon dioxide, infusions of isopreternol, infusions of yohimbine, voluntary hyperventilation , and administrations of caffeine. However, all of the studies using biological challenges lead to different explanations for the cause of panic. Recent psychological models of panic (i.e., Beck, 1988; Clark, 1986) offer an alternative explanation to account for challenged induced panic: namely, that panic results from the catastrophic misinterpretation of challenge-induced bodily cues.

1.5.1. Background

Early research has shown that the interpretation of emotions can be explained by cognitive factors. Schachter and Singer (1962) found that an individual's emotional and physiological state can be affected by the cognitions that are attached to them. As individuals become aware of arousal, they may appraise the context to determine an appropriate label for the arousal. For example, if an individual was aroused while jumping out of a plane without a parachute, the arousal would be described as fear, while the same level of arousal during sexual relations would be described as sexual excitation.

Given that cognitions can influence and determine an individual's emotional and physiological state, panic researchers have manipulated various psychological factors in the context of biological challenges. Perceptions of safety and perceived control are some of the cognitive factors that have been shown to influence panic patients' and nonclinical subjects' emotional responding to biological challenge. One property that these factors (e.g., perceived control, perceived safety) share is alteration of the subjects' threat appraisal. The influence of cognitive factors runs contrary to the view that panic disorder subjects' heightened emotional responding is due solely to a biochemical defect. An explanation that accounts for these cognitive factors is the fear-of-fear hypothesis. This hypothesis predicts that subjects who are fearful of physical sensations should show heightened emotional responding when exposed to the somatic perturbations created by a challenge task.

1.5.2. Psychological Parameters that have been Manipulated in Biological Challenges.

1.5.2.1. Perceived Control

Barlow (1988) posits that perceived control is likely to influence anxiety. He suggests that at the core of the affective component of the complex cognitive-affective structure of anxiety is a sense of uncontrollability and unpredictability. Accordingly, patients undergoing biological challenge procedures who experience greater perceived control should experience less distress and be less likely to panic than patients who have less control.

Sanderson, Rapee, and Barlow (1989) manipulated perceived control during a 15-minute inhalation of 5.5% carbon dioxide with panic disordered patients. The authors gave the patients identical instructions as to the expected effects of CO_2. A dial was placed in front of the patients and they were told that they could control the amount of CO_2 they received if, and only if, a light on top of the dial illuminated. The light was illuminated (giving perceived control) for half of the subjects. Findings indicate that patients who believed they could not control the CO_2 administration: (1) reported a greater number of panic symptoms, (2) rated the symptoms as more intense, (3) reported greater subjective anxiety, and (4) reported a greater number of catastrophic cognitions.

1.5.2.2. Perceived Safety

The effect of safety cues is another factor that has been examined affecting the psychological mediation of response to a biological challenge. Safety cues are thought to

reduce anxiety to a stimulus by providing information that the unconditioned stimulus (UCS) or anticipated threat will not occur.

Rapee, Telfer, and Barlow (1991) manipulated safety by placing subjects in either a safe (doctor present) or unsafe (no doctor present) group before a 15-minute inhalation of 5.5% CO_2. In the safe group, the subject had contact with a laboratory-coated "doctor" and the presence of a professionally dressed graduate student assistant. In the unsafe group, subjects were informed that the "doctor" was called away on an emergency and had the CO_2 delivered by a poorly dressed, "student" assistant. Compared to subjects in the high safety group, subjects in the low safety groups reported higher levels of fear and greater likelihood of panic.

In a similar study, Carter, Hollon, Carson, and Shelton (1995) examined the effect of having a safe person present during a 5.5% CO_2 inhalation procedure. Panic patients exposed to the CO_2 gas without a safe person reported greater distress, a greater number of catastrophic cognitions, and a greater level of physiological arousal compared to those patients with a safe person.

Schmidt and Telch (1994) investigated the singular and joint effects of fear of somatic sensations and perceived safety of hypocapnia-induced bodily cues on nonclinical subjects' subjective and psychophysiological response to a hyperventilation challenge. Fear of fear was assessed with the Body Sensations Questionnaire (BSQ; High versus Low). The authors found that when anticipating hyperventilation, High BSQ-Safety Information subjects reported higher subjective anxiety compared to Low BSQ-

Safety Information subjects. However, subjects with pre-challenge information showed less subjective fear during the challenge itself.

Rapee, Mattick and Murell (1986) manipulated perceived safety with panic disordered patients in a 50% carbon dioxide challenge. Subjects were randomly assigned to receive either: (1) a highly detailed and objective description of CO_2, or (2) minimal information about CO_2 and its effects. By giving a minimal explanation about the effects of CO_2, the subjects could engage in their usual associations when experiencing unexplained somatic sensations. Panic disordered patients who were given no explanation reported a greater proportion of catastrophic cognitions, and a greater frequency of panic attacks compared to those who received a full explanation.

Clark, Salkovskis, and Anastiasiades (1990) replicated the previous study with panic disorder patients using a sodium lactate infusion. Again, one group was given a complete explanation of the effects of sodium lactate whereas the other group was given a minimal explanation of the effects of sodium lactate infusion. Consistent with Rapee et al. (1986) panic disorder patients in the high information group reported less anxiety, and were less likely to panic, and had less heart rate activity.

1.5.3. Coping Strategies Related to Panic

One aspect of panic disorder that has been given little attention is the use of coping strategies. No work has examined the role of coping strategies in the context of a biological challenge.

Coping behavior is considered an important mediator between life stress and mental health (Folkman, Lazarus, Gruen, DeLongis, 1986). Maladaptive coping has been

shown to be associated with both mental disorders (Woodruff, Goodwin & Guze, 1974) and adaptation to stressful life events (Cohen & Lazurus, 1979; Moos, 1982).

Folkman and Lazarus (1980) define coping as the cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person. The authors hypothesize that coping has two major and widely recognized functions: the regulation of distressing emotions (emotion-focused coping) and generating alternative solutions to change the problem causing the distress (problem-focused coping). Folkman and Lazarus's (1980) theory of coping has direct relevance for the study of panic disorder. Panic disordered patients are necessarily extremely focused on regulating their emotions, primarily their emotion of fear.

Some research has in fact shown this to be the case. Vitaliano, Katon, Russo, Maiuro, Anderson and Jones (1987) investigated coping strategies in patients with panic disorder and normal controls. Findings indicated that panic disordered patients used proportionately more emotion focused coping than normal controls. Similarly, Vollrath and Angst (1993) investigated coping strategies between patients with panic, with other anxiety disorders versus normal controls. Patients with panic used more emotion focused coping strategies than patients with other anxiety disorders or normal controls.

1.6. Conditioning of Somatic Sensations

1.6.1. Pavlovian Interoceptive Conditioning

Razran (1961) defines interoceptive conditioning as: "classical conditioning in which either the conditioned stimulus (CS) or the unconditioned stimulus (US) or both are delivered to the mucosa of some specific viscus"(pp. 81). In the case of interoceptive

conditioning in panic disorder, an internal cue (e.g., heartbeat) may become a CS by becoming paired with a panic attack (US).

Razran's (1961) research showed that the conditioned stimuli in interoceptive conditioning are internal bodily sensations. Using Razran's (1961) work Goldstein and Chambless (1978) proposed that the fear of fear evidenced in agoraphobia may result from Pavlovian interoceptive conditioning. The authors note that individuals become hyperalert to internal bodily sensations following a panic episode. Goldstein and Chambless (1978) further state that physiological cues can become the conditioned stimuli for the conditioned response of a panic attack. However, Goldstein and Chambless's (1978) reanalysis of agoraphobia becomes problematic when strictly adhering to learning principles. In their analysis the US is considered the person's first panic attack as well as the CR. In addition, the bodily sensations that make up panic have already been assigned the functional role of both CS and CR. Using learning principles to explain panic in this manner becomes confusing as to what is the US and what is the UR (McNally, 1994).

Although researchers have tried to explain panic attacks through Pavlovian interoceptive conditioning, there are conceptual difficulties when interpreting panic in learning terms. Given this, it is more accurate to describe a panic attack as a feedback loop where there is a conditioned linkage between internal bodily sensations and resulting catastrophic ideation.

1.6.2. Somatic Awareness

Individuals with greater levels of anxiety report more somatic symptoms than do nonanxious individuals (Pennebaker, 1982). Biological challenge procedures produce somatic symptoms which, in turn, are associated with impending threat by individuals with panic disorder.

Psychological models of panic disorder propose that panic attacks result from the patient's catastrophic misinterpretation of these benign body sensations (Beck & Emery, 1985; Clark, 1986; Barlow, 1988). Interoception, the perception of bodily cues, is a necessary element, and can be, a primary trigger in psychological models of panic. Previous research has found that accuracy in visceral perception is greater under conditions of heightened physiological arousal (Katkin 1985; Montogomery, Jones & Hollandsworth 1984; Schandry & Specht 1981). Pennebaker (1982) explains the discrepancy between these studies as resulting from a "competition of cues" where awareness of internal stimuli is a function of the ratio of internal to external stimuli. The "competition of cues" hypothesis implies that when internal information is constant, the amount of potential external information will be inversely related to symptom reporting. Conversely, when external information is invariant, the degree of potential internal information will be positively correlated with symptom reporting (Pennebaker, 1982).

Because of the continuous bombardment of internal and external information, incoming data must be organized and reduced. Synthesizing and organizing incoming data occurs through schemas that guide the search for information. Depending on the

schema that individuals adopt, they will attend to schema-consistent information and tend to ignore schema-inconsistent information. The active processing of perceptions leads individuals to look for relevant information. The perception and interpretation of internal sensations that are vague, diffuse, and ambiguous will be greatly influenced by the schema adopted. Thus, the perception of any given body state can substantially vary in quality (e.g., pleasant versus unpleasant) as a function of the schema.

Pennebaker's research (Pennebaker, 1982; Pennebaker et al., 1985) indicates four aspects of interoception that may explain why panic patients are more likely than other people to experience somatic symptoms, that can lead to anxiety: (1) Panic patients may be physiologically more reactive such that they experience more fluctuations in their physiological functions, (2) Panic patients may have an increased ability to perceive their physiological state, (3) Panic patients may focus their attention on their body and will, therefore, be more likely to detect physiological changes, (4) Panic patients may attach more significance to benign physiological changes (Ehlers,1993).

1.6.3. Cardiac Awareness in Panic Disordered Patients

Palpitations are among the most common symptoms of panic attacks (Barlow, 1988, Taylor, Ehlers, Roth & Argras, 1987). Ehlers, and Maddock (1986) found that panic disorder patients scored higher on self-reported cardiac awareness than control subjects without panic attacks. Previously reviewed psychological models of panic posit that the catastrophic misinterpretation of bodily cues can lead to a panic attack. Ehlers, Margraf and Roth (1988a) suggested that increased cardiac awareness coupled with misinterpreting the cardiac sensations as threatening could be involved in the

development and maintenance of panic disorder. Ehlers & Breuer (1992) posited that the degree to which panic disorder patients can perceive their heartbeat may be related to the frequency of panic attacks and may motivate patients to avoid situations in which these sensations occur. To date, studies investigating heartbeat perception in panic disorder have found inconsistent findings. Ehlers, Breuer, Dohn, & Fiegenbaum (1995) findings indicated that panic disorder patients with agoraphobia were better able to perceive how fast their heart was beating compared to normal controls. Similarly, Ehlers & Breuer (1992) found that panic disorder patients reported greater cardiac awareness and were better able to perceive their heartbeats than normal controls. However, Ehlers, Magraf, and Roth (1988a) and Ehlers, Margraf, Roth, Taylor & Birbaumer (1988b) found that panic disordered patients were not better able to perceive their heartbeats than normal controls. These findings are inconsistent because the latter studies required subjects to compare an external signal (series of tones) with the rhythm of their heartbeats. By comparing the external signal to their heartbeat subjects may have been distracted from their internal cues (Pennebaker, 1982).

1.7. The Present Study

1.7.1. Gaps in the Literature that the Present Study Addresses

The preceding literature review illustrates the various biological challenge agents that researchers have used to investigate the etiology of panic disorder. The diversity of challenge agents that provoke panic has led to different explanations for the cause of panic. The biological challenge paradigm has also been used to investigate the contribution of various psychological factors in the generation of fear. Tests of the

cognitive model of panic have largely focused on the appraisal of threat and perceived safety cues. Other work has focused on the role of attention to internal bodily sensations in panic disorder. This line of work has suggested that patients with panic disorder show greater interoceptive acuity compared to normal controls.

Examination of the role of attentional focus in the context of a biological challenge is an extension of previous work. Evaluation of attentional focus to bodily sensations, in the context of heightened arousal, has obvious relevance to cognitive models of panic in which the perception of internal bodily sensations is necessary for their catastrophic misappraisal.

1.7.2. Study Overview

The present study adds to our knowledge in several ways. First, the study will assess differences in attentional focus to bodily cues under conditions of normal arousal as well as heightened arousal. Second, the study offers a test of cognitive models of panic in terms of whether attentional focus affects fearful responding to the heightened somatic cues.

1.7.3. Study Hypotheses

1. Consistent with the work of Ehlers (1992,1995) and Pennebaker (1982,1985), it is expected that patients with panic disorder will be naturally more internally focused compared to normal controls. During conditions of heightened arousal, it is also expected that panic disorder patients would show increased internal focus to the somatic cues they perceive as threatening.

Hypothesis 1a: It is hypothesized that panic disorder patients will show greater internal focus compared to normal controls.

Hypothesis 1b: In addition, it is also hypothesized that panic disorder patients will show greater internal focus during the biological challenge compared to normal controls regardless of experimental condition.

2. Consistent with cognitive models of panic, it is expected that subjects in the internal focus condition will show greater fearful responding compared to those in the external focus and no focus conditions.

Hypothesis 2: It is hypothesized that there will be a main effect for experimental condition such that subjects in the internal focus condition will show more fearful responding compared to subjects in the other conditions.

3. Consistent with the biological challenge literature, it is expected that patients with panic disorder will show heightened fearful responding to the CO_2 challenge compared to normal controls.

Hypothesis 3: A main effect for group status is hypothesized such that panic disorder patients will show greater subjective and physiological distress during the CO_2 challenge compared to normal controls.

4. Consistent with the literature indicating that patients with panic disorder individuals are more fearful of somatic perturbations, it is expected that panic disorder patients in the internal focus condition will show heightened fearful responding relative to panic disorder patients in the external focus condition. In addition, the difference in

fearful responding between normals in the external versus internal focus conditions are expected to be minimal.

Hypothesis 4a.: It is hypothesized that panic disorder patients in the internal focus condition will show heightened fearful responding relative to panic disorder patients in the external focus condition.

Hypothesis 4b.: It is hypothesized that the difference in fearful responding between normals in the external versus internal focus conditions are expected to be minimal.

Hypothesis 4c.: It is hypothesized that panickers in the internal focus condition will not differ from panickers in the no focus condition because patients in the no focus condition will normally turn to their focus toward internal sensations.

5. Consistent with the literature, it is expected that panic disorder patients will report greater use of emotion focused coping compared to normal controls. In addition, it is expected that level of emotion focused coping will predict fearful responding to the biological challenge.

Hypothesis 5. It is hypothesized that level of emotion focused coping will predict fearful responding to the biological challenge.

CHAPTER 2

METHOD

2.1. Overview

Forty-five subjects with a principal DSM-IV diagnosis of panic disorder and forty-five subjects with no history of any Axis I disorder were randomly assigned to one of three experimental conditions (internally focused, externally focused, and no focus). The internal focus condition was trained to attend to their heart beat. The external focus condition attended to an audio tape and was asked to count the number of times the letter G follows the letter Z in a string of letters. The no focus condition was given no instructions regarding focal attention. After completing several training trials within each of the conditions, subjects completed a carbon dioxide (CO_2) challenge. Subjects were assessed before, during and after the experimental manipulation using physiological and self-report measures of emotional responding.

2.2. Subject Screening and Selection Process

2.2.1. Subject Phone Screen Interview (see Appendix A)

A brief medical history was conducted by the initial interviewer to exclude potential subjects with physical disorders that could put them at risk during the experimental procedures. Subjects who report having a history of heart problems, epilepsy, uncontrolled hypertension, diabetes, ulcers, thyroid problems, kidney problems, head injury, hearing or visual problems, and respiratory problems (e.g., asthma, chronic obstructive pulmonary disease, cystic fibrosis or lung cancer) were excluded.

2.2.2. Panic Subject Screening

Subjects were recruited from community mental health agencies. Volunteers were accepted for the study if they met DSM-IV criteria for panic disorder with or without agoraphobia. All subjects completed a structured diagnostic interview with a graduate student. Findings from the interview were reviewed by a licensed clinical psychologist.

2.2.3. Diagnostic Reliability

If a consensus diagnosis could not be obtained for a patient, the videotaped diagnostic interview was examined. Subjects were excluded from the study if a consensus diagnosis could not be reached. Twenty percent of the video tapes were randomly reviewed to establish interrater reliability.

2.2.4. Normal Control Screening

Subjects were recruited from the greater Washington metropolitan area and were contacted by phone. A phone screen was conducted to determine their interest and screened for eligibility (see Subject Phone Screen Interview, Appendix A). Those subjects contacted by phone and accepted into the study completed a structured diagnostic interview for Axis I disorders. Subjects that did not meet DSM-IV criteria for any Axis I disorder were eligible for the study.

2.3. Experimental Design

A 2 (Panic Disorder versus. Normal Controls) X 3 (Internal Focus versus. External Focus versus. No Focus) randomized factorial design was employed to test for main effects of group status, instructional set, and their interaction (see Figure 1).

