SKIN CONDUCTANCE AND CARDIOVASCULAR RESPONSES OF INFREQUENT PANICKERS AND NONPANICKERS TO THREAT OF SHOCK

by

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A thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

MASTER OF ARTS

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ABSTRACT

Psychophysiological studies have shown that patients suffering from anxiety disorders, including panic disorder, display high levels of autonomic nervous system arousal. This heightened physiological arousal in conjunction with habituation deficits may be a predisposing factor for the development of panic. The present study investigated whether differences in autonomic nervous system activity could be observed between subjects who experience infrequent panic attacks and normal controls. The former subjects show some of the clinical attributes of panic disorder patients and may also show similar elevated levels of physiological arousal. The effects of a stressor (threat of shock) on autonomic nervous system activity was investigated in a threat of shock paradigm comprising baseline, threat, and recovery periods. The subjects were 48 females selected from a group of introductory psychology students prescreened with the Panic Attack Questionnaire. The subjects comprised four equal size groups: nonpanicker-low trait anxiety, nonpanicker-high trait anxiety, panicker-low trait anxiety, and panicker-high trait anxiety. Measures of electrodermal activity, heart rate, and blood pressure were obtained every

2 minutes throughout the experiment. The infrequent panicker group had a significantly higher heart rate than nonpanickers across all phases of the experiment; however, there was no differential reactivity to the threat of shock between these groups, nor between high and low trait anxiety subjects. The infrequent panickers scored higher than nonpanickers on several subscales of the Hopkins Symptom Checklist, and on State anxiety. It was concluded that the infrequent panickers showed evidence of heightened autonomic nervous system arousal and could be viewed as distinct from nonpanickers on a dimension not accounted for by trait anxiety level.

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INTRODUCTION

As defined in the DSM-III, panic disorder is a syndrome characterized by unpredictable strikes of intense fear and apprehension known as panic attacks. Panic attacks are accompanied by numerous somatic symptoms in the cardiovascular, respiratory, and musculoskeletal systems (Shader, Goodman & Gever, 1982), including palpitations, dyspnea, chest pains, paresthesias, trembling, choking sensations, dizziness, sweating, and faintness. In addition to these autonomic symptoms, patients may also experience a variety of cognitive symptoms such as feelings of unreality, fear of dying, or fear of "going crazy" (Gorman, Fyer, Glicklich, King, & Klein,1981).

In order to be given the diagnosis of panic disorder, a patient must report having had 3 panic attacks in a 3 week period, and must experience at least 4 of the 12 symptoms associated with panic in the DSM III. While not a diagnostic criterion, another important characteristic of panic disorder is whether the patient can identify antecedent cues to the panic attack or if these attacks occur "out of the blue". Panic disorder patients often report unprecipitated or spontaneous panic attacks, whereas

patients with simple phobias may only panic when faced with the phobic stimulus which they can readily identify (Barlow, 1986).

Epidemiology

Panic disorder is not uncommon, affecting 2 to 5 % of the general population (Shader, Goodman & Gever, 1982; Sheehan, 1982; Sheehan & Sheehan, 1983). Eighty to eighty-five percent of those affected by this disorder are female, and the typical age of onset of early panic symptoms is during the late teens or early twenties (Anderson, Noyes & Crowe, 1984; Gorman, Liebowitz & Klein, in press; Sheehan & Sheehan, 1983).

There is a view that there are biological factors that underlie the development of panic disorder, and the importance of these factors is underscored by several lines of evidence. First, a number of studies have demonstrated that panic disorder shows a pattern of familial incidence which suggests a genetic contribution (Crowe, Noyes, Pauls, & Slymen, 1983; Crowe, Pauls, Slymen & Noyes, 1980; Harris, Noyes, Crowe & Chaudry, 1983). Second, panic attacks can be induced chemically in subjects who experience spontaneous panic attacks, but rarely in normal controls (Appleby, Klein, Sachar & Levitt, 1981; Kelly, Mitchell-Heggs &

Sherman, 1971; Klein, 1981). Third, psychophysiological studies have shown that psychiatric patients suffering from anxiety disorders, including panic disorder, display high levels of autonomic nervous system arousal, evidenced by increased skin conductance level, by a greater number of spontaneous skin conductance fluctuations, and higher heart rate levels (Bond, James & Lader, 1974; Chattopadhyay, Bond & Lader, 1975; Hart, 1974; Lader, 1967; Lader & Wing, 1966; Marks & Lader, 1973). Fourth, there is drug specificity in treating panic and generalized anxiety. It has been found that tricyclic antidepressants and MAO inhibitors are effective in treating panic attacks but not generalized anxiety, whereas sedatives and tranquilizers do not affect panic but are effective with anticipatory anxiety (Sheehan, Ballangar & Jacobson, 1980; Margraf, Ehlers & Roth, 1986). Fifth, there is evidence that shows increased sensitivity to caffeine in subjects with panic disorder (Charney, Heninger & Jatlow, 1985; Uhde, Boulenger, Post, Siever, Vittone, Jimerson, & Roy-Byrne, 1984).

Natural History

It has been suggested that some of the biological characteristics associated with panic disorder may precede the actual onset of panic attacks. Roberts (1984) has identified early prodromal signs of panic which include easy

blushing, cold or wet hands, palpitations, and faintness. Research has shown that individuals who later develop panic disorder have displayed these symptoms in their teens (Cloninger, Martin, Clayton & Guse, 1981; Klein, 1981). However, the first actual panic may not occur for several years after the development of these prodromal signs. It is not uncommon for this first attack to occur quite spontaneously while the individual is engaging in some innocuous activity. (Roberts, 1984; Sheehan & Sheehan, 1983). Suddenly the individual will experience overwhelming somatic symptoms which may induce the belief that he or she is having an acute medical crisis such as a heart attack. As a result, the person will often seek help at a hospital emergency room or see a physician (Gorman, Liebowitz & Klein, in press). Sheehan and Sheehan (1983) report that 70 percent of the panic disorder patients they encounter had consulted their physicans about their symptoms more than 10 times.

General Considerations

Although the first panic attack may appear spontaneously, several events nevertheless seem to be related to the initial occurrence. The attack may occur during a severe illness, bodily injury, thyroid dysfunction, drug withdrawal, or following childbirth (Gorman et al., in

press). Specific psychosocial factors, such as loss of a loved one and interpersonal conflict, have also been linked to the first panic episode (Gorman et al., in press; Last, Barlow & O'Brien, 1984; Raskin, Peeke, Dickman & Pinsker, 1982). After the first panic attack, the problem may progress to numerous full blown panic attacks. If these attacks occur in the same situation a number of times, the situation itself may elicit anticipatory anxiety. As a result, the individual may begin to avoid situations which are paired with panic attacks (Klein, 1981; Zitrin, Woerner & Klein, 1981). As these attacks occur in more and more situations, avoidance behavior may generalize to a variety of other situations (Sheehan & Sheehan, 1983). In isolated cases, the individual may develop numerous phobias and eventually become agoraphobic (Sheehan, Ballenger & Jacobson, 1981; Sheehan & Sheehan, 1983; Thyer & Himle, 1985).

The likelihood and rate of progression from the first panic attack to the polyphobic stage depends on the frequency and intensity of the panic attacks. Individuals who experience intense, frequent panic attacks tend to progress to the polyphobic stage very rapidly, while those individuals who experience mild, infrequent panic attacks may never reach that stage (Sheehan & Sheehan, 1983).

There appears to be a substantial number of people within the general population who do experience infrequent panic attacks. These "infrequent panickers" have recently been investigated by Norton, Harrison, Hauch and Rhodes (1985a). In a survey of university undergraduates they have found that 34 % of their sample reported having had one or more panic attacks in the last year. Over two percent reported having had three or more panic attacks in the past three weeks; a frequency consistent with the diagnosis of panic disorder. The authors also found that, like panic disorder patients, these infrequent panickers showed higher levels of depression, anxiety, and psychopathology than nonpanickers (Norton, Dorward & Cox, 1986). In addition, Norton, Hauch and Harrison (1985) found that infrequent panickers report a higher incidence of psychiatric disorders among their parents than do nonpanickers. This is also consistent with the family history reports of actual panic disorder patients (Norton, et. al., 1985a).

Norton et al., (1985a) have observed several similarities in the panic symptom profile of infrequent panickers and patients with panic disorder and agoraphobia. Both groups report experiencing palpitations, shaking, and sweating during panic attacks, but the clinical patients experience them more severely. Infrequent panickers also report that the onset of their panic attacks is rapid (0 to

10 minutes), and for some, may also occur unpredictably. The infrequent panickers who experience unpredictable panic attacks also appear to have more severe panic episodes than those who only experience predictable panic attacks (Norton et al., 1986). These results suggest that the frequency and severity of panic attacks may be on a continuum, where infrequent panickers display characteristics similar to panic disorder patients, but with less frequency and severity (Norton et al., 1985a).

These findings indicate that the experience of panic attacks is a relatively common phenomenon (34%) in the general population. The identification of individuals who are infrequent panickers offers a great research opportunity to investigate the biological basis of panic among nonclinical subjects, particularly since it has been suggested that there is a progression from prodromal subpanic symptoms to full scale panic disorder (Roberts, 1984; Sheehan & Sheehan, 1983).

PSYCHOPHYSIOLOGY OF ANXIETY

It is well founded that anxious patients show a psychophysiological response pattern characterized by heightened autonomic arousal. Even under resting conditions, anxiety patients display higher levels of palmar skin conductance (SCL) and a greater number of spontaneous skin conductance fluctuations (Bond, James & Lader, 1974; Chattopadhyay, Bond & Lader, 1975; Hart, 1974; Lader, 1967; Marks & Lader, 1973).

In studies of passive stimulation where a series of tones is presented following an initial rest period, anxious patients tend to adapt less during baseline, habituate to the tones slower, and have more spontaneous skin conductance fluctuations than normal subjects (Bond, et. al. 1974;Chattopadhyay et al., 1975; Lader & Wing, 1966).

Lader and Mathews (1968) formulated a model of chronic anxiety in which anxiety prone individuals are said to have an innately slow rate of habituation. When these individuals are exposed to repetitive activating stimuli, their habituation rate is slowed even further, resulting in autonomic nervous system overarousal. Thus, in this model, habituation is seen as a control for arousal, and panic

attacks are triggered because of a poor ability to habituate (Lader & Mathews, 1968).

Claridge (1967) proposed a related theory of chronic anxiety in which habituation deficits are seen as secondary to increased physiological activity. Within this view, anxiety prone individuals are seen as having a nervous system which is set at a heightened level of activity, which subsequently attenuates habituation. A study by Lader and Wing (1966) supports this theory. In their investigation of pharmacologically induced changes in arousal, the authors found that high arousal slowed habituation to repetitive auditory stimuli. In addition, Bohlin (1976) manipulated level of arousal using cognitive tasks rather than chemical agents and similarily found that increased arousal,beyond a particular level, slowed habituation rates. Bohlin suggested that there is a threshold in level of arousal, below which habituation is unaffected.

These two theories can be viewed as complementary in that habituation is an arousal dampening mechanism which becomes ineffective when arousal levels increase beyond a certain level.

In an attempt to further clarify this issue, Raskin (1975) has criticized the past habituation studies with anxiety patients on the basis that they have failed to take

into account the increased threat appraisal that anxiety patients experience in test situations. To investigate this, Raskin (1975) carried out a habituation study which investigated the presence of threat appraisal in subjects. Following a typical auditory habituation study, subjects were interviewed to assess their interpretation of the presented tones. The author found that the patient group attributed meaning (signal value) to the tones significantly more than the control group. Patients frequently reported feeling that the tones would be paired with painful shock, or would harm them in some way. None of the subjects in either group who experienced these fears habituated to the tones. This is not surprising since habituation is a simple form of learning where an individual learns that repetitive stimuli, not followed by any harmful event, are innocuous (Schwartz, 1984). This learning process may be attenuated by the increased threat appraisal shown by anxiety patients.