Preliminary power analyses indicated that 90 subjects (15 per cell) should provide sufficient power to detect significant main effects and interactions. Sample size analyses are provided in Table 1 and power analyses are provided in Appendix B.

Figure 1.
Experimental Design

Initial Assessment Measures

a. SCID - I/P
b. SPRAS
c. STAI
d. WCCL
e. PAI
f. ASI
g. APQ-R
h. BAI
i. BDI
j. ACQ
k. BAQ
l. FQ-Ago
m. MI
n. BSQ
o. BVS
p. Panic Freq Int
q. Med Screen Quest

Panic Disorder Subjects | **Nonclinical Control Subjects**

Pre-Manipulation Assessment

a. ACQ
b. API (Symptoms, SUDS, Panic)
c. Physio Measures
d. Focus Quest I

Random Assignment

Internal Focus | **External Focus** | **No Focus**

Training/Assessment Phase

a. ACQ(X1)	a. ACQ(X1)	a. ACQ (X1)
b. API(Symptoms, SUDS, Panic, X2)	b. API(Symptoms, SUDS, Panic, X2)	b. API(Symptoms, SUDS, Panic, X2)
c. Focus Quest II (X5)	c. Focus Quest III (X5)	c. Focus Quest IV(X5)
d. Physio Measures (X5)	d. Physio Measures (X5)	d. Physio Measures (X5)

Post-Manipulation Assessment

a. ACQ
b. API(Symptoms, SUDS, Panic)
c. Physio Measures

Table 1.
Sample Size Analyses

Effect Sizes and Projected Sample Size Requirements for Several Dependent Measures
Analyses are based on alpha = .05 and Power = .70

MEASURE	EFFECT SIZE (d)	REQUIRED SAMPLE SIZE
Acute Panic Inventory (API) (2)	1.70	11
State Trait Anxiety Inventory (STAI) (3)	5.41	36
Fear Questionnaire-Agoraphobic Subscale (3)	1.90	36
Heart Rate (1)	1.55	12
Systolic Blood Pressure (1)	3.30	12
Diastolic Blood Pressure (1)	2.00	12
Overall Average:	2.64	20

1. Obtained from table found in Bystritsky & Shapiro (1992) p. 770.
2. Beitman, Logue, Thomas & Bartels (1992).
3. Carter, Hollon, Carson, & Shelton (1995).

2.4. General Procedure

Subjects were seated in a comfortable chair in a 4m X 4m well-lit room. The room contained a reclinable chair, 2 tables, one swivel chair, a storage cabinet, a computer, 1 Size H tank with a mixture of 35% CO_2 and 65% O_2 and physiological monitoring equipment.

Subjects completed informed consent procedures (see Appendix C) and were assessed for all Axis I, DSM-IV diagnoses through a structured clinical interview (SCID-I/P). Subjects also completed a medical screening questionnaire as a way of detecting cardiovascular or respiratory problems as part of the initial assessment. After completing

the initial procedures, subjects were given a battery of self-report measures that assessed for panic symptoms (Acute Panic Inventory), attentional focus (Focus Questionnaire), panic related ideation (Agoraphobic Cognitions Questionnaire), coping skills (Ways of Coping Checklist), panic related appraisal (Panic Appraisal Inventory), depression (Beck Depression Inventory), subjective anxiety (Beck Anxiety Inventory, Sheehan Patient Rating Anxiety Scale, State-Trait Anxiety Inventory), fear of fear (Anxiety Sensitivity Index, Body Sensations Questionnaire, Body Vigilance Scale, Body Awareness Questionnaire), phobic avoidance (Fear Questionnaire-Agoraphobia Subscale, Mobility Inventory for Agoraphobia), autonomic perception (Autonomic Perception Questionnaire-Revised).

2.4.1. Pre-Manipulation Assessment

After the initial assessment measures have been completed subjects were attached to a heart rate and blood pressure monitor for a 9-minute period. During this monitoring period, subjects filled out the ACQ-1, API-1 and Focus Questionnaire I. After the subjects completed all of the pre-manipulation measures they were randomly assigned to their experimental condition (see Experimental Procedures).

(1) Internal focus condition. Subjects were instructed to close their eyes and pay attention to and count the number of times their heart beats in one minute.

(2) External focus condition. Subjects were instructed to listen to an audio tape recording and were asked to count the number of times the letter G follows the letter Z in a string of letters.

(3) No focus condition. Subjects were provided with no specific instructions regarding their attentional focus.

After the fourth trial each subject inhaled a single vital capacity breath mixture of 35% CO_2 and 65% O_2. Subjects completed post-manipulation procedures and were debriefed.

2.5. Experimental Procedures

2.5.1. Internal Focus Condition

The subjects in this group sat in a comfortable reclinable chair and were asked to silently count their heart beats. This procedure closely follows that used by Ehlers and Breuer (1992). Each subject was given headphones to use during the trial to block out extraneous noise. The subject was instructed to close their eyes and refrain from taking their pulse or doing any other physical manipulations that might facilitate the detection of heart beats. The task takes one minute and was repeated 4 times. After the first trial the subject filled out the ACQ-2, API-2 and Focus Questionnaire II. After trials 2 through 3 the subject only filled out Focus Questionnaire II. After trial 4 the subject filled out the API-3, ACQ-3 and Focus Questionnaire II. Upon completion of trial 4 the subject was asked to take their headphones off their head. Subjects were not provided information about the accuracy of their counting. Next, the subject was instructed about the CO_2 challenge (see Carbon Dioxide Challenge). Subjects were given the following specific instructions:

"For the fifth trial you will close your eyes and pay attention to and count the number of times your heart beats after inhaling a gas mixture consisting of 65% oxygen and 35% carbon dioxide."

2.5.2. External Focus Condition

Subjects in this condition sat in a comfortable reclinable chair and were asked to listen to an audio tape through headphones and count how many times the letter G follows the letter Z in a string of letters. Each subject was given headphones to use during the trial to block out extraneous noise. The task takes one minute and was repeated 4 times. After the first trial the subject filled out the ACQ-2, API-2 and Focus Questionnaire III. After trials 2 through 3 the subject filled out Focus Questionnaire III. After trial 4 the subject filled out the API-3, ACQ-3 and Focus Questionnaire III. Upon completion of trial 4 the subject was asked to take their headphones off their head. Subjects were not provided information about the accuracy of their counting. Next, the subject was instructed about the CO_2 challenge (see Carbon Dioxide Challenge). Subjects were given the following specific instructions:

"For the fifth trial you will listen to an audio tape and count the number of times the letter G follows the letter Z in a string of letters after inhaling a gas mixture consisting of 65% oxygen and 35% carbon dioxide."

2.5.3. No Focus Condition

The subjects within this condition sat in a comfortable reclinable chair for 5 one minute intervals. Each subject was given headphones to use during the trials to block out

extraneous noise. After the first trial the subject filled out the ACQ-2, API-2 and Focus Questionnaire IV. After trials 2 through 3 the subject only filled out Focus Questionnaire IV. After trial 4 the subject filled out the API -3, ACQ-3 and Focus Questionnaire IV. Upon completion of trial 4 the subject was asked to take their headphones off their head. Subjects were not provided information about their attentional focus. After the fourth training trial was completed the subject was instructed about the CO_2 challenge (see Carbon Dioxide Challenge). Subjects were given the following specific instructions: "For the next measurement phase you will inhale a gas mixture consisting of 65% oxygen and 35% carbon dioxide."

2.5.4. Carbon Dioxide Challenge

Subjects in each condition were provided the following information about carbon dioxide and oxygen:

"Both of these gases are found naturally in the air we breathe and are also used and produced by our bodies. Therefore, there is no danger associated with the task. I will answer any questions that you have after the procedure is finished but let me assure you that this gas is completely safe and harmless. Before we perform this procedure I need to measure the vital capacity of your lungs."

After providing information about carbon dioxide and oxygen, each subject's vital capacity was measured. The subjects placed the set of headphones on their head and the blood pressure and heart rate monitor was reset for one minute intervals. Next, the subject was instructed to take a vital capacity breath of the CO_2/O_2 mixture and hold it for five seconds. After they exhaled the gas they repeated their specific experimental

condition for one minute. When the trial was completed the physiological monitoring tape was marked and they were handed the ACQ-4, API-4 and their specific Focus Questionnaire to complete. Subjects then completed post-manipulation assessment measures and were debriefed.

2.6. Assessments

Subjects completed initial assessment measures consisting of a structured clinical interview, and a battery of paper and pencil measures. Subjects' psychophysiological responses were also assessed.

2.6.1. Psychophysiological Measures

Data was continuously monitored by a Critikon Dinamap Vital Signs Monitor, Model 1846 SX. Heart rate, systolic and diastolic blood pressure were measured using this monitoring system. The subjects' blood pressure was measured from their left arm using continuous noninvasive monitoring techniques described by Shapiro et al. (1981). This allows the ability to record systolic (SBP), diastolic blood pressure (DBP) and heart rate (HR) simultaneously at every heartbeat.

2.6.2. Clinical Interview (see Appendix D)

Structured Clinical Interview for Axis I DSM-IV Disorders, Patient Edition (SCID - I/P (Version 2.0).

Current and past DSM-IV (American Psychiatric Association, 1994) diagnoses for Axis I disorders were established through a structured diagnostic interview (First, Spitzer, Gibbon & Williams, 1994).

2.6.3. CO_2/O_2 Equipment

The physiologic stimulus is compressed gas composed of 35% carbon dioxide and 65% oxygen. The gas mixture is stored in a standard H-sized tank. The gas was supplied to a bag with a maximum capacity of 5L. The bag is attached to a hose that supplies the gas to the subject.

2.6.4. Self-Report Measures (see Appendices)

2.6.4.1. Acute Panic Inventory (API, Appendix E)

The API is a 17-item inventory for assessing symptoms of arousal associated with panic attacks (Liebowitz, Gorman, Fyer, Dillon, & Klein, (1984). The API has been used extensively in panic provocation studies (Gorman et al. 1990; Harrison et al., 1989). Subjects rate the severity of each symptom from 0 (absent) to 3 (severe). Examples include, "Did you feel faint?", "Were you afraid of dying?." The API includes a SUDS rating of self-reported anxiety and breathlessness (0 - Not Disturbed at All, 100 - The Worst Imaginable Experience). The API also includes a "Yes" or "No" response question used to assess subjective report of panic in response to the challenge. The presence of a panic attack was determined by a composite index of self-reported distress including: (a) endorsing "yes" on the API panic attack question, (b) reporting a 30 point increase in SUDS from baseline to challenge; and (c) reporting four or more symptoms as moderate to severe during the challenge.

2.6.4.2. Agoraphobic Cognitions Questionnaire (ACQ, Appendix F)

Items on the ACQ are composed of typical catastrophic ideation noted during exposure to anxiety provoking experiences and their consequences (Chambless, Caputo, Bright, and Gallagher, 1984). Predicted consequences of panicking refer to the

consequences such as heart attack, going crazy, and acting foolish. Each item is scored on a 5-point Likert scale ranging from 1 (thought never occurs) to 5 (thought always occurs), of the frequency with which this thought occurred when the client was anxious. The total score is computed by averaging responses across the individual items. Subjects will be asked to "please rate how much you believe that each of the following would occur if you experienced a panic attack when you ...." Each item is rated on a 0 to 4 point scale, where 0 = not at all and 4 = complete belief.

2.6.4.3. Anxiety Sensitivity Index (ASI, Appendix G)

The ASI measures sensitivity to and discomfort with a number of physical sensations commonly associated with anxiety (Reiss, Peterson, Gursky and McNally, 1986). The ASI is a 16 item, self-report inventory in which subjects are requested to rate the extent to which they agree with each item by selecting one of five points on a Likert scale. The scale ranges from "very little" (scored as 0 points) to "very much" (scored as 4 points). The ASI has a high degree of internal consistency, with alpha coefficients ranging from .82 to .91 (Peterson & Reiss, 1992) and has satisfactory test-retest reliability over 3 years (r = .71; Maller & Reiss, 1992).

2.6.4.4. Autonomic Perception Questionnaire-Revised (APQ-R, Appendix H)

The APQ-R (Mandler, Mandler & Uviller, 1958; Shields ,1984;) consists of 30 questions regarding the frequency in which certain bodily symptoms are experienced during anxiety. These questions cover seven areas: heart rate, perspiration, temperature changes, respiration, gastrointestinal disturbance, muscle tension, and blood pressure (e.g., When you feel anxious, how often are you aware of any change in your heart

action?). Each item is rated on a 9 point Likert scale ranging from 1 (not at all true about me), 5 (neutral; not sure) to 9 (very true about me). The scale has good internal consistency, reported at .86 and has acceptable test-retest reliability (r = .80).

2.6.4.5. Beck Anxiety Inventory (BAI, Appendix I)

The BAI is a 21-item self-report inventory for measuring the severity of anxiety in psychiatric populations. Subjects are asked how much they are bothered by each symptom with ratings from not at all, mildly, moderately and severely. The BAI showed high internal consistency (alpha = .92) and test-retest reliability over 1 week = .75.

2.6.4.6. Beck Depression Inventory (BDI, Appendix J)

The BDI is designed to measure the severity of depression in adolescents and adults (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The measure consists of 21 items, or sets of statements, answered on a 0 to 3 scale of severity of depressive problems. Instructions tell the respondent to report on level of depressive symptoms experienced over the preceding week. Each of the 21 items has four sentences, ranging from no complaint to a severe complaint (e.g., "0-I do not feel sad" to "3-I am so sad or unhappy that I cannot stand it"). The internal consistency rated by Cronbach's coefficient alpha (Beck, Steer and Garbin, 1988) for 25 studies ranged from .73 to .95. The mean coefficient alphas for the nine psychiatric populations were .86. The mean coefficient alpha for the 15 nonpsychiatric populations was .81. Pearson correlations for the nonpsychiatric samples ranged from .60 to .83. The test-retest reliability with psychiatric samples had correlations from .48 to .86 and with nonpsychiatric samples from .60 to .90.

The internal consistency is .86 with psychiatric patients (Beck et al., 1988) and .81 with nonpsychiatric subjects (Beck et al., 1988).

2.6.4.7. Body Awareness Questionnaire (BAQ, Appendix K)

The BAQ is an 18-item scale designed to assess self-reported attentiveness to normal nonemotive bodily processes, specifically, sensitivity to bodily cycles and rhythms, ability to detect small changes in normal functioning, and ability to anticipate bodily reactions (Shields, Mallory, & Simon, 1989). Each item is rated on a 7-point Likert scale ranging from 1 (not at all true about me) to 7 (very true about me). It has good convergent and discriminant validity, adequate internal consistency, (Cronbach's alpha consistency = .82), and has acceptable test-retest reliability (r = .80).

2.6.4.8. Body Sensations Questionnaire (BSQ, Appendix L)

The BSQ is a 17-item scale that assesses fear associated with common sensations of autonomic arousal (e.g., heart palpitations, dizziness). Subjects are requested to note the sensations that they experienced during exposure to phobic situations that they found to be particularly distressing. Each item is rated on a 5-point Likert scale, ranging from 1 (not frightened or worried by this sensation) to 5 (extremely frightened by this sensation), which indicates how anxiety-provoking the patient found each sensation. The total score is derived by averaging the individual item ratings. The scale has high internal consistency (Cronbach alpha = .87) and adequate test-retest reliability (r = .67, Chambless, Caputo, Bright, Gallagher, 1984).

2.6.4.9. Body Vigilance Scale (BVS, Appendix M)

The BVS is a 23-item scale that assesses the amount of time and attention an individual spends attending to internal bodily sensations. Subjects are asked whether they are sensitive to internal bodily sensations (e.g., heartbeat, breathing, dizziness) and the amount of time they spend scanning for these sensations. Each item is rated on a 10-point Likert scale, ranging form 0 (no attention) to 10 (extreme attention).

2.6.4.10. Fear Questionnaire-Agoraphobia Subscale. (FQ-Ago, Appendix N)

The Agoraphobia subscale of the Fear Questionnaire (Marks & Mathews, 1979) will be used to assess level of phobic avoidance. The FQ consists of 15 items representing three separate phobic domains (agoraphobia, blood or injury phobia, and social phobia). For each item, the subject rates the degree of avoidance to the object or situation. The five-item agoraphobia subscale (FQ-Ago) has demonstrated adequate psychometric properties and is most widely used self-report measure for assessing agoraphobia in treatment outcome research (Jacobsen, Wilson, & Tupper, 1988).

2.6.4.11. Focus Questionnaire (FQ, Appendix O)

The author constructed Focus Questionnaire assesses the percentage of time a subject is focused on internal cues (e.g., breathing, heart rate, dizziness), external cues (e.g., looking around round room, examining equipment) or no cues in particular.

2.6.4.12. Medical Screening Questionnaire (Appendix P)

A Medical Screening Questionnaire assesses the following areas: personal medical history, personal psychiatric history, family medical history and status of current health.

2.6.4.13. Mobility Inventory for Agoraphobia. (MI, Appendix Q)

The Mobility Inventory for Agoraphobia (Chambless, Caputo, Jasin, Gracely, & Williams, 1985) is a 27-item questionnaire designed primarily to assess avoidance. Instructions require ratings of the severity of avoidance both when alone and when accompanied. The Avoidance When Alone subscale is highly reliable (r=.90), is internally consistent (alpha = .94), and discriminates well between agoraphobic and nonagoraphobic samples (r=.80).