To investigate the neurophysiological correlates of anxiety in conjuction with behavioral characteristics, anxiety patients have also been compared to normal subjects under more demanding conditions presumed to be stressful. In these studies subjects were required to perform complex cognitive tasks. The purpose of these stress arousing manipulations was to simulate the high physiological arousal of anxiety patients in normal controls. In these studies,

normal subjects actually show a greater increase in electrodermal activity than anxiety patients. In reaction time studies, patients consistently respond to the task with smaller changes in electrodermal activity than normals (Bond, James, and Lader, 1974; Chattopadhyay, Bond, and Lader,1975). Bond et.al. (1975) also gave subjects a variety of cognitive tasks that differed in level of difficulty, while measuring their electrodermal activity. The authors again found that normals responded with a greater increase in activation during the tasks than patients, and the impairment of performance in the patient group increased with the increasing difficulty of the task.

It is presumed that these differences in electrodermal activation occur because the high resting levels in the patient group leads to a ceiling effect for these subjects (Lader, 1975). That is, since patients start out with such high levels of physiological activity, they quickly reach their maximum responsiveness, and thus show smaller increases in arousal compared to normals.

Resting heart rate and blood pressure have also been found to differentiate anxiety patients from normal controls (Kelly, Mitchell-Heggs & Sherman, 1971; Michelson & Mavissakalian, 1985). Patients consistently show higher resting heart rates and systolic blood pressure. This suggests that anxious patients may also exhibit higher

levels of arousal within the cardiovascular system. Hugdahl, Fredrikson and Ohman (1977) suggest that heightened physiological arousal levels, in conjunction with habituation deficits, are a prerequisite for the development of panic. When encountering a stressful situation, arousal levels may increase to the extent that a panic attack is triggered.

ELECTRODERMAL ACTIVITY AND THE THREAT OF SHOCK PARADIGM

The previously discussed "stress" induction studies with anxious patients using complex cognitive tasks, which found normal subjects to show greater increases in arousal than anxiety patients, have confounded increased electrodermal activity due to cognitive/perceptual activity with increased electrodermal activity in response to "stress". To avoid this interaction, it seems practical to study physiological reactivity within a paradigm which does not blend cognitive tasks with psychological stress. The threat of shock paradigm fits this requirement.

The threat of shock paradigm has been a widely used anxiety inducing procedure which comprises baseline, threat, and recovery periods. Generally, subjects are told that they will receive an electric shock at some point during a specified time period. It is generally observed that heart rate, spontaneous skin conductance fluctuations, and occasionally skin conductance level when there is secondary cognitive mediation of fear, increase in anticipation of the electric shock.

The probability of receiving a shock and the point at which the shock is expected in the threat phase has varied across studies. Monat, Averill, and Lazarus (1972) have found that under temporal uncertainty, when the subject does not know exactly when to expect the shock, the maximum physiological response to the threat of shock occurred early on in the phase and dwindled as time passed. Under conditions of event uncertainty where the subject is told that the probability of actually recieving a shock may be 100%, 50%, or 5%, the maximum physiological response did not occur until later on in the threat period and levels of uncertainty did not differ in their fear arousing effect.

To test internal vs external determinants of fear in response to a threat of shock, Mansueto and Desiderato (1971) told subjects to expect a shock at the end of a 12 minute period and provided only one group with a clock (external cue). The authors found that while both groups started out at about the same point at the start of the threat of shock phase, the level of physiological arousal increased steadily and peaked at the 12 minute point for the group provided with an external time cue (clock), but the level of arousal actually dropped steadily to baseline level by the end of the 12 minutes in the group not provided with an external time cue. Thus, external time cues are necessary in order to increase arousal within this design.

Bancroft and Elliot (1974) tested the effect of prior experience with the shock on the fear arousing ability of the threat of shock paradigm. The authors found that the group with prior experience with the shock showed less physiological reactivity to the threat of shock than subjects who had no idea what type of shock to expect.

It appears that the most effective threat of shock procedure is one in which subjects are told that they will receive a shock at the end of a specified period, have no experience with shock, and are provided with a clock.

The threat of shock paradigm may be a particularly effective tool to maximize physiological arousal for the study of reactivity in anxiety patients because, as Raskin (1975) has shown, anxiety patients tend to be fearful in experimental situations and assign threat value to auditory tones in a simple habituation study. As well, using a modified version of the Stroop colour naming task, Mathews and MacLeod (1985) have found that anxiety patients spend more time processing words related to threat or danger. Together, these studies suggest that anxiety patients can be characterized by hypervigilance in experimental settings and are particularily sensitive to aspects of their environment which may predict danger.

Anxiety patients actually have been compared with normals using the threat of shock paradigm. Rabavilas, Boulougouris, Stefanis, and Vaidakas (1977) studied the anticipatory fear response to the threat of shock in obsessive-compulsive patients (anxiety disorder) and control subjects. The shock anticipation elicited greater changes from baseline in patients than in normals for both heart rate and skin conductance responses.

Similar studies have been carried out using normal subjects who differed in level of trait anxiety (Katkin, 1965; Katkin, 1966; Chattopadhyay et al., 1980). Generally, it is found that during the threat phase all subjects show a marked increase in electrodermal activity, but there is no difference in the magnitude of increase between high and low anxiety subjects. Katkin (1965) investigated the effect of threat of shock on subjects who differed in level of anxiety on the Taylor Manifest Anxiety scale and found no difference in phasic electrodermal activity between high anxious subjects and low anxious subjects. In a subsequent study employing another measure of anxiety (Affect Adjective Check List) Katkin (1966) again failed to find differences in normal subjects varying in trait anxiety. Low anxious and high anxious subjects showed similar levels of spontaneous fluctuations during the threat phase, although the low anxious normals did recover faster following the threat of

shock phase. Hodges and Spielberger (1966) evaluated the effect of threat of shock on heart rate for subjects who differed in fear of shock and trait anxiety. The authors found no differential increase in heart rate between high trait anxious and low trait anxious subjects, but did find that high fear subjects showed greater increases in heart rate than low fear subjects, and that fear of shock was unrelated to trait anxiety.