2.6.4.14. Panic Appraisal Inventory (PAI, Appendix R)

The PAI consists of three sections that assess: (1) perceived likelihood of having a panic attack in certain activities or situations; (2) specific threat appraisals related to panic attacks, and (3) coping self efficacy related to having a panic attack. In section one individuals rate the likelihood that they will panic in 15 different activities on a panic continuum from 0 = no chance of panic to 100 = definite panic. In the second section, individuals rate types of panic related appraisals related to three threat domains including physical threat, social threat, and loss of control on a scale from 0 = not at all troubling to 100 = extremely troubling. In section three ratings are made regarding individuals ability to effectively cope with panic attacks in a variety of situations on a scale ranging from 0 = not at all confident to 100 = completely confident (Telch et al. 1989).

2.6.4.15. Panic Frequency Interview (Appendix, S)

The Panic Frequency Interview is a semi-structured clinical interview for assessing the frequency of full and limited symptom panic attacks during the past week and past month.

2.6.4.16. Sheehan Patient Rating Anxiety Scale (SPRAS, Appendix T)

The SPRAS (Sheehan, 1983) is a 35-item self-report scale for assessing the intensity of anxiety symptoms. Each of the 35 symptoms (e.g., shaking or trembling) is rated on a five-point scale ranging from 0 (not at all distressing) to 4 (extremely distressing). The instructions were modified so that symptom ratings were based on a one week time frame.

2.6.4.17. State-Trait Anxiety Inventory (Form X-1, Form X-2,Appendix U)

The State-Trait Anxiety Inventory, Form X-1 (State Anxiety) and the Form X-2 (Trait Anxiety) is a measure of anxiety (Spielberger, Gorsuch, & Lushene, 1970). The State version of the STAI-X asks individuals to indicate how they are feeling right at the moment. The Trait version of the STAI-X asks individuals to indicate how they feel in usual situations.

2.6.4.18. Ways of Coping Checklist (WCCL, Appendix V)

The Revised Ways of Coping Checklist (Vitaliano, Russo, Carr et al., 1985; Aldwin, Folkman, Schafer, Coyne & Lazurus, 1980; Folkman and Lazurus, 1980) assesses the coping style used by individuals when dealing with stress and requires the patient to list a current stressor of concern and then to examine a 42-item Likert-type checklist containing various coping strategies and rate how frequently each is used. Recently, Vitaliano et al. (1985) performed a principal components analysis and showed that five homogeneous coping subscales have been identified by factor analysis: one problem-focused subscale, three emotion-focused subscales (wishful thinking, avoidance, and self-blame), and one subscale combining elements of both (seeking social support).

These scales have shown adequate internal consistency (coefficient alphas range: .82 - .83).

CHAPTER 3

Results

3.1. Analytic Overview

Simple means comparisons were used initially to test for group differences across variables. Analysis of variance (ANOVA) was used to test for main effects of group status, instructional set, as well as interaction effects between group status and instructional set. Regression analyses were conducted to test the relative contribution of coping in predicting the emotional response to the biological challenge.

3.2. Subject Classification and Exclusion

3.2.1. Panic Disorder Subjects

Subjects were classified as having a primary diagnosis of Panic Disorder (PD) using the Structured Clinical Interview for Axis I DSM-IV Disorders (SCID; see section 2.6.2).

One hundred thirty-two subjects contacted the laboratory and were processed through a telephone screening interview. Subjects were initially excluded for the following reasons: (a) eighteen subjects decided not to participate after being read a brief description of the study, (b) twenty-seven subjects showed no indication of having a primary diagnosis of PD during the phone screen interview, (c) seven subjects were excluded because of various medical problems and (d) two subjects were excluded because of age criteria. The remainder of the subjects (n=78) were scheduled for the assessment described in Section 2.6. After completing the assessment, thirty-three subjects were excluded for the following reasons: (a) twelve subjects did not present for

the appointment, (b) eleven subjects did not meet criteria for a primary diagnosis of PD, and (c) five subjects did not complete the assessment, (d) four subjects refused to inhale the O_2/CO_2 mixture, and (e) one subject was excluded for medical reasons. PD subjects current drug usage (see Table 2) shows that 24% are taking benzodiazepines, 13% are taking anti-depressants and 22% are taking a combination of benzodiazepines and anti-depressant medication. Overall, 60% of PD subjects were taking some psychotropic medication. There was no difference across experimental conditions in terms of percentage of PD subjects taking medication $\chi^2(2, N = 90) = .32, p > .05$. Evaluation of current caffeine consumption indicates that 27% consume coffee with an average of 210 mg/24 hrs., 31% consume cola with an average of 100 mg/24 hrs., 29% consume tea with an average of 135 mg/24 hrs. Overall, 64% of PD subjects take caffeine with an average consumption of 445 mg/24 hrs. Whereas 64% of nonclinical control subjects took caffeine but at higher doses (825 mg/24 hrs). The total amount of caffeine consumption was comparable between subjects and across conditions. This analysis had no effect for subject type or condition indicating no significant difference between conditions ($F(2,90) = .73, p = .49$). PD subject's intake of nicotine (see Table 2) shows that in the past 24 hours 16% smoked and smokers averaged 23 cigarettes per 24 hour period. An analysis of variance was performed on the number of cigarettes smoked that found no significant group or condition main effects and no significant group by condition interaction ($F(2,90) = .08, p = .92$).

3.2.2. Nonclinical Control Subjects

One hundred sixty-one volunteers contacted the laboratory and were processed through a telephone screening interview. Subjects were initially excluded for the following reasons: (a) nine volunteers after being read a brief description of the study decided not to participate, (b) eight volunteers were excluded because of medical problems, and (c) two volunteers were not within the acceptable age range for the study. The remainder of eligible volunteers (n = 142) were then placed in a hold file. Forty-seven volunteers from the hold file were matched by gender and age with PD subjects that had already completed the assessment measures. After the assessment one control subject was excluded for a current psychiatric diagnosis, (i.e., obsessive compulsive disorder), and another was excluded for high blood pressure. Nonclinical control subjects current drug usage (see Table 2) shows that none of the subjects are taking benzodiazepines, anti-depressants or a combination of both. In addition, their current caffeine consumption shows that 47% consume coffee with an average of 420 mg/24 hrs., 18% consume cola with an average of 135 mg/24 hrs., 27% consume tea with an average of 270 mg/24 hrs. Seven percent of nonclinical controls reported intake of nicotine (see Table 2). Smoker's averaged 10 cigarettes during the 24 hour period prior to the experiment.

3.3. SCID Diagnoses

The percentage of PD and nonclinical control subjects meeting diagnostic criteria for current and lifetime DSM-IV Axis I Disorders are presented in Table 3.

Table 2.
Group Comparisons of Current Drug and Medication Usage

Drug	Panic Disorder (n=45)		Nonclinical Controls (n=45)	
Benzodiazepines	24%	(n=11)	0%	
Anti-Depressants	13%	(n=6)	0%	
Benzodiazepines and Anti-Depressants	22%	(n=10)	0%	
Avg. psychotropic medication	60%	(n=27)	0%	
Nicotine	16%	(n=7)	7%	(n=3)
Avg. number of cigarettes in past 24 hrs[a]	23		10	
Caffeine				
Coffee consumption	27%	(n=12)	47%	(n=21)
Avg. mg of caffeine per 24 hrs[a]	210		420	
Cola consumption	31%	(n=14)	18%	(n=8)
Avg. mg of caffeine per 24 hrs.[a]	100		135	
Tea consumption	29%	(n=13)	27%	(n=12)
Avg. mg of caffeine per 24 hrs.[a]	135		270	
Total mg of caffeine consumed per 24 hrs[a]	445		825	
Percentage of subjects consuming caffeine	64%	(n=29)	64%	(n=29)

Note. [a] Only subjects who smoke and or take caffeine are included.

Approximately 58% (26/45) of PD subjects met diagnostic criteria for agoraphobia, 24% met criteria for specific phobia (11/45), 18% met criteria for social phobia (8/45), 18% met criteria for generalized anxiety disorder (8/45), 9% met criteria

for obsessive compulsive disorder (4/45) and 9% met criteria for posttraumatic stress disorder (4/45). In addition, 24% of the group met criteria for major depression (11/45), 4% met criteria for dysthymia (2/45). and an additional 2% met criteria for alcohol dependence (1/45). Approximately 27% of the PD subjects (12/45) also had at least one additional co-occurring diagnosis. No nonclinical control subjects met diagnostic criteria for any current DSM-IV Axis I disorder.

Approximately 58% of the PD subjects met lifetime diagnostic criteria for agoraphobia (26/45) , 27% met criteria for specific phobia (12/45), and an additional 18% met criteria for social phobia (8/45). Approximately 58% of PD subjects met criteria for a lifetime diagnosis of major depression (26/45), 27% met criteria for alcohol abuse (12/45), 22 % met criteria for alcohol dependence (10/45) and 18% of the group met criteria for a lifetime diagnosis of other substance abuse/dependence disorders (8/45).

In contrast, the nonclinical control group had no lifetime diagnosis of PD or agoraphobia. Approximately 4 % of nonclinical controls met lifetime diagnostic criteria for specific phobia (2/45), 2% met criteria for social phobia (1/45), and 2% met criteria for PTSD (1/45). In addition, 18% of nonclinical control subjects had a lifetime diagnosis of alcohol abuse (8/45), 11% met criteria for major depression (5/45), 9% met criteria for alcohol dependence and 9% met criteria for cannabis abuse/dependence (4/45).

Table 3.
Group Comparisons of Current and Lifetime Prevalence of DSM-IV Axis I Disorders

Diagnostic Category	Panic Disorder		Nonclinical Controls	
	Current %(N)	Lifetime %(N)	Current %(N)	Lifetime %(N)
Agoraphobia	57.8(26)	57.8(26)	0.0	0.0
Specific Phobia	24.4(11)	26.7(12)	0.0	4.4(2)
Social Phobia	17.8(8)	22.2(10)	0.0	2.2(1)
GAD	17.8(8)	--------	0.0	--------
OCD	4.4(2)	4.4(2)	0.0	0.0
PTSD	4.4(2)	13.3(6)	0.0	2.2(1)
Maj Dep	24.4(11)	55.6(25)	0.0	11.1(5)
Dysthymia	4.4(2)	--------	0.0	--------
Alcohol				
Abuse	2.2(1)	26.7(12)	0.0	17.8(8)
Dependence	0.0	15.6(7)	0.0	11.1(5)
Cannabis				
Abuse	0.0	6.7(3)	0.0	6.7(3)
Dependence	0.0	2.2(1)	0.0	2.2(1)
Cocaine				
Dependence	0.0	2.2(1)	0.0	0.0
Stimulant				
Dependence	0.0	2.2(1)	0.0	0.0
Poly Drug				
Dependence	0.0	4.4(2)	0.0	0.0
Eating Disorder	2.2(1)	6.7(3)	0.0	8.9(4)
Somatoform Disorder	2.2(1)	2.2(1)	0.0	0.0

Note. GAD = generalized anxiety disorder; OCD = obsessive compulsive disorder; PTSD = posttraumatic stress disorder; Maj Dep = major depression.

3.4. Interrater Reliability of Diagnosis

Kappa coefficients were calculated for all diagnostic categories. The diagnoses in Table 4 are representative of the study sample. Given the data in Table 4 there was a high interrater reliability in panic disorder as well as other disorders.

Table 4.
Interrater Reliability of Diagnosis

Diagnostic Category	Kappa Coefficient
Overall Diagnoses	.84 (7/210)
Panic Disorder	1.00
Agoraphobia	.77 (2/18)
Specific Phobia	.82 (1/18)
Social Phobia	.64 (1/18)
Generalized Anxiety Disorder	1.00
Major Depression	1.00
Alcohol Abuse	1.00

3.5. Group Differences on Baseline Variables

3.5.1. Demographic Variables

As shown in Table 5, the two groups did not differ in age, $t(88) = .03$, $p > .05$, gender, $\chi^2(1, N = 90) = 1.00$, $p > .05$, employment status, $\chi^2(3, N = 90) = 5.96$, $p > .05$, or ethnicity, $\chi^2(3, N = 90) = 2.63$, $p > .05$. Control subjects reported a higher level of education, $\chi^2(3, N = 90) = 19.32$, $p < .05$, and significantly fewer controls reported being married, $\chi^2(3, N = 90) = 7.89$, $p < .05$.

3.5.2. Symptom Variables

As expected, PD subjects reported significantly more symptoms of anxiety and depression than nonclinical controls on self report measures (see Table 6). PD subjects were significantly more depressed as indexed by the BDI, $t(88) = 6.88$, $p < .05$, and reported higher levels of anxiety as indexed by the BAI, $t(88) = 10.02$, $p < .05$, and SPRAS, $t(88) = 10.60$, $p < .05$. In addition, PD subjects scored significantly higher on measures of state anxiety (STAI X-1, $t(88) = 6.96$, $p < .05$) and trait anxiety (STAI X-2, $t(88) = 10.16$, $p < .05$). This group also reported greater phobic avoidance (MIACCOM, $t(88) = 5.65$, $p < .05$, MIALONE, $t(88) = 6.54$, $p < .05$, FQ-Ago, $t(88) = 6.19$, $p < .05$).

Table 5.
Group Comparisons on Demographic Variables Between Panic Disorder and Nonclinical Control Subjects

Demographic Variable	Panic Disorder (n=45)	Nonclinical Controls (n=45)	(t)	χ^2
Age				
M	39.18	39.11	(.03)	
SD	12.61	12.31		
Gender				.00
%Male	33.3	33.3		
%Female	66.7	66.7		
Education				19.32*
%HS Grad or Less	26.6	2.2		
%Part College	31.1	13.3		
%College Grad	15.6	33.3		
%Grad Wrk/Prof Sch	22.2	46.7		
Ethnicity				2.63
%Caucasian	88.9	77.8		
%African American	6.7	11.1		
%Hispanic	2.2	2.2		
%Asian	1.0	4.0		
Marital Status				7.89*
%Married	66.7	44.4		
%Widowed	4.4	0.0		
%Divorced/Separated	6.6	15.6		
%Never Married	22.2	40.0		
Employment Status				5.97
%Employed	84.4	82.2		
%Unemployed	0.0	4.4		
%Student	4.4	11.1		
%Homemaker	11.1	2.2		

* $p < .05$

Table 6.
Group Comparisons of Symptom Measures

Variable	Panic Disorder (n=45) M	SD	Nonclinical Controls (n=45) M	SD	t
Symptom Measures					
BDI	15.53	10.67	3.33	5.27	6.88*
BAI	23.27	14.31	1.62	2.32	10.02*
SPRAS	65.42	38.53	4.09	4.62	10.60*
MIACCOM	1.72	.75	1.07	.14	5.65*
MIALONE	2.26	1.12	1.15	.20	6.54*
FQ-Ago	11.02	10.49	1.13	2.20	6.19*
STAI X-1	45.18	13.41	29.29	7.42	6.96*
STAI X-2	50.27	11.59	29.84	6.89	10.16*

Note. BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; SPRAS = Sheehan Patient Rating Anxiety Scale; MIACCOM = Mobility Inventory for Agoraphobia - accompanied; MIALONE = Mobility Inventory for Agoraphobia - alone; FQ-Ago = Fear Questionnaire-Agoraphobia Scale; STAI X-1 = State-Trait Anxiety Inventory - state anxiety; STAI X-2 = State-Trait Anxiety Inventory - trait anxiety.
* $p < .05$

3.5.3. Cognitive Variables

Cognitive self report measures (see Table 7) indicated that PD subjects reported significantly more cognitive distress than nonclinical control subjects. PD subjects reported significantly more agoraphobia-related cognitions as indexed by the ACQ, $t(88) = 8.81$, $p < .05$. They reported being significantly more afraid of the symptoms of anxiety as indexed by the ASI, $t(88) = 9.44$, $p < .05$ and the BSQ, $t(88) = 10.67$, $p < .05$.

The group also reported experiencing more bodily symptoms as indexed by the APQ-R, $t(88) = 8.24$, $p < .05$. They had higher scores on time spent attending to internal bodily sensations as indexed by the BVS, $t(88) = 8.41$, $p < .05$ but were not significantly more attentive to normal bodily processes than nonclinical controls as indexed by the BAQ, $t(88) = 1.88$, $p > .05$. Upon inspection of the BAQ subscales, one subscale showed

Table 7.
Group Comparisons of Cognitive Measures

Variable	Panic Disorder (n=45) M	SD	Nonclinical Controls (n=45) M	SD	t
Cognitive Measures					
ACQ	29.00	10.18	15.39	1.91	8.81*
ASI	30.51	11.61	10.00	8.81	9.44*
APQ-R	161.44	49.89	77.27	47.02	8.24*
BAQ	65.76	23.62	56.40	23.59	1.88
BSQ	44.06	14.23	20.10	4.94	10.67*
BVS	117.76	61.54	27.61	37.20	8.41*
PAI 1	575.22	302.60	52.07	85.89	11.16*
PAI 2	479.56	356.13	66.42	264.00	6.25*
PAI 3	559.78	325.66	1369.11	283.66	12.57*
WCCL	66.04	18.45	55.93	20.71	2.45*

Note. ACQ = Agoraphobic Cognitions Questionnaire; ASI = Anxiety Sensitivity Index; APQ-R = Autonomic Perception Questionnaire-Revised; BAQ = Body Awareness Questionnaire; BVS = Body Vigilance Scale; PAI 1 = Panic Appraisal Inventory Part 1; PAI 2 = Panic Appraisal Inventory Part 2; PAI 3 = Panic Appraisal Inventory Part 3; WCCL = Ways of Coping Checklist.