It seems apparent that arousal differences during threat of shock, as shown by heart rate and spontaneous skin conductance fluctuations, are more likely to be seen between anxiety patients and normal controls than between subjects who merely differ in self report anxiety. The discrepancy in these two bodies of research may be due to a biological difference between these two groups. Anxiety patients (including panic disorder) have been shown to display a psychophysiological response pattern that is characterized by heightened autonomic arousal (Bond, James, & Lader, 1974). In addition, panic disorder shows a pattern of familial incidence, and a sensitivity to chemicals (lactate & caffeine) which is suggestive of an underlying biological mechanism (Appleby, Klein, Sachar, & Levitt, 1981; Uhde, Boulenger, Post, Siever, Vittone, Jimerson, & Roy-Byrne, 1984).

The purpose of this experiment was to determine whether differences in autonomic reactivity could be observed between infrequent panickers and normal subjects who have never experienced a panic attack. It has been suggested that the development of panic disorder involves a biological predisposition towards states of autonomic hyperarousal which follows a progression from early prodromal symptoms (faintness, heart palpitations, sweaty hands, and easy blushing) to full blown episodes of panic (Roberts, 1984). Since infrequent panickers have been shown to possess some of the clinical attributes of anxiety patients, they may show the heightened physiological activity that underlies the experience of panic, yet precedes the diagnosis of a psychiatric syndrome (Sandler & Wilson, 1986). This is not an unreasonable prediction since the panic attack symptom checklist that is used to identify infrequent panickers, largely reflects symptoms of high autonomic arousal. According to Hugdahl et al. (1977) heightened physiological arousal along with habituation deficits can be seen as a prerequisite for the development of panic.

In order to address the discrepancy in the stress research with anxiety patients and trait anxious university students, trait anxiety levels of infrequent panickers and nonpanickers were also examined in the present research. Any physiological differences found between infrequent

panickers and nonpanickers, while controlling for anxiety level, would suggest that the experience of panic is not just another indicator of self reported anxiety level, and may reflect an underlying biological mechanism. To this end, high and low trait anxious levels were included within the infrequent panicker and nonpanicker groups in this experiment.

Because autonomic hyper-reactivity or overarousal is a characteristic of anxiety patients, skin conductance level and the frequency of spontaneous skin conductance fluctuations were examined. Both of these measures are due to the stimulation of the eccrine sweat glands and are indicative of the degree of sympathetic arousal (Raskin, 1973). Spontaneous fluctuations occur in the absense of specific stimulation and have been shown to be an effective index of emotional response to stimuli as well as a valid measure of chemically induced sympathetic activation (Katkin, 1965). Heart rate, mean arterial pressure, systolic blood pressure, and diastolic blood pressure were also used to reflect the level of arousal. Since anxiety states have been characterised by selective processing of danger related cues, a three phase threat of shock procedure which has been shown to greatly increase electrodermal activity and heart rate was employed (Petry & Desiderate, 1978).

The following hypotheses were proferred:

1. If the threat of shock were effective in inducing anxiety, within group comparisons would show greater physiological activity in the threat phase than in the rest phases.

2. If infrequent panickers respond physiologically like anxious patients, they would show higher resting levels of all physiological measures than nonpanickers. However, if infrequent panickers respond like anxious university students they would show no physiological differences.

3. Infrequent panickers would be more physiologically reactive to the threat phase than the nonpanickers. That is, they would show greater increases in electrodermal activity and cardiovascular measures than nonpanickers in this phase.

4. There would be no difference in physiological reactivity to the threat of shock between high trait anxiety and low trait anxiety subjects.

5. If infrequent panickers are like anxious patients and are more sensitive to threat than nonpanickers, they would report higher state anxiety than the nonpanickers at the start of the shock anticipation period.

Experimental Design and Analysis

The experimental design was a 2 (high anxiety/low anxiety) by 2 (panicker/nonpanicker) by 3 (phase) mixed factorial design. The between subjects factors were trait anxiety (high/low) and subject classification (panicker/nonpanicker) and the within subjects factor was phase (Baseline, Threat, Recovery).

For the analysis of the questionnaire data, MANOVAs were performed to obtain omnibus F ratios and univariate ANOVAs were applied to the individual elements of each questionnaire.

Prior to the analysis of the physiological data, the statistical assumptions underlying the use of ANOVA were tested. The physiological data satisfied the assumptions of normality, homogeneity of variance, and skewness.

Keselman and Keselman (1988) suggest that robust tests of repeated measures dependent variables can be obtained by performing univariate ANOVAs and adopting a corrected df where physiological data do not meet sphericity requirements. Sphericity tests were applied to the physiological data in this study, and it was found that ANOVAs with uncorrected df were appropriate. Therefore, univariate ANOVAs were used to test the hypotheses, and post hoc pairwise comparisons were used to investigate any significant interactions.

METHOD

Subjects

The subjects in this study were 48 female introductory psychology students at the University of Manitoba chosen from 278 prescreened with the Panic Attack Questionnaire (PAQ). Since the incidence of panic attacks is much greater in females, only females were chosen for this study. The subjects were run individually for 1 hour and each subject received 1 experimental credit. To be included in this study, nonpanickers must have reported never having experienced a panic attack and panickers must have reported having at least 5 panic attacks in the past year. The panickers in this study reported a frequency of panic attacks in the past year that ranged from 5 to 11, with a mean of 7.25 and SD of 2.72.

The subjects were divided into 2 equal sized groups (panicker/nonpanicker) with 2 levels of trait anxiety (high/low) within each group. There were also equal numbers of subjects within each level of each group. The division into the 2 anxiety levels was based on the median trait anxiety score (43) for the entire screening sample.