* $p < .05$

that PD subjects were better able to predict their body's reaction to injury, exertion and sleep pattern than nonclinical controls ($t(88) = 2.13$, $p < .05$). As expected, PD subjects reported a significantly higher perception in the likelihood of having a panic attack in certain situations or activities (PAI 1: $t(88) = 11.16$, $p < .05$). In addition, PD subjects reported a significantly greater amount of specific threat appraisals related to panic attacks (PAI 2: $t(88) = 6.25$, $p < .05$) and a significantly lower self-efficacy in coping with a panic attack (PAI 3: $t(88) = 12.57$, $p < .05$). PD subjects as a group reported using significantly more emotion focused coping skills than problem focused coping skills when compared to nonclinical controls as indexed by the WCCL, $t(88) = 4.47$, $p < .05$ (see Table 9). More specifically, two subscales in the WCCL showed that PD subjects used significantly more emotion focused coping skills than nonclinical controls in coping with stressful encounters as indexed by the wishful thinking subscale, $t(88) = 5.22$, $p < .05$ and the avoidance subscale, $t(88) = 5.30$, $p < .05$ (see Table 8).

Table 8.
Group Comparisons in Coping Style

	Panic Disorder (n=45)		Nonclinical Controls (n=45)		
	M	SD	M	SD	t
Problem Focused Coping	25.16	8.96	27.69	9.28	1.32
Emotion Focused Coping	40.89	12.85	28.24	13.96	4.47*

* $p < .05$

Table 9.
Means and Standard Deviations of Emotion Focused Coping Scales in the Ways of Coping Checklist

Scales	Panic Disorder (n=45)		Nonclinical Controls (n=45)		
	M	SD	M	SD	t
Avoidance	13.91	5.27	7.82	5.63	5.30*
Self Blame	3.49	2.84	2.96	2.64	.92
Social Support	10.91	4.81	10.20	3.96	.77
Wishful Thinking	12.58	4.62	7.27	5.02	5.22*

Note. Avoidance, self blame, social support and wishful thinking are subscales of the emotion focused coping scale.

$p < .05$

3.5.4. Physiological Variables

Baseline physiological measures indicated that PD subjects had a significantly higher baseline heart rate, $t(88) = 2.98$, $p < .05$ but the groups did not differ on systolic blood pressure, $t(88) = 1.21$, $p > .05$, or diastolic blood pressure, $t(88) = .31$, $p > .05$ (see Table 10).

Table 10.
Baseline Physiological Measures for the Panic and Nonclinical Control Samples

Physiological Measure	Panic Disorder (n=45)		Nonclinical Controls (n=45)		
	M	SD	M	SD	t
Heart Rate	68.69	10.57	62.67	8.48	2.98*
Systolic Blood Pressure	119.71	15.16	116.02	13.68	1.21
Diastolic Blood Pressure	69.84	9.46	69.20	9.99	.31

* $p < .05$

3.6. Evaluation of Internal/External Focus

Attentional focus was assessed using the following questionnaires at baseline: (a) Focus Questionnaire, (b) APQ-R, (c) BAQ and (d) BVS. Consistent with prediction, PD subjects were significantly more internally focused (Focus Questionnaire $t(88) = 3.28$, $p < .05$, see Table 11), had a greater frequency of experiencing bodily symptoms (APQ-R, $t(88) = 8.24$, $p < .05$, see Table 7) and scored significantly higher in the amount of time they attend to internal bodily sensations (BVS, $t(88) = 8.41$, $p < .05$, see Table 7). However, PD subjects were not significantly more attentive to normal bodily processes than nonclinical controls (BAQ, $t(88) = 1.88$, $p > .05$, see Table 7). There was also no difference between groups on the amount of external focus or no particular focus reported on the Focus Questionnaire at baseline (see Table 11).

Table 11.
Evaluation of Attentional Focus

Focus Questionnaire	Panic Disorder (n=45) M	SD	Nonclinical Controls (n=45) M	SD	t
External Focus Question	38.44	27.84	50.67	32.19	1.93
Internal Focus Question	31.33	27.35	15.07	19.00	3.28*
No Focus Question	29.56	32.59	34.27	34.23	.67

* $p < .05$

Table 12.
Attentional Focus During the Biological Challenge

Focus Questionnaire	Panic Disorder (n=45) M	SD	Nonclinical Controls (n=45) M	SD	t
External Focus Question	23.00	26.72	33.00	36.56	1.48
Internal Focus Question	71.89	29.04	55.04	36.24	2.43*
No Focus Question	5.11	13.16	11.89	26.59	1.53

* $p < .05$

Repeated measures analyses were used to assess within group changes in the attentional focus over time. Before conducting the repeated measures analyses the four practice trials in Table 13 were averaged because there were relatively few differences in internal focus over these trials. Three sets of comparisons were made: (a) baseline to practice trial, (b) practice trial to CO_2 and (c) baseline to CO_2. The analyses indicated

that baseline focus when compared to practice trials showed no significant differences for PD subjects ($F(1,90) = 1.34$, $p = .25$) but showed significant increases for nonclinical controls ($F(1,90) = 11.25$, $p = .00$). There were also significant increases for both PD subjects ($F(1,90) = 71.47$, $p = .00$) and nonclinical controls ($F(1,90) = 25.00$, $p = .00$) when comparing the practice trial to the CO_2 focus. Similarly, analyses also indicated significant increases from baseline to CO_2 within PD subjects ($F(1,90) = 53.20$, $p = .00$) and nonclinical controls ($F(1,90) = 52.12$, $p = .00$).

Consistent with prediction, PD subjects were significantly more internally focused during the biological challenge than nonclinical control subjects as indexed by the Focus Questionnaire, $t(88) = 2.43$, $p < .05$ (see Table 12). After controlling for baseline levels of internal focus, PD subjects had slightly more internal focus than nonclinical controls during the biological challenge ($F(1, 90) = 3.30$, $p = .07$). There were also no significant differences between the groups in the external focus ($F(1,90) = 1.19$, $p = .28$) or no focus ($F(1,90) = 1.91$, $p = .17$) conditions during the biological challenge when controlling for baseline levels of external focus and no focus.

3.7. Effects of Focus Condition on Fearful Responding

The effect of experimental condition (internal focus, external focus, no focus) on fearful responding was assessed using the following dependent variables: (a) ACQ, (b) API, (c) SUDS, (d) panic, and (e) physiological measures. Residualized change scores were calculated for each dependent variable while controlling for scores at baseline (see Table 14). There was no significant effect for experimental condition on any of the

Table 13.
Between Group Comparisons in Internal Focus Across Experimental Phase

Time Interval	Panic Disorder (n=45) M	SD	Nonclinical Control (n=45) M	SD	t
Baseline	31.33	27.35	15.07	19.00	3.28*
Time 1	36.78	31.89	32.56	40.14	.55
Time 2	39.71	36.51	35.84	41.10	.47
Time 3	37.82	35.86	36.10	41.18	.21
Time 4	37.36	35.53	34.98	40.69	.30
Time 5 (CO_2)	71.89	29.04	55.04	36.24	2.43*

* $p < .05$

variables including physical symptoms (API, $F(2, 90) = .24$, $p = .79$), anxiety ($F(2,90) = .39$, $p = .68$), panic ($\chi^2(2, N = 90) = .41$, $p > .05$), catastrophic ideation (ACQ, $F(2, 90) = .08$, $p = .93$), heart rate ($F(2,90) = 1.65$, $p = .20$), systolic blood pressure ($F(2,90) = 2.13$, $p = .13$) and diastolic blood pressure ($F(2,90) = .00$, $p = 1.0$). Contrary to prediction, the focus manipulation did not appear to have any substantial impact on fearful responding.

Table 14.
Means and Standard Deviations of Dependent Measures Indexing Fearful Responding Across Experimental Conditions

Measure	External Focus (n=30)		Internal Focus (n=30)		No Focus (n=30)	
	M	SD	M	SD	M	SD
ACQ	17.45	5.32	16.83	3.79	17.48	4.03
API	12.97	11.24	13.13	12.56	12.77	9.34
SUDS	35.83	26.91	31.00	28.69	32.67	30.62
HR	66.17	12.53	72.87	16.25	70.20	12.36
SBP	133.63	34.81	141.03	26.26	130.47	22.26
DBP	77.90	16.89	77.77	12.51	75.33	17.19
Panic (%)	(30)		(30)		(37)	

Note. ACQ = Agoraphobic Cognitions Questionnaire; API = Acute Panic Inventory; SUDS = subjective units of distress; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

$p > .05$

3.8. Manipulation Check

The level of attentional focus during the CO_2 challenge among the subjects in the three experimental conditions is illustrated in Table 15. An analysis of variance with post-hoc means comparisons was performed to compare each of the experimental conditions by level of internal, external and no attentional focus. Findings revealed that those subjects assigned to the external focus condition showed significantly more external focus relative to internal or no focus ($F(2,90) = 30.52$, $p = .00$). Comparing the level of internal focus during the biological challenge across conditions showed that the internal focus condition produced significantly greater internal focus relative to external or no

focus ($F(2,90) = 28.88$, $p = .00$). Comparing the level of no specific attentional focus across conditions indicated no significant differences ($F(2,90) = 1.80$, $p = .17$). Overall the subject's response to the three questions asked after the CO_2 challenge indicates that their attentional focus was generally adjusted according to the assigned focus condition thus supporting the integrity of the manipulation.

Table 15.
Comparing the Manipulation of Attentional Focus in Experimental Conditions

Focus question during CO_2	Internal Focus (n=30) M	External Focus (n=30) M	No Focus (n=30) M
Level of external focus	9.00	56.60	18.40
Level of internal focus	87.17	35.73	67.50
Level of no particular focus	3.83	7.67	14.00

Note. Means represent answers to the level of external, internal or no particular focus in the three experimental conditions during the CO_2 challenge.

3.9. Effect of Group Status on Fearful Responding

The effect of group status (PD subject, nonclinical control subject) on fearful responding was assessed using the same dependent variables as above. As predicted, there was a main effect for group status with PD subjects showing greater subjective distress compared to nonclinical controls (see Table 16). There were significant group differences in physical symptoms (API, $F(1, 90) = 10.20$, $p = .002$), anxiety ($F(1,90) = 56.17$, $p = .000$), panic ($\chi^2(1, N = 90) = 26.91$, $p < .05$), and catastrophic ideation (ACQ,

$F(1, 90) = 3.99$, $p = .049$). On the other hand, there were no group differences in physiological responding as indexed by heart rate ($F(1,90) = .68$, $p = .41$), systolic blood pressure ($F(1,90) = .13$, $p = .72$) and diastolic blood pressure ($F(1,90) = 1.01$, $p = .32$).

Table 16.
Means and Standard Deviations of Dependent Measures Indexing Fearful Responding Across Groups

Measure	Panic Disorder (n=43)		Nonclinical Controls (n=43)	
	M	SD	M	SD
ACQ*	19.33	5.00	15.12	1.67
API*	19.05	10.71	6.93	6.32
SUDS*	54.77	21.79	12.56	15.90
HR	72.42	12.97	65.40	13.44
SBP	136.40	31.42	135.14	25.60
DBP	75.54	15.68	78.72	15.94
Panic*	(58)		(7)	

Note. ACQ = Agoraphobic Cognitions Questionnaire; API = Acute Panic Inventory; SUDS = subjective units of distress; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

$p > .05$

Tables 17 and 18 show the means and standard deviations of subjective and physiological responding for PD subjects as well as nonclinical controls across time. Figures 2 and 3 illustrate subjective and physiological responding for PD and nonclinical controls across time.

Table 17.
Means and Standard Deviations of Subjective and Physiological Responding for PD Subjects Across Time

Measure	Baseline		Time 1		Time 4		Time 5 (CO_2)	
	M	SD	M	SD	M	SD	M	SD
ACQ	29.00	10.18	15.68	3.39	15.33	2.93	19.44	5.14
API	8.96	9.10	5.93	6.64	5.73	7.68	19.11	11.35
SUDS	22.44	19.44	14.11	16.07	13.33	15.67	54.11	22.44
HR	68.69	10.57	67.90	10.69	67.93	11.18	73.36	13.43
SBP	119.71	15.16	121.02	17.96	116.07	16.71	136.07	30.95
DBP	69.84	9.46	71.92	11.04	67.24	10.29	75.78	15.42

Note. ACQ = Agoraphobic Cognitions Questionnaire; API = Acute Panic Inventory; SUDS = subjective units of distress; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure

Table 18.
Means and Standard Deviations of Subjective and Physiological Responding for Nonclinical Controls Across Time

Measure	Baseline		Time 1		Time 4		Time 5 (CO_2)	
	M	SD	M	SD	M	SD	M	SD
ACQ	15.39	1.91	14.04	.21	14.00	.00	15.07	1.65
API	.49	.97	.67	1.33	.56	1.14	6.80	6.21
SUDS	2.67	10.95	1.33	4.60	1.78	5.35	12.22	15.65
HR	62.67	8.48	63.18	9.29	63.42	8.17	66.13	13.66
SBP	116.02	13.68	115.73	15.42	113.20	14.84	134.02	25.68
DBP	69.20	9.99	69.11	10.88	67.38	9.02	78.22	15.75

Note. ACQ = Agoraphobic Cognitions Questionnaire; API = Acute Panic Inventory; SUDS = subjective units of distress; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure

Figure 2.
Means of Subjective Measures for Panic Disorder and Nonclinical Controls Across Time

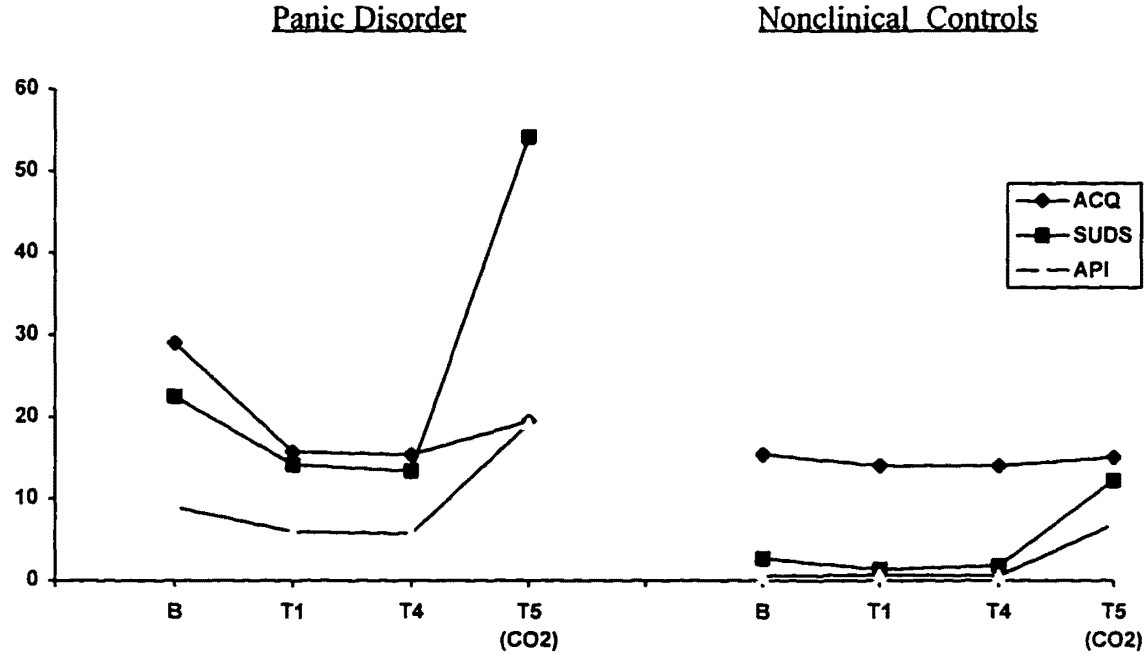
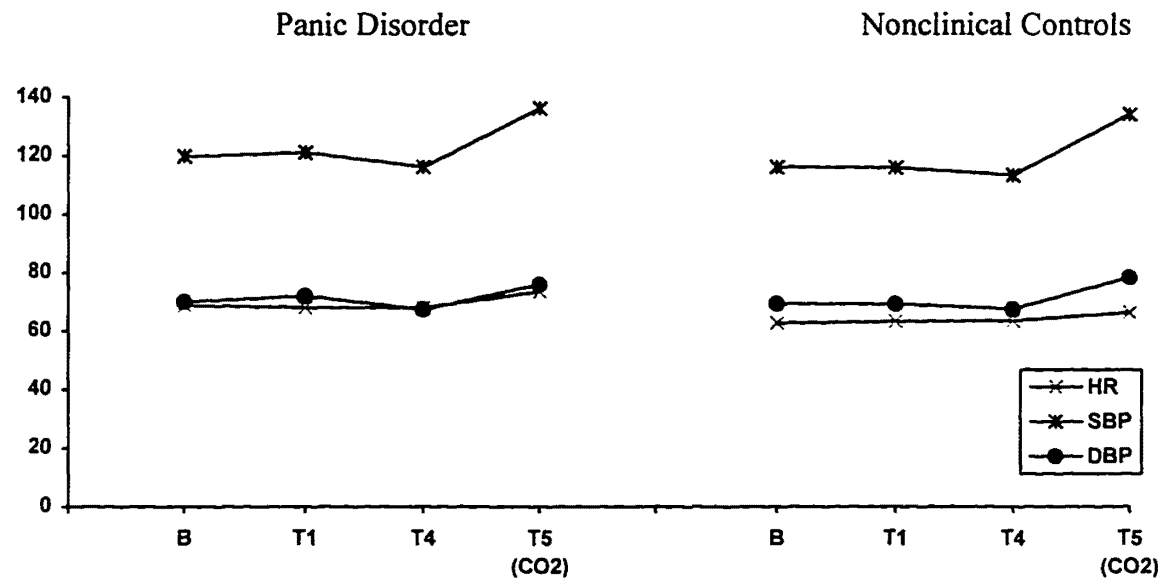