Subjects were assigned to the low anxiety level if their anxiety score was below the median, or to the high anxiety level if their anxiety score was above the median. To establish that the actual trait anxiety scores in each level of anxiety did not differ between groups (panicker/nonpanicker), a 2 (panicker/nonpanicker) by 2 (high anxiety/low anxiety) ANOVA was performed. The two high anxiety groups did not differ in their mean trait anxiety scores, nor did the two low anxiety groups. As would be expected though, the low anxiety groups differed significantly from the high anxiety groups, $F(1,44)=99.7$, $p<.01$.

A total of 63 subjects were actually run in this experiment. Fifteen subjects could not be used, leaving 48 subjects for the final analysis. Eleven subjects were eliminated (all nonpanickers) because they did not believe they would receive a shock, 3 subjects were eliminated due to equipment failure, and 1 subject (nonpanicker) asked to be removed from the apparatus during phase II.

The mean ages for the panicker and nonpanicker groups were 20.4 (SD=2.8) and 19.9 (SD=2.5) respectively, and there was no significant age difference between the groups, $F(1,44)=1.09$, $p<.50$.

Apparatus and Materials

Physiological Recording

The subject was seated comfortably in a reclining armchair, in a sound attenuated, electrically shielded room with an ambient light intensity of 40 Watts. The subject room was equipped with an intercom which connects with the adjacent room housing the experimenter and the recording apparatus.

A MFE Model M-22CAHA Strip chart recorder was employed to record skin conductance by interfacing it with a DC skin conductance unit based on a design by Venables and Christie (1975). Silver-silver chloride electrodes were attached to the subject using a bipolar placement on the palmar surface of the subjects nondominant hand. Adhesive collars were used to hold the electrodes in place and Johnson and Johnson K-Y Jelly was used as an electrolyte.

Measures of mean arterial blood pressure (MAP), heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded every 2 minutes throughout the experiment by a Critikon Dinamap 845 XT Vital Signs Monitor. The unit was attached to the subject's dominant arm via an arm cuff.

Questionnaires

The primary screening instrument was a questionnaire derived from Norton et. al.'s (1985) Panic Attack Questionnaire (PAQ). This questionnaire is a self report inventory investigating the occurrence, severity, frequency, and symptoms associated with panic attacks (Appendix A). A description of an actual panic attack has been added to the PAQ in order to minimize the chance that subjects may confuse a panic attack with being tense or nervous. Speilberger's Trait anxiety scale was given in order to categorize high and low trait anxiety subjects. The Hopkins Symptom Checklist (HSCL-90) (Derogatis, Lipman, Rickels, Uhlenhuth, & Coul, 1974) was given to obtain information on the nine symptom dimensions commonly observed in psychiatric patients. The laboratory symptom checklist, a replica of the panic attack symptom list in the PAQ, was given to investigate whether any of the subjects experienced the symptoms of panic while awaiting the shock. A post experimental questionnaire (PEQ) aimed at investigating the subject's beliefs about the experiment was also administered.

Procedure

Initial phase: Upon arriving at the laboratory, the subjects were given preliminary instructions (Appendix B) informing them that we were investigating the effect of shock on anxiety level. The subjects were also told that they would receive a strong brief shock, but it would not be harmful. Following this, if the subject wished to participate in the study, she was asked to sign the consent form (Appendix C).

The experimenter then seated the subject in the research room and attached the skin conductance electrodes, blood pressure cuff, and the "mock" shock electrodes to the subject, while describing the function of each piece of equipment.

Phase I (adaptation): This phase lasted 15 minutes during which time resting measures were obtained for only the last 10 minutes. Average heart rate, blood pressure, skin conductance level and spontaneous fluctuations were obtained for successive 2 minute intervals.

Following the adaptation phase, the subjects received instructions reminding them that they would be given a strong brief shock exactly 10 minutes following the start of the next phase, and to watch the wall clock so they would know when to expect the shock. Just prior to the beginning

of the threat phase, the subjects were asked to complete Speilberger's State anxiety scale which had been placed on a table beside the subject (Appendix D).

Phase II: During this 10 minute phase, the subject's physiological responses were again measured every 2 minutes. At the end of this phase, the subjects were told that,in fact, they would not be receiving a shock, and that we would like them to relax for an additional 10 minutes while we continued to record their physiological responses.

Phase III: This phase began after the time of the scheduled shock and lasted 10 minutes, during which time physiological measures were obtained every 2 minutes as in previous phases. Following this final phase, subjects were removed from the apparatus, given the PEQ (Appendix E), the Laboratory Symptom Checklist (Appendix F), and debriefed.

Response Definitions

Skin conductance level (SCL) was measured in micromhos,and the number of spontaneous skin conductance fluctuations (SCF), larger than 0.05 micromhos occuring within each sampling period was calculated. Heart rate was measured in beats per minute, and blood pressure and mean arterial pressure were measured in mmHg.

RESULTS

Physiological Data

To get an overall view of the data and to investigate the effectiveness of the threat of shock, each physiological measure (MAP, HR, SBP, DBP, SCL, SCF) was analyzed using a 2(group) X 2(anxiety) X 3(phase) repeated measures analysis of variance. A significant main effect of phase was found for all measures. As indicated in Table 1, the means of the threat phase (phase II) were higher than the means of either rest phase, indicating that the threat of shock was effective in increasing arousal. Summary ANOVA statistics are found in Appendix G.

A significant main effect of group was found only with HR (Table 2). The panickers had significantly higher heart rates than the nonpanickers throughout the entire experiment ($F(1,44) = 10.49$, $p < .01$). A graph of these results can be found in Figure 1. It is interesting to note that during the threat of shock phase, the nonpanickers did not reach even the resting levels of the panicker group. Summary ANOVA statistics are found in Appendix H.

A significant main effect of anxiety was found only in SBP, with low anxiety subjects showing higher SBP than high anxiety subjects, $F(1,44) = 5.60$, $p < .05$. A graph of these results can be found in Figure 2.

The only other significant effect found was a phase X group X anxiety interaction in DBP, $F(1,44) = 5.89$, $p < .01$. The disordinal interactions in Figure 3 reveal that the panicker-low anxiety and nonpanicker-high anxiety groups do not increase as much as the panicker-high anxiety and nonpanicker-low anxiety groups when going from phase I to phase II. To futher pursue this interaction statistically, pairwise comparisons were performed on all of the means of each phase (I,II,III). It was found that no means differed significantly from the others, although the difference between the panicker-high anxiety and nonpanicker-high anxiety groups in phase III was approaching significance.

Table 1

Summary Statistics of the Physiological Variables Across Phases

Measure	Phase Means I	II	III	F	Tail Prob.
HR	71.92	74.86	69.82	35.85	.0000 *
MAP	83.18	84.29	81.65	13.28	.0000 *
SBP	117.29	119.29	115.20	23.59	.0000 *
DBP	67.34	68.74	67.01	8.21	.0005 *
SCL	4.79	6.81	5.14	37.19	.0000 *
SCF	5.96	9.39	3.65	59.94	.0000 *

Note: Degrees of freedom for each test were 2 and 88.
* p< 01.

Table 2

Summary Table for the Main Effects for Each Physiological Variable

Measure	Source	F	Tail Prob.
HR	Group	10.59	.0022 **
	Anxiety	2.10	.1542
MAP	Group	.98	.3276
	Anxiety	2.70	.1075
SBP	Group	.29	.5936
	Anxiety	5.60	.0224 *
DBP	Group	1.10	.3001
	Anxiety	.16	.6878
SCL	Group	.18	.6719
	Anxiety	1.54	.2208
SCF	Group	.50	.4847
	Anxiety	2.02	.1620

Note: degrees of freedom for each test were 1 and 44.
* $p < .05$; ** $p < .01$.

Figure 1: Plot of the Group Differences in Heart Rate (HR) for Each Phase.

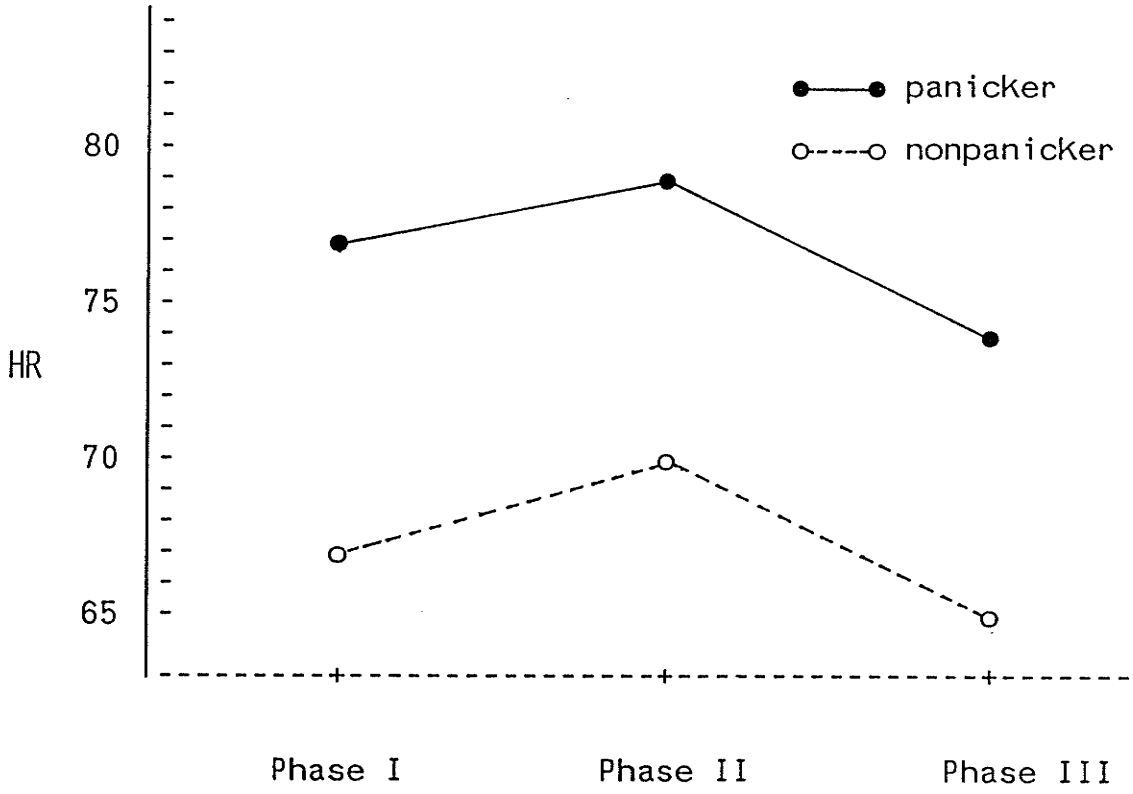


Figure 2: Plot of the Main Effect of Anxiety for Systolic Blood Pressure (SBP).