Figure 3.
Means of Physiological Measures for Panic Disorder and Nonclinical Controls Across Time

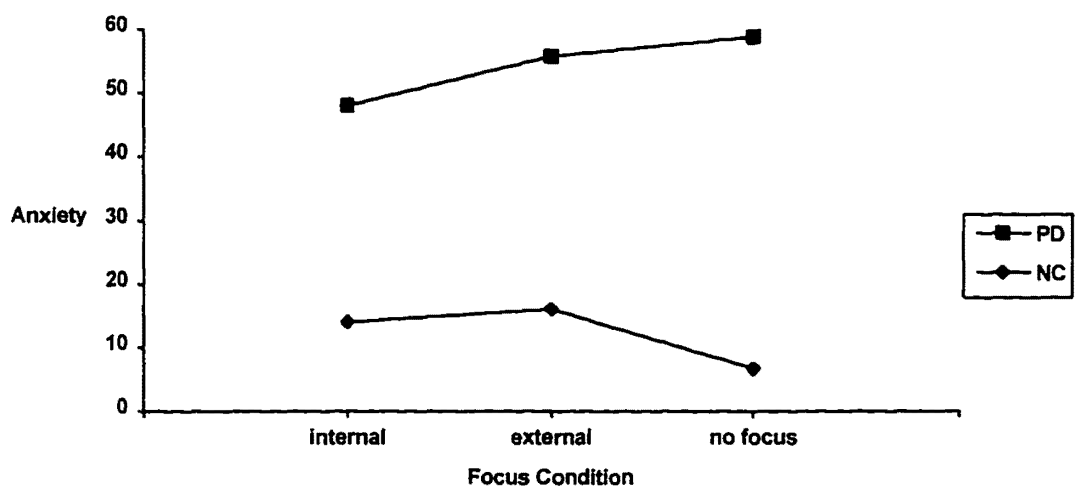


3.10. Interaction of Group Status and Focus Condition on Fearful Responding

The interaction of group status (PD subject, nonclinical control subject) and focus condition (external focus, internal focus and no focus) on fearful responding was assessed to test the main hypothesis of this study. Contrary to prediction there were no significant group by condition interactions for any outcome variable that indicated greater fearful responding for PD subjects in the internal focus condition relative to PD subjects in the external focus condition. There were no significant group by focus condition differences in physical symptoms (API, $F(2, 90) = .17$, $p = .84$), anxiety ($F(2,90) = 1.30$, $p = .28$), panic ($\chi^2(2, N = 90) = .41$, $p > .05$), catastrophic ideation (ACQ, ($F(2, 90) = .77$, $p = .47$),

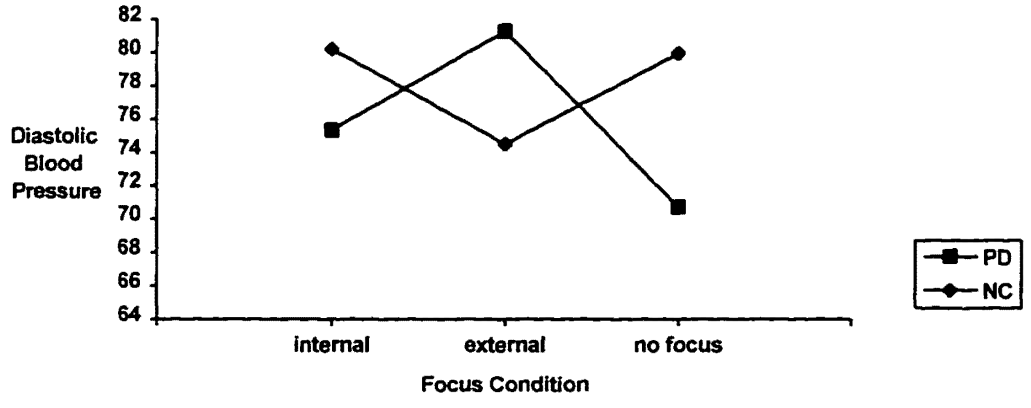
heart rate ($F(2,90) = .26$, $p = .77$), systolic blood pressure ($F(2,90) = .64$, $p = .53$) and diastolic blood pressure ($F(2,90) = 2.05$, $p = .14$). Figure 4 shows anxiety ratings between group status and focus condition and Figure 5 shows diastolic blood pressure. Although the interactions were not significant, Figures 4 and 5 illustrate group by condition measures for subjective (i.e., anxiety) and physiological (i.e., diastolic blood pressure) responding. In addition, Table 19 displays the means and standard deviations of subjective and physiological measures across subject type and experimental condition during the CO_2 challenge.

Figure 4. Mean anxiety ratings of PD subjects and nonclinical control across experimental conditions during the CO_2 challenge.



Note. Group by condition interaction is not significant.

Figure 5. Mean diastolic blood pressure of PD subjects and nonclinical control across experimental conditions during the CO_2 challenge.



Note. Group by condition interaction is not significant

Table 19.
Means and Standard Deviations of Subjective and Physiological Responding Across Experimental Conditions During the CO_2 Challenge

	Panic Disorder			Nonclinical Controls		
Measure	External Focus	Internal Focus	No Focus	External Focus	Internal Focus	No Focus
ACQ						
M	20.17	18.30	19.87	14.73	15.37	15.10
SD	6.44	4.38	4.52	1.16	2.44	1.00
API						
M	18.60	18.87	19.87	7.33	7.40	5.67
SD	13.17	12.93	7.95	4.51	9.45	3.06
SUDS						
M	55.67	48.00	58.67	16.00	14.00	6.67
SD	20.10	25.97	21.00	15.95	20.28	7.24
HR						
M	71.47	75.00	73.60	60.87	70.73	66.80
SD	11.03	17.25	11.89	11.97	15.48	12.27
SBP						
M	135.93	142.00	130.27	131.33	140.10	130.67
SD	42.31	27.75	19.39	26.61	25.62	25.49
DBP						
M	81.27	75.33	70.73	74.53	80.20	79.93
SD	20.11	12.47	11.28	12.72	12.49	20.96
Panic						
(%)	(53)	(47)	(73)	(7)	(13)	(0)

Note. ACQ = Agoraphobic Cognitions Questionnaire; API = Acute Panic Inventory; SUDS = subjective units of distress; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure

3.11. Post-Hoc Analyses--Alternative Hypotheses

As shown in Table 15, the experimental manipulation increased attentional focus in the expected direction. Despite this increase in attention to internal sensations for the PD subjects in the internal focus condition, the hypothesized increase in anxiety during the CO_2 challenge was not found.

There are a variety of reasons why the hypothesized interaction was not obtained. For example, the manipulation of attentional focus may have acted as a distractor task that increased the subject's perception of control. Focusing on the experimental task in the internal and external focus conditions may have distracted subjects from focusing on the physical symptoms associated with the CO_2 challenge. Directing their attentional focus to the experimental task may have given subjects more control over their response to the challenge.

Alternatively, the internal and external focus tasks may have created increased social evaluation concerns. Subjects may have felt that their performance during the experimental task was being evaluated by the experimenter. Subjects with heightened social evaluation concerns may have responded with increased fear to the internal and external focus conditions due to the task demands. For example, subjects were not told whether performance was adequate upon completing the practice trials in the internal or external focus conditions. The PAI 2 social concerns subscale was examined to test this hypothesis. If the task demands increased evaluation anxiety, there should be an interaction between the PAI 2 social concerns subscale and the focus conditions. Those subjects scoring high on social concerns should report greater fear during the external and

internal conditions relative to the no focus condition. Post-hoc analyses using the PAI 2 social concerns subscale did not reveal the hypothesized interaction but only a main effect for social evaluation ($\beta = .60$, $t(88) = 7.06$, $p < .05$) suggesting that increased social evaluation concerns generally increases fearful responding to the challenge.

Another hypothesis is that the independent variables (i.e., PD status, focus condition) might not have been specific enough to show the hypothesized effect. Another way of examining the data is to substitute anxiety sensitivity (AS) for PD status. After all, it is AS that is believed to mediate fearful responding among PD patients as well as nonclinical controls. In addition, evaluation of specific attentional focus questions provides a more specific account of subject's focus compared to condition assignment. In other words, although the manipulation check demonstrated its integrity, the manipulation per se did not guarantee an expected shift in attentional focus. Using self-reported attentional focus questions provides a more exact measure of focus during the CO_2 challenge.

Data was reanalyzed to examine whether AS and attentional focus may yield the originally hypothesized interaction. First, correlations between attentional focus and baseline measures of fearful responding were examined (see Table 20). As shown in Table 20, the baseline internal focus question was moderately correlated to baseline anxiety (r=.40) and physical symptoms (r=.38) and mildly correlated to baseline catastrophic ideation (r=.27). In addition, the baseline external focus question was negatively correlated to baseline anxiety (r=-.22) and physical symptoms (r=-.21). However, the no attentional focus question was not significantly associated with fearful

responding (ps > .05). Overall, this pattern of associations is consistent with expectation and suggests that greater internal focus is associated with increased fear and greater external focus is associated with decreased fear.

Additional correlations were performed between anxiety sensitivity and baseline measures of fearful responding (see Table 21). As shown in Table 21, anxiety sensitivity was highly correlated with catastrophic ideation (r=.72) and physical symptoms (r=.60) and moderately correlated with anxiety (r=.45). Consistent with expectation, subjects who have higher anxiety sensitivity show greater catastrophic ideation, more physical symptoms and greater subjective anxiety.

Table 20.
Correlations of Attentional Focus to Baseline Measures of Fearful Responding, and Physiological Responding

	BDBP	BHR	BSBP	BAPIT	BANX	BACQT	FOCQ1.1	FOCQ1.2	FOCQ1.3
BDBP									
BHR	.2069 (90) P= .050								
BSBP	.6270 (90) P= .000	-.0292 (90) P= .785							
BAPIT	-.0620 (90) P= .562	.2427 (90) P= .021	-.0395 (90) P= .712						
BANX	-.1213 (90) P= .255	.1394 (90) P= .190	-.0305 (90) P= .776	.6212 (90) P= .000					
BACQT	-.0440 (90) P= .681	.2625 (90) P= .012	-.0053 (90) P= .961	.5522 (90) P= .000	.4630 (90) P= .000				
FOCQ1.1	.0911 (90) P= .393	-.1963 (90) P= .064	.0803 (90) P= .452	-.2131 (90) P= .044	-.2207 (90) P= .037	-.1745 (90) P= .100			
FOCQ1.2	-.0600 (90) P= .574	.1156 (90) P= .278	.0836 (90) P= .434	.3774 (90) P= .000	.3984 (90) P= .000	.2692 (90) P= .010	-.2877 (90) P= .006		
FOCQ1.3	-.0498 (90) P= .641	.0966 (90) P= .365	-.1394 (90) P= .190	-.0873 (90) P= .413	-.1036 (90) P= .331	-.0331 (90) P= .757	-.6911 (90) P= .000	-.4874 (90) P= .000	

Note. BDBP = baseline diastolic blood pressure; BHR = baseline heart rate; BSBP = baseline systolic pressure; BAPIT = baseline acute panic inventory total; BANX = baseline anxiety; BACQT = baseline agoraphobic cognitions questionnaire total; FOCQ1.1 = baseline external focus question; FOCQ1.2 = baseline internal focus question; FOCQ1.3 = baseline no focus question.

Table 21.
Correlations of Anxiety Sensitivity to Baseline Measures of Fearful Responding, and Physiological Responding.

	BACQT	BANX	BAPIT	BDBP	BHR	BSBP	ASIT
BACQT							
BANX	.4630 (90) P= .000						
BAPIT	.5522 (90) P= .000	.6216 (90) P= .000					
BDBP	-.0440 (90) P= .681	-.1213 (90) P= .255	-.0620 (90) P= .562				
BHR	.2625 (90) P= .012	.1394 (90) P= .190	.2427 (90) P= .021	.2069 (90) P= .050			
BSBP	-.0053 (90) P= .961	-.0305 (90) P= .776	-.0395 (90) P= .712	.6270 (90) P= .000	-.0292 (90) P= .785		
ASIT	.7174 (90) P= .000	.4506 (90) P= .000	.5959 (90) P= .000	.0272 (90) P= .799	.1203 (90) P= .259	.1925 (90) P= .069	

Note. BACQT = baseline Agoraphobic Cognitions Questionnaire total; = BANX = baseline subjective anxiety; BAPIT = baseline Acute Panic Inventory total; BDBP = baseline diastolic blood pressure; BHR = baseline heart rate; BSBP = baseline systolic blood pressure; ASIT = Anxiety Sensitivity Index total.

Regression analyses were conducted to examine the main effects and interaction of AS and level of internal focus during the biological challenge. The baseline measure of each dependent variable was entered as a covariate along with the ASI, the degree of internal focus and the interaction between the ASI and focus question. Findings indicated a main effect for AS in predicting the API ($\beta = .32$, $t(87) = .55$, $p < .05$) with high AS subjects reporting more physical symptoms than low AS subjects during the biological challenge. In addition, analyses revealed a significant interaction for subjective anxiety ($\beta = .29$, $t(86) = 2.14$, $p < .05$, see Figure 4) and catastrophic ideation ($\beta = .31$, $t(87) =$

2.67, $p < .05$, see Figure 5). Consistent with hypotheses, subjects with high AS reported greater subjective anxiety when they were more internally focused compared to high AS when they were externally focused. Whereas the low AS subjects showed no significant difference whether they were internally or externally focused. Figure 4 shows the same pattern of interaction for catastrophic ideation with high AS subjects displaying greater subjective anxiety when they are more internally focused. In sum, these findings are consistent with the original hypothesis as they indicate that high AS subjects will respond with greater subjective anxiety and catastrophic ideation when their attentional focus is internal.

Similar regression analyses investigated the level of external focus during the biological challenge and found that there was a main effect for AS ($\chi^2(3, N = 90) = 26.53$, $p < .05$) indicating that subjects with high AS scores were more likely to panic. Findings also indicated that subjects with high AS reported more physical symptoms ($\beta = .32$, $t(87) = 3.36$, $p < .05$) and had greater anxiety ($\beta = .49$, $t(87) = 5.75$, $p < .05$) during the biological challenge but there were no significant interactions.

Analyses using level of no attentional focus during the biological challenge showed a main effect for AS predicting panic ($\chi^2(3, N = 90) = 26.74$, $p < .05$) as subjects with high AS were more likely to panic. In addition, high AS subjects reported a greater frequency of physical symptoms ($\beta = .32$, $t(87) = 3.36$, $p < .05$), significantly more subjective anxiety ($\beta = .49$, $t(87) = 5.75$, $p < .05$) and a greater frequency of catastrophic ideation ($\beta = .55$, $t(88) = 6.10$, $p < .05$). However, no interactions were significant.

Additional regression analyses investigated whether there was an interaction between anxiety sensitivity and focus condition. The baseline measure of each dependent variable was entered as a covariate along with the ASI, the focus condition and the interaction between the ASI and focus condition. Findings indicated a main effect for AS in predicting physical symptoms (β = .32, $t(87) = 3.36$, $p < .05$), subjective anxiety (β = .49, $t(87) = 5.75$, $p < .05$) and a significant interaction for catastrophic ideation (β = .22, $t(87) = 2.00$, $p < .05$). Similar analyses were performed for physiological variables and no significant interaction was found for any of the variables ($p > .05$).

In sum, there was an expected interaction for AS by focus condition in the internal focus condition when examining subjective anxiety and catastrophic ideation. Similar analyses for the external and no focus conditions failed to find an interaction for any subjective or physiological variable.

3.12. Analysis Covarying Marital Status and Education

Because there were group differences on two demographic variables (i.e., marital status, education), the data were reanalyzed for all major analyses while covaring for marital status and education. The analyses produced a highly similar pattern of findings. Five findings differed in the reanalysis. The wishful thinking subscale was associated with reporting physical symptoms (β = .21, $t(83) = 2.43$, $p < .05$) but was no longer associated with diastolic blood pressure (β = -.12, $t(84) = -1.28$, $p > .05$). In addition, the social support (β = -.12, $t(84) = -1.29$, $p > .05$), and wishful thinking (β = -.16, $t(84) = -1.71$, $p > .05$) subscales were no longer associated with systolic blood pressure and the

self-blame subscale was no longer associated with catastrophic ideation (β = -.07, $t(84)$ = -.70, $p > .05$).

Figure 4. Subjective anxiety during 35% CO_2 for high and low AS by high and low internal focus.