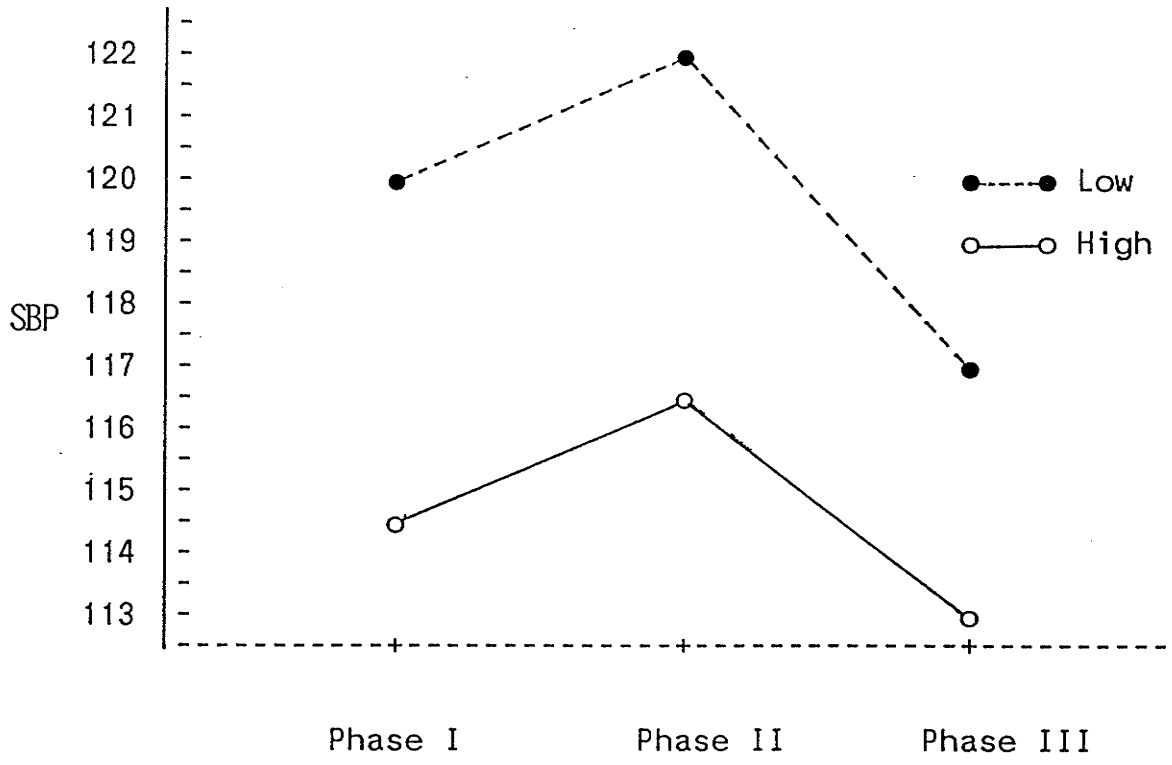
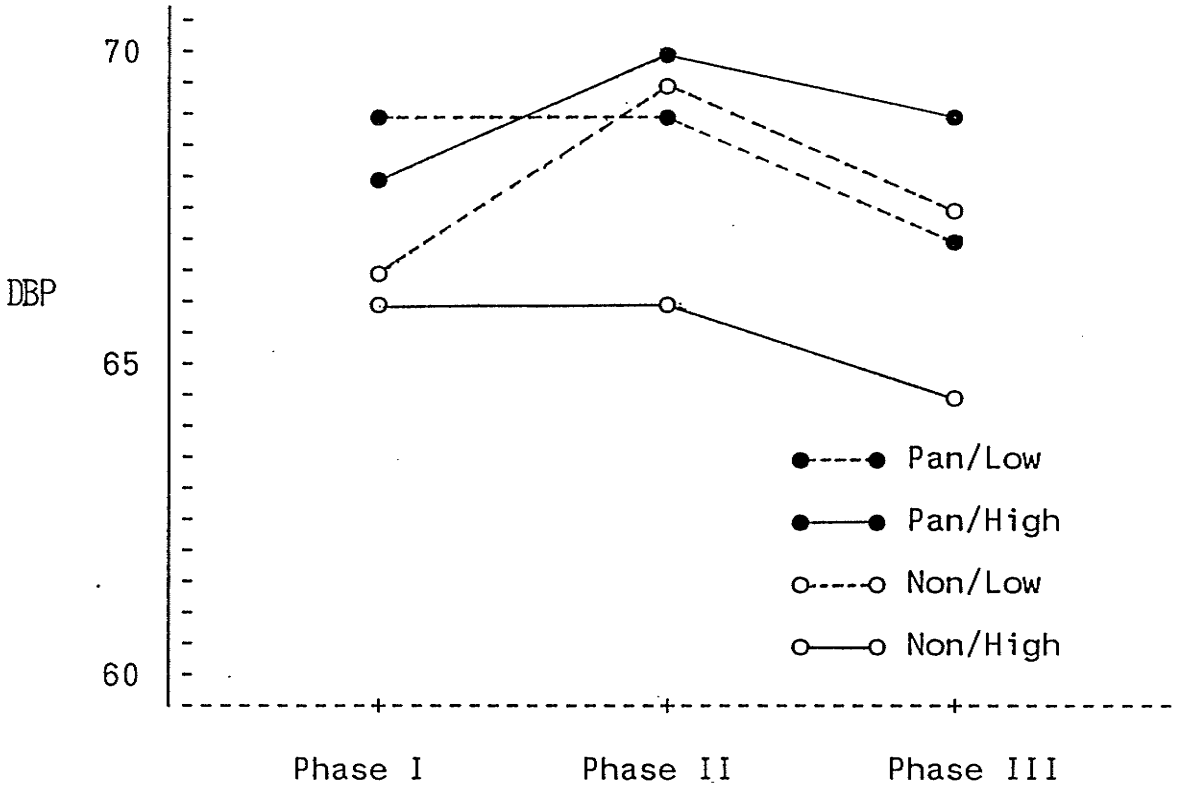


Figure 3: Plot of the Group x Anxiety x Phase Interaction For Diastolic Blood Pressure (DBP).



Since there was an overall significant main effect of group, showing panickers to have higher HR levels than nonpanickers across all phases of the experiment, these results were probed further for exploratory purposes. Resting HR levels for each rest phase (I,III) were examined separately with a 2(groups) X 2(anxiety) repeated measures analysis of variance.

Again, significant group differences were found in phase I, $F(1,44)=11.63$, $p<.01$, and in phase III, $F(1,44)=7.05$, $p<.01$. As shown in Table 3, panickers have a higher resting HR than nonpanickers in both rest phases.