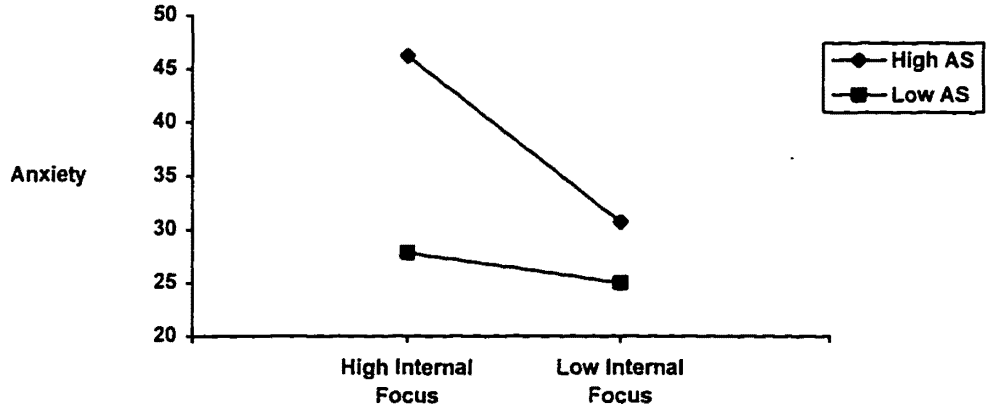
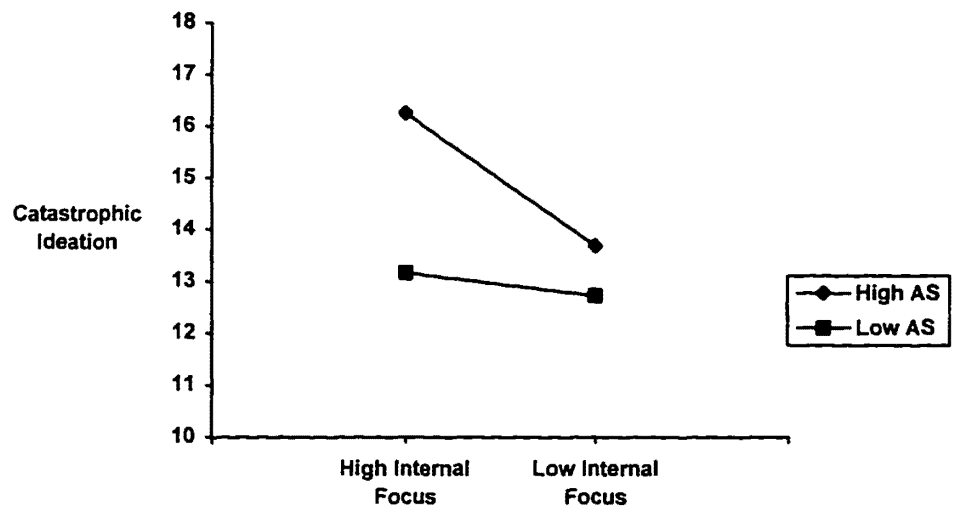


Figure 5. Catastrophic ideation during 35% CO_2 for high and low AS by high and low internal focus.



3.13. Coping Skills Predicting Fearful Responding to the CO_2 Challenge

Multiple regression analyses were conducted to test the relative contributions of coping skills in predicting fearful responding during the CO_2 challenge. The analyses assessed the relative contribution of the WCCL scale, its two main subscales (i.e., problem focused coping and emotion focused coping) and the four subscales within the emotion focused coping scale. Three separate sets of regression analyses were conducted for each dependent variable (i.e., the WCCL overall scale, simultaneous regression of the

problem focused coping scale and the emotion focused coping scale, and simultaneous regression of the four subscales that comprise the emotion focused coping scale). The findings for the four subscales are found in Tables 22, 23, and 24.

Examination of subjective measures of fear indicated that the overall WCCL scale did not predict anxiety($R^2 = .33$, $\beta = .13$, $p > .05$). The problem focused coping scale did not predict anxiety ($\beta = -.17$, $t(86) = -1.73$, $p > .05$) but the emotion focused coping scale did predict anxiety indicating that using more emotion focused coping skills was associated with greater anxiety ($\beta = .30$, $t(86) = 2.91$, $p < .05$). In addition, the avoidance subscale significantly predicted anxiety of was a significant variable indicating that using avoidance as a coping skill was associated with greater anxiety ($\beta = .29$, $t(84) = 2.15$, $p < .05$).

The overall WCCL scale did not predict catastrophic ideation (ACQ: ($R^2 = .30$, $\beta = -.00$, $p > .05$). Similarly, the problem focused coping ($\beta = -.15$, $t(86) = -1.49$, $p > .05$) and emotion focused coping ($\beta = .15$, $t(86) = 1.29$, $p > .05$) subscales did not predict catastrophic ideation. However, the avoidance ($\beta = .41$, $t(84) = 3.07$, $p < .05$) and self blame ($\beta = -.21$, $t(84) = -1.96$, $p < .05$) subscales were significant predictors suggesting that using avoidance and self blame coping skills is associated with an increase in catastrophic ideation.

The overall WCCL scale predicted physical symptoms as indexed by the API ($R^2 = .45$, $\beta = .19$, $p < .05$). The problem focused coping scale did not predict physical

symptoms (β = .02, $t(86)$ = .26, $p > .05$) but the emotion focused coping scale (β = .19, $t(86)$ = 2.03, $p < .05$) did predict physical symptoms. This finding shows that using more emotion focused coping skills was associated with experiencing a greater amount of physical symptoms during the manipulation. The four subscales of the emotion focused coping scale shown in Table 13 did not predict physical symptoms.

The overall WCCL scale did not predict the occurrence of panic ($\chi^2(1, N = 90)$ = 3.44, $p > .05$). In addition, the problem focused coping subscale ($\chi^2(1, N = 90) = 4.83$, $p < .05$) and the emotion focused coping subscale ($\chi^2(1, N = 90) = 11.67$, $p < .05$) simultaneously and uniquely predicted panic. This finding indicates that using either problem focused coping or emotion focused coping was associated with a higher incidence of panic. Of the four emotion focused coping subscales, only the avoidance subscale predicted panic ($\chi^2(1, N = 90) = 9.49$, $p < .05$) indicating that using avoidance coping skills was associated to panic.

Table 22.
Summary of Simultaneous Regression Analyses for WCCL Subscales Predicting Fearful Responding to the CO_2 Challenge (N=90)

Variable	B	SE B	β
		SUDS	
Baseline SUDS	.66	.15	.43*
Avoidance	1.33	.62	.29*
Self Blame	-1.52	1.14	-.15
Social Support	.09	.61	.01
Wishful Thinking	.48	.71	.09
		API	
Baseline API	.81	.13	.57*
Avoidance	.21	.23	.12
Self Blame	-.15	.41	-.04
Social Support	.13	.22	.05
Wishful Thinking	.23	.26	.11
		ACQ	
Baseline ACQ	.24	.04	.54*
Avoidance	.29	.09	.41*
Self Blame	-.34	.17	-.21*
Social Support	.06	.09	.06
Wishful Thinking	-.16	.11	-.20
		Panic	
Avoidance	-.21	.07	9.49*
Self Blame	.21	.19	1.23
Social Support	-.11	.07	.90
Wishful Thinking	.01	.07	1.01

Note. Avoidance, self blame, social support and wishful thinking are subscales of the emotion focused coping scale. ACQ = agoraphobic cognitions questionnaire; API = acute panic inventory; SUDS = subjective units of distress.

* $p < .05$

Analysis of the WCCL subscales was performed to evaluate whether the subscales are distinct from phobic avoidance measures. Correlations were performed between the subscales of the WCCL, the Mobility Inventory and the Fear Questionnaire - Agoraphobic Scale (see Table 23). Findings indicated that the Avoidance subscale was only moderately correlated with the FQ-Ago ($r = .36$), the MI - accompanied ($r=.35$) and the MI - alone ($r=.36$). The Wishful Thinking subscale was also moderately correlated with the FQ-Ago ($r = .32$), the MI - accompanied ($r=.32$) and the MI - alone ($r=.35$). Correlations also indicated that the measures of phobic avoidance were highly correlated with each other ($r= .78$ - $.94$). These findings suggest that the WCCL Avoidance and Wishful Thinking subscales are measuring something reasonably different from phobic avoidance.

The relationship of the WCCL scale and the physiological measures of fearful responding were also examined. The overall WCCL as well as the two main subscales did not predict any physiological variable ($p > .05$). Analyses of the four subscales of the emotion focused coping scale (see Table 24) showed that the social support ($\beta = .22$, $t(84) = 2.29$, $p < .05$) and wishful thinking ($\beta = -.30$, $t(84) = -2.16$, $p < .05$) predicted systolic blood pressure. These findings indicate that seeking social support and using wishful thinking as coping strategies was associated with an increase in systolic blood pressure. In addition, high scores on the wishful thinking subscale predicted increased diastolic blood pressure ($\beta = -.29$, $t(84) = -1.93$, $p = .05$).

Table 23.

Correlation of the Four Emotion Focused Coping Subscales with Measures of Phobic Avoidance

	AVOID	BLAME	SOCSPT	WISHTHNK	FQTOT	MIACCOM	MIALONE
AVOID							
BLAME	.5459 (90) P= .000						
SOCSPT	.2145 (90) P= .042	.3013 (90) P= .004					
WISHTHNK	.7377 (90) P= .000	.5571 (90) P= .000	.3663 (90) P= .000				
FQTOT	.3599 (90) P= .000	.1365 (90) P= .199	.0031 (90) P= .977	.3195 (90) P= .002			
MIACCOM	.3519 (90) P= .001	.1654 (90) P= .119	-.0776 (90) P= .467	.3184 (90) P= .002	.7767 (90) P= .000		
MIALONE	.3612 (90) P= .000	.1146 (90) P= .282	-.0694 (90) P= .516	.3536 (90) P= .001	.9366 (90) P= .000	.8227 (90) P= .000	

Note. AVOID = avoidance subscale; BLAME = self blame subscale; SOCSPT = social support subscale; WISHTHNK = wishful thinking subscale; FQTOT = fear questionnaire -agoraphobic scale total; MIACCOM = mobility inventory - accompanied; MIALONE = mobility inventory - alone.

Table 24.
Summary of Simultaneous Regression Analyses for WCCL Subscales Predicting Physiological Fearful Responding to the CO_2 Challenge (N=90)

Variable	B	SE B	β
		Heart Rate	
Baseline HR	.90	.11	.64*
Avoidance	.19	.28	.09
Self Blame	-.63	.52	-.12
Social Support	-.01	.28	.00
Wishful Thinking	-.28	.33	-.11*
		Systolic Blood Pressure	
Baseline SBP	1.07	.17	.55*
Avoidance	.43	.62	.10
Self Blame	-.20	1.14	-.02
Social Support	1.42	.62	.22*
Wishful Thinking	-1.56	.72	-.30*
		Diastolic Blood Pressure	
Baseline DBP	.71	.15	.44*
Avoidance	.53	.36	.21
Self Blame	.24	.67	.04
Social Support	-.04	.36	-.01
Wishful Thinking	-.83	.43	-.29

Note. Avoidance, self blame, social support and wishful thinking are subscales of the emotion focused coping scale. HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

* $p < .05$

In sum, the problem focused coping scale did not predict any subjective measure of fearful responding except panic, whereas the emotion focused coping scale predicted anxiety, physical symptoms and panic but did not predict catastrophic ideation. In addition, the avoidance subscale appears to account for much of the predictive power of the emotion focused coping scale as it specifically predicted anxiety, catastrophic

ideation, and panic. The overall WCCL as well as the two main subscales did not predict any physiological variable ($p > .05$). However, the wishful thinking subscale predicted systolic and diastolic blood pressure and the social support subscale predicted systolic blood pressure.

CHAPTER 4

Discussion

4.1. Overview of Findings

The principal aim of this study was to assess whether attentional focus affects fearful responding. Specifically, the present study tested a cognitive model of panic (Clark, 1986) to determine whether attentional focus affects fearful responding to heightened somatic cues. Consistent with predictions, PD subjects were more internally focused before the experimental manipulation and were more internally focused during the biological challenge. PD subjects also showed more subjective but not physiological distress during the biological challenge. These findings support the fear of fear hypothesis, which predicts that subjects who are fearful of physical sensations show heightened emotional responding when exposed to a biological challenge. The findings also indicated that subjects' level of emotion focused coping skills predicted fearful responding to the biological challenge.

Several hypotheses were not supported. Contrary to prediction there was no effect for condition on fearful responding. Specifically, there was no significant effect for condition on any of the subjective or physiological variables indicating that the focus manipulation did not have any substantial impact on fearful responding. There was also no group by condition interactions indicating greater fearful responding for PD subjects in the internal focus condition relative to PD subjects in the external focus condition.

4.2. Specific Findings

4.2.1. Evaluation of Internal and External Focus

The literature on somatic awareness (Pennebaker, 1982) and interoception (Ehlers, 1993) in PD patients led to the hypothesis that PD subjects would show greater internal focus at baseline compared to nonclinical control subjects. These studies also led to the hypothesis that PD subjects would show greater internal focus during the biological challenge compared to nonclinical control subjects regardless of condition.

Baseline measures relevant to internal focus found that PD subjects were significantly more internally focused. In addition, the baseline internal focus question and the baseline API were moderately correlated. This suggests that PD subjects were focused more on physical symptoms than nonclinical controls. Consistent with Pennebaker's work indicating that internal focus will increase the perception of physical sensations (1982; Pennebaker, et al., 1985) these findings indicate that PD subjects were naturally more internally focused than nonclinical controls.

Ehler's (1993) research on interoception and PD has found that although PD patients experience higher levels of somatic symptoms such as palpitations, dyspnea, or dizziness. PD patients respond with anxiety when they experience these symptoms and tend to avoid situations in which the symptoms occur. According to Ehlers (1993) PD patients also rate bodily symptoms associated with anxiety or panic as more dangerous and appear to be better than controls in detecting bodily processes. Because PD patients have an internal attentional focus it is suggested that they have a greater probability of

perceiving theses physiological changes (Pennebaker, Gonder-Frederick, Cox, and Hoover, 1985).

Ehlers (1993) research on interoception is consistent with our findings during the biological challenge. We found that PD subjects were more internally focused during the biological challenge compared to nonclinical control subjects regardless of condition. It is likely that PD patients were more internally focused because the biological challenge brought about the interoceptive cues that signal the fear of having a panic attack.

4.2.2. Effects of Focus Condition on Fearful Responding

It was hypothesized that directing a subject's attentional focus to internal bodily sensations would increase fearful responding. Specifically, subjects in the internal focus condition would show greater fearful responding compared to those subjects in the external and no focus conditions. The initial findings failed to support this hypothesis as there were no significant effects for condition on any of the subjective or physiological variables assessing fearful responding. These findings run contrary to the cognitive model of panic (Clark, 1986) which implies that as PD patients focus more on internal bodily sensations, they should catastrophically misinterpret them as threatening.

Although the focus manipulation did not have any substantial impact on fearful responding, after performing a manipulation check, findings indicated that subjects in the internal focus condition were more internally focused compared to those in the external and no focus conditions. Similarly, subjects in the external focus condition were more externally focused compared to the internal and no focus conditions. What may have happened during the manipulation is that even though subjects were more internally

focused they may have felt somewhat safe with the experimenter and being in a controlled setting. Consistent with this idea, Carter et al. (1995) found that PD patients exposed to CO_2 without their "safe person" reported greater distress than patients exposed with their "safe person".

4.2.3. Effects of Group Status on Fearful Responding

Extensive literature on PD subject's response to a biological challenge led to the hypothesis that PD subjects would show greater subjective and physiological distress during the CO_2 challenge compared to nonclinical control subjects. Findings supported this hypothesis as PD subjects showed greater subjective distress compared to nonclinical controls but failed to support group differences in physiological responding. These findings are consistent with other studies that have indicated that PD patients respond with greater anxiety to a variety of biological challenge procedures relative to nonanxious controls (Barlow, 1988; Margraf, Ehlers, & Roth, 1986a; Sanderson & Wetzler, 1990).

Gorman, Papp, Martinez, Goetz, Hollander, Liebowitz, and Jordan (1990) examined the anxiogenic effects of 35% CO_2 inhalation in patients with PD , social phobia and normal controls. They found that 13 of 26 panic patients (50%), 8 of 22 social phobics subjects (36%) and 3 of 14 normal controls (21%) had panic attacks to 35% CO_2 inhalation. Papp, Klein, Martinez, Schneier, Cole, Liebowitz, Hollander, Fyer, Jordan, and Gorman (1993) examined the anxiogenic effects of 35% CO_2 inhalation in patients with PD, social phobia and normal controls. They found 13 of the 18 panic patients (72%), six of the 20 patients with social phobia (30%) and one of the 23 normal controls (4%) panicked in response to the 35% CO_2 inhalation. Fyer, Uy, Martinez,

Goetz, Klein, Fyer, Liebowitz and Gorman (1987) examined the anxiogenic effects of 35% CO_2 inhalation in PD patients and normal controls. They found that five (63%) of the eight PD patients and none of the five normal control subjects experienced panic attacks with 35% CO_2 inhalation. In the current study 26 of the 45 PD subjects (58%) and three of the 45 nonclinical controls (7%) experienced a panic attack with the 35% CO_2 inhalation. The findings of the present study are consistent with the percentage of PD patients reporting panic to the CO_2 inhalation in other studies. Meaning that the challenge procedure used in this study is equivalent to the challenge procedures used in other studies.

We failed to find differences between PD patients and nonclinical controls on physiological measures (i.e., heart rate, diastolic blood pressure and systolic blood pressure) in response to the biological challenge. The study's failure to detect differences on the psychophysiological measures is consistent with other challenge studies that have measured autonomic arousal (Beck & Scott, 1988; Salkovskis & Clark, 1990). Those studies indicated that subjects experience virtually the same change in autonomic responding during the challenge. This finding is consistent with the cognitive theory (Clark, 1986) of panic. Cognitive theory implies that it is individual's catastrophic misinterpretation of benign sensations as threatening that leads to a panic attack.

4.2.4. Interaction of Group Status and Focus Condition on Fearful Responding

The extensive literature on somatic awareness (Pennebaker, 1982; Pennebaker et al., 1985; Pennebaker & Hoover, 1984) and the cognitive theory of panic (Clark, 1986)

led to the hypothesis that there would be an interaction of group status and focus condition on fearful responding. Specifically PD subjects in the internal focus condition were expected to show heightened fearful responding relative to PD subjects in the external focus condition.

Findings indicated that there was no significant group by condition interactions for any outcome variable. The suspicion was that the independent variables (i.e., PD status, focus condition) may not have been specific enough to show the hypothesized effect. The data was reexamined substituting anxiety sensitivity for PD status. This was done because AS is believed to mediate fearful responding among PD patients as well as nonclinical controls. For example, challenge research has revealed that healthy subjects with high ASI scores respond like panic patients to voluntary hyperventilation, reporting more physical sensations and more anxiety than do subjects with low ASI scores (Holloway & McNally, 1987).

The process of examining AS involved substituting the internal focus question for condition because it would provide a more specific account of subject's focus compared to condition assignment. Although the manipulation demonstrated its integrity, it did not guarantee an expected shift in attentional focus. Using the self-reported attentional focus questions provided a more exact measure of focus during the CO_2 challenge.

Consistent with other research, findings indicated that the ASI predicted anxious responding to the carbon dioxide challenge (Rapee, Brown, Antony, and Barlow, 1992a). Findings indicated that subjects who had higher anxiety sensitivity showed greater catastrophic ideation, more physical symptoms and greater subjective anxiety. More

importantly, subjects with high AS reported greater subjective anxiety when they were more internally focused compared to high AS when they were externally focused. Whereas the low AS subjects showed no significant difference whether they were internally or externally focused. These findings are consistent with the original hypotheses in that they indicate that high AS subjects will respond with greater subjective anxiety and catastrophic ideation when their attentional focus is internal.

Group by condition interaction effects on diastolic blood pressure are illustrated in Figure 5. Consistent with similar findings of previous research, Figure 5 shows that nonclinical controls performing the mental tracking task in the external focus condition had a decrease in diastolic blood pressure. Whereas nonclinical controls in the internal focus condition who were silently counting their heart beats had an increase in diastolic blood pressure (Lacey, Kagan, Lacey & Moss, 1963). Lacey et al. (1963) reports that changes in response patterns produced by stimulus changes are simple quantitative modifications: one physiological response is greater in one stimulus condition than in another, while another response shows the reverse effect. Lacey et al. (1963) have called these physiological responses "directional fractionation" where the direction of change in one physiological variable is contrary to what might be expected from Cannon's interpretation (1926) where there is an over-all sympathetic activation by stress. Lacey (1967) also found that attentive observation of the external environment typically results in cardiac deceleration, cardiac stabilization, and either a blood pressure decrease or a marked diminution of pressure increase.

4.2.5. Coping Skills a Predictor of Fearful Responding to the CO_2 Challenge

The literature on coping skills in panic disorder patients led to the hypothesis that PD subjects will report greater use of emotion focused coping compared to nonclinical controls. An additional hypothesis was that the level of emotion focused coping would predict fearful responding to the biological challenge. Findings indicated that the problem focused coping subscale did not predict any subjective measure of fearful responding except panic, whereas the emotion focused coping scale predicted anxiety, physical symptoms and panic. In addition, the avoidance subscale appears to account for much of the predictive power of the emotion focused coping scale as it specifically predicted anxiety, catastrophic ideation and panic. The overall WCCL as well as the two main subscales did not predict any physiological variable. The findings of the study support the hypothesis that PD subjects use more emotion focused coping skills and that these are predictive of fear under stress.

Folkman, et al.(1986) found that coping behavior was an important mediator between life stress and mental health. PD can be brought about by stressful life experiences that tax coping resources that can later affect an individual's mental health. Maladaptive coping was found to be associated with both mental disorders (Woodruff, Goodwin & Guze, 1974) and adaptation to stressful life events (Cohen & Lazarus, 1979; Moos, 1982). The present study provides evidence that the greater amount of emotion focused coping is suggestive of a coping style that may predispose an individual to mental illness.

The findings of this study are consistent with research indicating that PD patients use less problem-focused coping and more emotion focused coping (Vitaliano et al., 1987). Vitaliano et al. (1987) found that PD patients exhibited relatively fewer beliefs in the efficacy of their available problem-focused strategies and exhibited more non-action oriented fantasy and ideation such as wishful thinking. They also found that PD patients cope more often by seeking affiliation and support, and by using rumination and cognitive avoidance. The authors suggest that in PD, individuals are not managing the stressor (i.e., panic attack) but rather the emotional result of the stressor. Because of continuously focusing on the emotions involved in a panic attack, a vicious emotional cycle may occur. During this cycle the individual is constantly dealing with the emotions caused by a panic attack and does not venture out of usual types of coping (i.e., style of coping) to learn a different way to try to solve the problem (i.e., panic).

Vollrath and Angst's (1993) also found that the emotion-focused avoiding coping "style" is more prevalent among subjects with panic, whether they react to their symptoms or whether they deal with stressful events in general. In addition, Telch, Brouillard, Telch and Argas (1989) found that PD subjects with agoraphobia have lower levels of perceived panic-coping efficacy suggesting that PD subjects with agoraphobia may not be effective problem solvers. These findings give further evidence that PD subjects with agoraphobia may have a certain coping "style" (i.e., emotion focused coping) that may make them feel ineffective in dealing with panic. In sum, the findings of coping studies in PD suggest that maladaptive coping may play an important role in the etiology and expression of the disorder.

4.3. Contributions to the Literature

This study is unique in that it is the first study to examine the effect of attentional focus on fearful responding in PD. The study also offered a test of cognitive models of panic in terms of whether attentional focus affects fearful responding to heightened somatic cues. The findings of this study support the current literature that individuals with high anxiety sensitivity and are more internally focused will have a tendency to panic. This study extends previous work by examining the differences in attentional focus to bodily cues under conditions of normal arousal as well as heightened arousal.

A unique aspect of the study is that it is the first study to have investigated PD subjects coping style in response to a biological challenge. An individual's belief about their abilities to cope with a stressor (i.e., panic attack) appears to influence the outcome of their attack. Individuals who use more emotion focused coping skills, specifically avoidance skills, may have a greater likelihood of panicking. This finding may also help in understanding the strength of the beliefs that particular symptoms lead to particular consequences and how coping style plays a critical role in the process. Current literature has suggested that identification of maladaptive coping strategies may assist clinicians in designing therapeutic strategies and in monitoring therapeutic change in patients (Maxim and Hunt, 1990).

This study's findings further contributed to the literature by illustrating that the WCCL subscales may be predictive of panic. Correlations of avoidance and wishful thinking subscales to measures of phobic avoidance indicate that the two subscales are measuring something reasonably different from phobic avoidance. In addition, the

avoidance subscale appears to account for much of the predictive power of the emotion focused coping scale as it specifically predicted anxiety, catastrophic ideation and panic.

The present study offers a comparison of PD and nonclinical control subjects on several measures of autonomic perception and body vigilance. Findings indicated that PD and nonclinical control subjects differed significantly on measures of autonomic perception and body vigilance. This is a significant contribution because not many studies have examined comparisons of autonomic perception and body vigilance with the APQ-R and the BVS in PD patients and nonclinical controls. The present study is the first study to provide a complete evaluation of different dimensions of body perception and vigilance.

4.4. Study Limitations

Because the main hypothesized interactions did not occur, post-hoc analyses were performed to explore why the expected effect did not occur. Caution must always be used in interpreting post-hoc findings. However, the post-hoc analyses were consistent with the original hypotheses of the study. Additional studies of anxiety sensitivity by focus condition are needed to replicate this finding.

Another limitation of this study is that the WCCL asked the individuals in the study their coping strategies with previous events and not specifically to the biological challenge. Future work should include examination of the type of coping used during the biological challenge. Another limitation of the study is that its findings on coping style can not be generalized to the daily coping strategies of PD patients. Additional studies are needed to evaluate coping in PD patients across different stressors to make a valid

determination on type coping style. Finally, the WCCL only measured limited number of coping strategies. Future research on coping in panic should use the WCCL along with other coping instruments to evaluate whether the WCCL needs to be revised for a clinical population.

4.5. Implications and Future Directions

This study has shown that anxiety sensitivity may be a better marker than PD status to examine subject's response to biological challenges. Examining fearful responding in reference to AS may also be a better discriminator between subjects. In addition, future studies should not only examine diagnostic category but also level of AS in subjects.

This study shows that PD subjects have an over reliance on emotion focused coping skills. Given this information, cognitive behavioral treatment for panic disorder may not be as effective with individual's who are more prone to use emotion focused coping skills when dealing with a panic attack. It is also suggested that cognitive behavioral skills training be supplemented with ways to counteract the PD patient's reliance on past emotion focused coping skills.

The cognitive model of panic posits that panic attacks result from the catastrophic misinterpretation of benign internal bodily sensations. Individuals can experience and misinterpret sensations such as heart palpitations as a heart attack, dizziness as a prelude to passing out, or derealization as going crazy. As the positive feedback loop is formed, catastrophic misinterpretations increase anxiety and intensify bodily sensations until they lead to a panic attack. The cognitive behavioral treatment paradigm involves abolishing

the persistent tendency to misinterpret bodily sensations catastrophically. This paradigm incorporates behavioral treatment (e.g., exposure to feared situations), but emphasizes the importance of altering the catastrophic misinterpretation of bodily sensations.`

Finally, this study has indicated that the type of coping style when engaging in treatment may influence the outcome of the treatment. Specifically, differences in coping style may affect treatment response. Furthermore, this study implies that teaching alternative problem solving skills in cognitive behavioral therapy may be a factor that mediates treatment outcome. Future studies should assess baseline coping skills before cognitive behavioral therapy and reassess coping skills at the termination of treatment. Other studies should also assess PD subjects coping response across stressors.

References

Adler, C. M., Craske, M. G., & Barlow, D. H. (1987a, November). The use of modified relaxation in the experimental induction of anxiety and panic. Paper presented at the annual meeting of the Association for Advancement of Behavior Therapy, Boston.

Adler, C. M., Craske, M. G., & Barlow, D. H. (1987b). Relaxation-induced panic (RIP): When resting isn't peaceful. Integrative Psychiatry, 5, 94-112.

Aldwin, C., Folkman, S., Schaefer C., Cohen, F., & Lazarus, R. (1980, September). Ways of coping: A process measure. Paper presented at meeting of American Psychological Association, Montreal, Canada.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Barlow, D. H. (1988). Anxiety and its disorders: The nature and treatment of anxiety and panic. New York: Guilford.

Beck, A. T., & Emery, G. (1985). Anxiety disorders and phobias: A cognitive perspective. New York: Basic Books, Inc.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. E., & Erbaugh, J. K. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 561-571.

Beitman, B. D., Logue, M. B., Thomas, A. M., & Bartels, K. (1992). Response to 35 % CO_2 in patients with chest pain and angiographically normal coronary arteries. International Journal Psychiatry in Medicine, 22, 197-203.

Boulenger, J. P., Uhde, T. W., Wolff, E. A., III, Post, R. M. (1984). Increased sensitivity to caffeine in patients with panic disorders: Preliminary evidence. Archives of General Psychiatry, 41, 1067-1071.

Bourdon, K. H., Boyd, J.H., Rae, D.S., Burns, B. J., Thompson, J. W., & Locke, B. Z. (1988). Gender differences in phobias: Results of the ECA community survey. Journal of Anxiety Disorders, 2, 227-241.

Bradwejn, J., Koszycki, D., Couetoux du Tertre, A., Bourin, M., Palmour, R., & Ervin, F. (1992). The cholecystokinin hypothesis of panic and anxiety disorders: A review. Journal of Psychopharmacology, 6, 345-351.

Bystritsky, A. & Shapiro, D. (1992). Continuous physiological changes and subjective reports in panic patients: A preliminary methodological report. Biological Psychiatry, 32, 766-777.

Carter, M. M., Hollon S. D., Carson, R., & Shelton, R. C. (1995). Effects of a safe person on induced distress following a biological challenge in panic disorder with agoraphobia. Journal of Abnormal Psychology, 104, 156-163.

Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, R. (1984). Assessment of fear of fear in agoraphobics: The Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. Journal of Consulting and Clinical Psychology, 52, 1090-1097.

Charney, D. S., Heninger, G. R. & Breier, A. (1984). Noradrenergic function in panic attacks. Archives of General Psychiatry, 42, 223-243.

Charney, D. S., Heninger, G.R., & Jatlow, P. I. (1985). Increased anxiogenic effects of caffeine in panic disorders. Archives of General Psychiatry, 42, 233-243.

Clark, D. M. (1986). A cognitive approach to panic. Behaviour Research and Therapy, 24, 461-470.

Clark, D. M., Salkovskis, P. M., & Anastiasiades, P. (1990). Cognitive mediation of lactate induced panic. Paper presented at the 24th annual AABT convention, San Francisco (Abstract).

Cohen, F., & Lazarus, R. S. (1979). Coping with the stress of illness. In G.C. Stone, N.E. Adler, F. Cohen (Eds), Health Psychology (pp 217-254). San Francisco: Jossey-Bass.

Cowley, D. S., & Arana, G. W. (1990). The diagnostic utility of lactate sensitivity in panic disorder. Archives of General Psychiatry, 47, 277-284.

de Montigny, C. (1989). Cholecystokinin tetrapeptide induces panic-like attacks in healthy volunteers: Preliminary findings. Archives of General Psychiatry, 46, 511-517.

Ehlers, A. (1993). Somatic symptoms and panic attacks: A retrospective study of learning experiences. Behaviour Research and Therapy, 31, 269-278.

Ehlers, A., & Breuer, P. (1992). Increased cardiac awareness in panic disorder. Journal of Abnormal Psychology, 101, 371-382.

Ehlers, A., Breuer, P., Dohn, D., & Fiegenbaum, W. (1995). Heartbeat perception and panic disorder: Possible explanations for discrepant findings. Behaviour Research and Therapy, 1, 69-76.

Ehlers, A., Margraf, J. & Roth, W. T. (1988a). Selective information processing, interoception, and panic attacks. In Hand, I. & Wittchen, H. U. (Eds.), Panic and phobias 2 (pp. 129-148). Berlin: Springer.

Ehlers, A., Margraf, J., Roth, W. T., Taylor, C. B., & Birbaumer, N. (1988b). Anxiety induced by false heart rate feedback in patients with panic disorder. Behaviour Research and Therapy, 26, 1-11.

First, M. B., Spitzer, R. L., Gibbon, M. & Williams, J. B. (1994). "Structured Clinical Interview for Axis I DSM-IV Disorders - Patient Edition (SCID-I/P, Version 2.0). Biometrics Research Department, New York State Psychiatric Institute, New York.

Folkman, S., & Lazarus, R. S. (1980). An analysis of coping in a middle-aged community sample. Journal of Health and Social Behavior, 21, 219-239.

Folkman, S., Lazarus, R. S., Gruen, R. J., DeLongis A. (1986). Appraisal, coping, health status, and psychological symptoms. Journal of Personality and Social Psychology, 50, 571-579.

Fyer, M., Uy, J., Martinez, J., Goetz, R., Klein, D., Fyer, A., Liebowitz, M., & Gorman, J. (1987). CO_2 challenge of patients with panic disorder. American Journal of Psychiatry, 144, 1080-1082.

Garssen, B., van Veenendaal, W., & Bloemink, R. (1983). Agoraphobia and the hyperventilation syndrome. Behaviour Research and Therapy, 21, 643-649.

Goldstein, A. J., & Chambless, D. L. (1978). A reanalysis of agoraphobia. Behavior Therapy, 9, 47-59.

Gorman, J., Papp, L., Martinez, J., Goetz, R., Hollander, E., Liebowitz, M., & Jordan, F. (1990). High-dose carbon dioxide challenge test in anxiety disorder patients. Biological Psychiatry, 28, 743-757.

Griez, E., Lousberg, H., van den Hout, M. A., van der Molen, G. M. (1987). CO_2 vulnerability in panic disorder. Psychiatry Research, 20, 87-96.

Griez, E., Zandbergen, J., Pols, H., de Loof, C. (1990). Response to 35% CO_2 as a marker of panic in severe anxiety. American Journal of Psychiatry, 147, 796-797.

Holloway, W., & McNally, R. (1987). Effects of anxiety sensitivity on the response to hyperventilation. Journal of Abnormal Psychology, 96, 330-334.

Jacobson, N. S., Wilson, L., & Tupper, C. (1988). The clinical significance of treatment gains resulting from exposure-based interventions for agoraphobia: A reanalysis of outcome data. Behavior Therapy, 19, 539-554.

Katerndahl, D. A., & Realini, J. P. (1993). Lifetime prevalence of panic states. American Journal of Psychiatry, 150, 246-249.

Keyl, P. M., & Eaton, W. W. (1990). Risk factors for the onset of panic disorder and other panic attacks in a prospective, population-based study. American Journal of Epidemiology, 131, 301-311.

King, R., Margraf, J., Ehlers, A., & Maddock, R. (1986). Panic disorder - Overlap with symptoms of somatization disorder.

Klein, D. F. (1993). False suffocation alarms, spontaneous panics, and related conditions: An integrative hypothesis. Archives of General Psychiatry, 50, 306-317.

Lacey, J. (1967). Somatic response patterning and stress: Some revisions of activation theory. In M. H. Appley & R. Trumbull (Eds.), Psychological Stress (pp. 14-42) New York: Appleton-Century-Crofts.

Lacey, J. I., Kagan, J., Lacey, B.C.,& Moos, H.A. (1963). The viscereal level: Situational determinants and behavioral correlates of autonomic response patterns. In P. H. Knapp, (Ed.), Expression of the emotions in man. New York: International Universities, (pp. 161-196).

Lazarus, R. & Folkman, S. (1984). Stress, Appraisal and Coping. New York: Springer.

Liebowitz, M. R., Gorman, J. M., Fyer, A. J., Dillon, D. J., & Klein, D. F. (1984). Effects of naloxone on patients with panic attacks. American Journal of Psychiatry, 141, 955-997.

Marks, I. M. & Mathews, A. M. (1979). Brief standard self-rating for phobic patients. Behaviour Research and Therapy, 17, 263-267.

Mandler, G., Mandler, J. M., & Uviller, E. T. (1958). Autonomic feedback: The perception of autonomic activity. Journal of Abnormal and Social Psychology, 56, 367-373.

Maxin, P., & Hunt, D. (1990). Appraisal and coping in the process of patient change during short-term psychotherapy. Journal of Nervous and Mental Disease, 178, 235-241.

McNally, R. J. (1994). Panic disorder: A critical analysis. New York: Guilford Press.

Moos, R. H. (1982). Coping with acute health crises. In T. Million, C. Green, Meagher (Eds), Handbook of clinical health psychology (pp 129-151). New York: Plenum.

Papp, L. A., Klein, D. F., Martinez, J., Schneier, F., Cole, R., Liebowitz, M. R., Hollander, E., Fyer, A. J., Jordan, F., & Gorman, J. M., (1993b). Diagnostic and substance specificity of carbon-dioxide-induced panic. American Journal of Psychiatry, 150, 250-257.

Pennebaker, J. W. (1982). The Psychology of Physical Symptoms. New York: Springer.

Pennebaker, J. W., Gonder-Frederick, L., Cox, D. J., & Hoover, C. W. (1985). The perception of general vs. specific visceral activity and the regulation of health-related behavior. In E. S. Katkin & S. B. Manuck (Eds.), Advances in behavioral medicine, Vol.1, (pp. 165-198). Greenwich, CT: JAI Press.

Pennebaker, J. W. & Hoover, C. W. (1984). Visceral perception versus visceral detection: Disentangling methods and assumptions. Biofeedback Self Regulation, 9, 339-352.

Pitts, F. N., Jr., & McClure, J. N., Jr. (1967). Lactate metabolism in anxiety neurosis. New England Journal of Medicine, 277, 1329-1336.

Pohl, R., Yeragani, V. K., Balon, R., Ortiz, A., & Aleem, A. (1990). Isoproterenol-induced panic: A beta-adrenergic model of panic anxiety. In J. C. Ballenger (Ed.), Neurobiology of panic disorder (pp. 107-120). New York: Wiley-Liss.

Rainey, J. M., Pohl, R. B., Williams, M., Kritter, E., Freedman, R. R., & Ettedugi, E. (1984). A comparison of lactate and isoproterenol anxiety states. Psychopathology, 17, 74-82.

Rapee, R. (1986). Differential response to hyperventilation in panic disorder and generalized anxiety disorder. Journal of Abnormal Psychology, 95, 24-28.

Rapee, R., Brown, T., Antony, M., & Barlow, D. (1992a). Response to hyperventilation and inhalation of 5.5% carbon dioxide-enriched air across the DSM-III-R anxiety disorders. Journal of Abnormal Psychology, 101, 538-552.

Rapee, R., Mattick, R., & Murrell, E. (1986). Cognitive mediation in the affective component of spontaneous panic attacks. Journal of Behavior Therapy and Experimental Psychiatry, 17, 245-253.

Rapee, R. M., Tefler, L. A., & Barlow, D. H. (1991). The role of safety cues in mediating the response to inhalations of CO_2 in agoraphobics. Behaviour Research and Therapy, 29, 353-355.

Razran, G. (1961). The observable unconscious and the inferable conscious in current Soviet psychophysiology: Interoceptive conditioning, semantic conditioning, and the orienting reflex. Psychological Review, 68, 81-147.

Reiss, S., Peterson, R. A., Gursky, D.M., & McNally, R. J. (1986). Anxiety sensitivity, anxiety frequency and the prediction of fearfulness. Behaviour Research and Therapy, 24, 1-8.

Robins, L. N., & Regier, D. A. (Eds.). (1991). Psychiatric disorders in America. New York: Free Press.

Sanderson, W. C., Rapee, R.M., & Barlow, D. H. (1989). The influence of an illusion of control on panic attacks induced via inhalation of 5.5% carbon dioxide-enriched air. Archives of General Psychiatry, 46, 157-162.

Schachter, S., & Singer, J. E. (1962). Cognitive, social, and physiological determinants of emotional state. Psychological Review, 69, 379-399.

Schmidt, N. B., & Telch, M. J. (1994). Role of fear of fear and safety information in moderating the effects of voluntary hyperventilation. Behavior Therapy, 25, 197-208.

Shapiro, D. Greenstadt, L. & Lane. (1981). Tracking-Cuff system for beat-to-beat recording of blood pressure. Psychopharmacology, 18, 129-136.

Shields, S. (1984). Reports of bodily change in anxiety, sadness, and anger. Motivation and Emotion, 8, 1-21.

Shields, S. A., Mallory, M. E., & Simon, A. (1989). The body awareness questionnaire: Reliability and validity. Journal of Personality Assessment, 53, 802-815.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Spitzer, R.L., Williams, J. B. W., Gibbon, M., & First, M. B. (1992). The Structured Clinical Interview for DSM-III-R (SCID): I. History, rationale, and description. Archives of General Psychiatry, 49, 624-629.

Taylor, C. B., King, R., Ehlers, A., Margraf, J., Clark, D., Hayward, C., Roth, W.t., & Argas, W. S. (1987). Treadmill exercise test and ambulatory measures in panic attacks. American Journal of Psychiatry, 143, 478-482.

Telch, M. J., Brouillard, M., Telch, C. F., & Agras, W. S., & Taylor, C. B. (1989). Role of cognitive appraisal in panic related avoidance. Behaviour Research and Therapy, 27, 373-383.

Uhde, T. W. (1990). Caffeine provocation of panic: A focus on biological mechanisms. In J. C. Ballenger (Ed.), Neurobiology of panic disorder (pp.219-242). New York: Wiley-Liss.

Uhde, T., Boulenger, J., Post, R., Siever, L., Vittone, B., Jimerson, D. and Post, R. (1984a). Fear and anxiety: Relationship to nonadrenergic function. Psychopathology, 17, 8-23.

Uhde, T. W., Boulenger, J. P., Vittone, B. J., Siever, L., & Post, R. M. (1985). Human anxiety and nonadrenergic function: Preliminary studies with caffeine, clonidine and yohimbine. In Proceedings of the Seventh World Congress of Psychiatry. New York: Plenum Press.

van den Hout, M. A. (1988). The explanation of experimental panic. In S. Rachman & J. D. Maser (Eds.), Panic: Psychological perspectives (pp.237-257). Hillsdale, NJ: Erlbaum.

van den Hout, M. A., & Griez, E. (1985). Peripheral panic symptoms occur during changes in aveolar carbon dioxide. Comprehensive Psychiatry, 26, 381-387.

Vitaliano, P., Russo, J., Carr, J, Maiuro, R., & Becker, J. (1985). The ways of coping checklist : Revision and psychometric properties. Multivariate Behavior Research, 20, 3-26.

Vitaliano, P., Katon, W., Russo, J., Maiuro, R., Anderson, K. & Jones, M. (1987). Coping as an Index of Illness Behavior in Panic Disorder. Journal of Nervous and Mental Disease, 175, 78-84.

Vollrath, M. & Angst, J. (1993). Coping and illness behavior among young adults with panic. Journal of Nervous and Mental Disease, 181, 303-308.

Woodruff, R. A., Goodwin, D. W., Guse, S. B. (1974). Psychiatric diagnosis. New York: Oxford University Press.

APPENDICES

POWER ANALYSIS, ASSESSMENT INSTRUMENTS, AND OTHER EXPERIMENTAL FORMS

1. Power and Sample Size Analyses Appendix B

Assessment battery (alphabetical order):

1. Acute Panic Inventory (APQ) Appendix E
3. Agoraphobic Cognitions Questionnaire (ACQ) Appendix F
4. Anxiety Sensitivity Index (ASI) Appendix G
5. Autonomic Perception Questionnaire-Revised (APQ-R) Appendix H
6. Beck Anxiety Inventory (BAI) Appendix I
7. Beck Depression Inventory (BAI) Appendix J
8. Body Awareness Questionnaire (BAQ) Appendix K
9. Body Sensations Questionnaire (BSQ) (Appendix L
10. Body Vigilance Scale (BVS) Appendix M
11. Fear Questionnaire-Agoraphobic Subscale (FQ-Ago) Appendix N
12. Focus Questionnaire (FQ) Appendix O
13. Medical Screening Questionnaire Appendix P
14. Mobility Inventory for Agoraphobia (MI) Appendix Q
15. Panic Appraisal Inventory (PAI) Appendix R
16. Panic Frequency Interview Appendix S
17. Sheehan Patient Rating Anxiety Scale (SPRAS) Appendix T
18. State-Trait Anxiety Inventory (STAI) Appendix U
18 Structured Clinical Interview for Axis I DSM-IV Disorders Appendix D
19. Ways of Coping Checklist (WCCL) Appendix V

Experimental forms:

1. Consent Form Appendix C
2. Demographic Information Survey Appendix X
3. Study Protocol Appendix W
3. Subject Phone Screen Interview Appendix A

Appendix A Subject Phone Screen

I. Panic Disorder Subjects

Hi, I am _____, a doctoral student in clinical psychology at the Uniformed Services University of the Health Sciences. I am calling to ask whether you are interested in participating in a research study. The purpose of the study is to examine the differences between panic disorder patients and control subjects in emotional, physiological and cognitive responding. The study involves coming in for one 3-5 hour visit where you will fill out some questionnaires, be interviewed by a clinical psychology doctoral student, and complete several tasks. If you have a diagnosis of panic disorder you will be eligible for a non-drug treatment procedure. There is no cost for evaluation or treatment in this study. None of the procedures are harmful or dangerous in any way. For instance there are no needles or blood draws or taking of any drugs. Do you think you might be interested in participating?

If "NO", say "Thank you anyway for your time. Good-bye.

If "YES", continue with the next part of the phone screen.

II. Normal Control Subjects

Hi, I am _____, a doctoral student in clinical psychology at the Uniformed Services University of the Health Sciences. I am calling to ask whether you are interested in participating in a research study. The purpose of the study is to examine the differences between panic disorder patients and control subjects in emotional, physiological and cognitive responding. The study involves coming in for one 3-5 hour visit where you will fill out some questionnaires, be interviewed by a clinical psychology doctoral student, and complete several tasks. If you have a diagnosis of panic disorder you will be eligible for a non-drug treatment procedure. There is no cost for evaluation or treatment in this study. None of the procedures are harmful or dangerous in any way. For instance there are no needles or blood draws or taking of any drugs. For your participation, you will be compensated with a 40 dollar check. Do you think you might be interested in participating?

If "NO", say "Thank you anyway for your time. Good-bye.

If "YES", continue with the next part of the phone screen.

PHONE SCREEN INTERVIEW

Interviewer: _____ Date: _____

Name: _____

Address: _____

_____ (Include zip code)

Home Phone: _____ Work Phone: _____

Sex: M F Age: _____

1. Have you ever received treatment for an anxiety disorder and/or panic attacks?
   Y N

2. Have you ever had a sudden surge of anxiety, when you suddenly felt frightened, anxious or extremely uncomfortable?
   Y N

3. After the episode did you worry that there might be something terribly wornng with you, like you were having a heart attack, losing control or were going crazy?
   Y N

4. During the episode/attack what types of symptoms were you experiencing? (Ask number of attacks in past week and past month, whether they worry about additional attacks and whether they avoid anything at the present time.)

5. Have you ever seen anyone (counselor, therapist, doctor) for any emotional or psychiatric problems?
   Y N

6. Have you ever been hospitalized for an emotional or psychiatric problem?
   Y N

7. Have you ever been hospitalized for alcohol or drug rehabilitation?
   Y N

8. Are you currently taking any psychiatric medications? (Get the name, dosage, reason for the drug and length of time taking it.)

Y N

9. Are you currently taking any other medications? (Get the name, dosage, reason for the drug and length of time taking it.)

Y N

10. Do you have any heart problems?

Y N

11. Do you have high blood pressure?

Y N

12. Do you have any significant medical problems such as diabetes, ulcers, thyroid problems, kidney troubles?

Y N

13. Have you ever had any head injuries?

Y N

14. Do you have a history of epilepsy or have you had a seizure?

Y N

15. Do you have a history of respiratory problems like asthma, chronic obstructive pulmonary disease (COPD), CF, lung cancer?

Y N

16. Do you have any type of hearing problems?

Y N

17. Do you have any serious visual impairment?

Y N

18. Do you have any other condition that might be affecting your current health status?

Y N

(For Women):

19. Are you currently pregnant?

Y N

(Last, check the calander and make an appointment time with the subject. Give them the number to call for any additional information or appointment changes (301-295-3651).

Appendix B Power and Sample Size Analyses

Power and Sample Size Estimates

An evaluation of the current literature was conducted to determine the sufficient power for detecting main and interaction effects for the major dependent variable (fearful responding).

Power analyses were used to determine whether an n = 90 would insure a reasonable level of power (approximately .75). Power is calculated for these effects in Table 1. These analyses were based on the following parameters: (a) alpha = .05, (b) effect size = .35, and (c) N = 90 (overall); n = 15 (cell). Analyses indicate that a main effect size of .35 would give a power of .70 (Cohen, 1988; Tables 8.3.16). The estimated effect size is consistent with similar studies that indicate large effect sizes (Papp et al., 1993). The sample of 15 subjects per cell should provide sufficient power (assuming an effect size of .35) to detect a significant main effects interaction.

Appendix C Consent Form

USUHS Research Consent Form

Study Title: Investigation of differences in cognitive and emotional responding between patients with panic disorder and normal controls.

Principal Investigator: Dr. N. Bradley Schmidt, Ph.D.

1. Purpose of the study:

You are invited to participate in a study that is examining the differences between panic disorder patient and normal controls in emotional and cognitive responding. There will be 90 subjects in this study that will take place at the Uniformed Services University of the Health Sciences.

2. Procedures Involved in the Study:

First there will be a structured interview asking about your past and current emotional and medical history. At various times during the study you will be asked to complete questionnaires designed to measure your response before, during and after the experimental manipulation. In addition to filling out the questionnaires, you will be asked to complete a physiological assessment that will involve having your heart rate and blood pressure prior to and after inhalation of a gas that consists of a higher concentration of oxygen and carbon dioxide than you usually breathe. This mixture is not harmful or dangerous in any way. The total time to complete each assessment including the questionnaires and the physiological measures will be approximately four hours.

3. Possible Discomfort and Risks Involved:

Please note that videotaping of some parts of the assessment and treatment procedures will be conducted for reliability purposes. These videotapes will be securely stored in a locked room and viewed only by Dr. Schmidt and authorized project personnel under Dr. Schmidt's supervision. All tapes will be erased after the study is completed.

Risks to participants are extremely minimal. There are no foreseeable risks associated with the self-report assessment procedures. There are no foreseeable risks associated with the assessment of heart rate or blood pressure.

The behavioral assessment procedures are safe and have been used for many years in a variety of clinical settings. There are no foreseeable risks associated with the inhalation of oxygen and carbon dioxide gas. You have the right to refuse or discontinue participation during this or any other portion of the assessment process. In addition, Dr. Schmidt will be available in the event of crisis.

If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Office of Research Administration at the

Uniformed Services University of the Health Sciences, Bethesda, MD 20814 at (301) 295-3303. This office can review the matter with you, can provide information about your rights as a subject, and may be able to identify resources available to you. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

4. Benefits Involved:

You will receive an extensive evaluation of you emotional history. In addition, you will be compensated with a 40 dollar check that will be mailed to you after your visit. Your participation will help us in our efforts to design more effective treatments for people who suffer from panic disorder and agoraphobia.

5. Use of Research Results:

Information from your participation may appear in medical or psychological journals. Your individual identity will not be connected to any published reports.

6. Special Circumstances:

Your decision whether or not to participate will not prejudice future relations with the Uniformed Services University of the Health Sciences. If you decide to participate, you are free to discontinue participation at any time without prejudice.

If you have any questions at a future time, Dr. Schmidt will be happy to answer them. He can be reached at (301) 295-3270.

Any information that is obtained in connection with this study and that can be identified with you will remain strictly confidential and will be disclosed only with your permission.

You are making a decision whether or not to participate. Your signature indicates that you have read the information provided above and have decided to participate. You may withdraw at any time without prejudice after signing this form should you choose to discontinue participation. You will receive a signed copy of this consent form if so desired.

Subject's Signature	Date
Witness' Signature	Investigator's Signature

Appendix D Structured Clinical Interview for Axis I DSM-IV Disorders

PLEASE NOTE

Copyright materials in this document have not been filmed at the request of the author. They are available for consultation, however, in the author's university library.

Appendices D - W

UMI

Appendix X Demographic Information Survey

USUHS Panic Disorder Project
Demographic Information Survey

Name: _____ Date: _____

Address: _____ Home Phone: _____
_____ Work Phone: _____
_____

Permanent Contact
Name: _____ Relation: _____

Address: _____ Home Phone: _____
_____ Work Phone: _____
_____

Referring Physician (if applicable)
Name: _____

Address: _____ Phone: _____
_____
_____

Sex: (circle) male female

Age: _____

Ethnicity:
_____ Caucasian
_____ African American
_____ Hispanic
_____ Other

Marital Status:
_____ Never Married
_____ Married
_____ Divorced
_____ Widowed

Employment Status:
_____ Employed
_____ Unemployed
_____ Student
_____ Homemaker

Have you ever received treatment for an anxiety problem? Y N
Have you ever been hospitalized for an anxiety problem? Y N