An interesting anxiety X group interaction was also found, $F(1,44) = 7.21$, $p < .05$. Scheffe post hoc comparisons revealed that panickers with low trait anxiety had a significantly higher resting HR than nonpanickers with low trait anxiety, $F(1,44) = 4.57$, $p < .05$. In addition, it was found that only within the nonpanicker group, did low trait anxiety subjects have a significantly higher resting HR than high trait anxiety subjects, $F(1,44) = 7.33$, $p < .01$. Cell means are shown in Table 4. These tests were done for exploratory purposes and are not meant to be used to form definitive conclusions.

Table 3

Mean Resting HR for Each Rest Phase by Group

	Phase I		Phase III	
	Panicker	Nonpanicker	Panicker	Nonpanicker
Mean	76.9	67.0	74.3	65.4
SD	2.29	3.12	.97	3.52

Table 4

Mean Resting HR in Phase III by Group and Anxiety

Group		Anxiety Level	
		Low	High
Panicker	Mean	74.3	74.3
	SD	.57	1.34
Nonpanicker	Mean	68.5	62.3
	SD	1.99	.39

Although there were no significant reactivity differences (groupXphase & anxietyXphase) indicated in the overall ANOVA, measures that showed group(HR), anxiety(SBP), or groupXanxiety (DBP) effects were further analyzed for exploratory purposes. To investigate differential reactivity between the groups to the threat of shock, a test of treatment by contrast, which compared the threat phase (II) with each rest phase (I & III), was performed on each measure using a 2(group) X 2(anxiety) X 3(phase) repeated measures analysis of variance with contrast. Although all measures showed a main effect of phase, no significant group by phase interactions were found. This lack of interaction indicates that no differences in reactivity to the threat of shock between panickers and nonpanickers had occurred. Table 5 contains summary statistics for each comparison.

In investigating differential reactivity to the threat of shock between high anxious and low anxious subjects, a main effect of anxiety was once again found only in SBP, $F(2,88) = 5.60$, $p < .05$. There were however no significant phase by anxiety interactions indicating that there was no differential reactivity to the threat of shock between high anxiety and low anxiety subjects for SBP nor for HR and DBP.

Table 5

Summary ANOVA Table for Phase by Group Interactions Testing Reactivity

Measure	F	Tail Prob.
HR	.49	.6165
SBP	.02	.9765
DBP	.04	.9633

Note: df for each test were 2 and 88.

Psychological Measures

To investigate group differences in state anxiety at the start of the threat of shock phase (II), a 2(groups) X 2(anxiety) ANOVA was performed. There was a significant main effect of group, $F(1,44)=5.17$, $p<.05$. Examination of the group means revealed that the state anxiety scores of the panicker group ($M=45.1$, $SD=12.3$) were higher than the nonpanicker group ($M=37.6$, $SD=10.8$). There was no effect of anxiety or groupXanxiety.

To compare panickers and nonpanickers on their scores of the HSCL-90, a 2(groups) X 2(anxiety) X 9(subscales) MANOVA was performed to obtain omnibus F statistics. Since the MANOVA showed an overall main effect of group, $F(11,34)=2.28$, $p<.05$, and anxiety, $F(11,34)=6.78$, $p<.01$, univariate tests were applied to each subscale. Overall, panickers scored higher than nonpanickers on all of the subscales, but the differences were statistically significant for only 6 of the 9 subscales, with the Hostility subscale approaching significance, $F(1,46)=3.28$, $p<.06$. Table 6 contains univariate statistics and group means for each subscale.

Examination of the main effect of anxiety revealed that high trait anxious subjects scored significantly higher than

low trait anxious subjects on all 9 subscales of the HSCL-90. Table 7 contains univariate statistics and means of each subscale for the main effect of anxiety.

Table 6

Table of Means and ANOVAs for the Subscales of the Hopkins Symptom Checklist by Group

Subscale Name	Subscale Mean		F	Tail Prob.
	Panicker	Nonpanicker		
Somatization	1.23	.75	5.46	.0240**
Obsessive/Compulsive	1.57	1.06	6.98	.0114**
Interpersonal Sensitivity	1.40	1.02	5.53	.0233**
Depression	1.50	.96	14.08	.0005**
Anxiety	1.44	.84	12.44	.0010**
Hostility	1.15	.75	3.83	.0586
Phobic Anxiety	.57	.34	1.58	.2150
Paranoid Ideation	1.26	.88	4.31	.0439*
Psychoticism	.74	.56	2.03	.1615

Note: degrees of freedom for all tests were 1 and 44.
* $p < .05$, ** $p < .01$.

Table 7

Table of Means and ANOVAs for the Subscales of the Hopkins Symptom Checklist by Anxiety

Subscale Name	Subscale Mean		F	Tail Prob.
	High	Low		
Somatization	1.27	.71	7.73	.0079**
Obsessive/Compulsive	1.80	.83	25.68	.0001**
Interpersonal Sensitivity	1.76	.66	48.11	.0001**
Depression	1.78	.68	59.77	.0001**
Anxiety	1.50	.78	18.21	.0001**
Hostility	1.27	.63	9.63	.0033**
Phobic Anxiety	.75	.16	10.20	.0026**
Paranoid Ideation	1.54	.60	27.31	.0001**
Psychoticism	.91	.39	18.53	.0001**

Note: degrees of freedom for all tests were 1 and 44.
** p <.01.

A 2(group) X 2(anxiety) X 14(symptoms) MANOVA was also performed on the symptom checklist that each subject completed at the end of the experiment. Although the omnibus F statistics did not approach significance for group, $F(14,31)=1.46$, $p<.19$, or for anxiety, $F(14,31)=1.44$, $p<.19$, univariate ANOVAs were applied to individual symptoms for exploratory purposes. As indicated in Table 8, panickers scored higher than nonpanickers on all but one symptom, but these differences were statistically significant for only 5 symptoms including dyspnea, $F(1,44)=6.30$, $p<.05$, palpitations, $F(1,44)=5.06$, $p<.05$, chestpains, $F(1,44)=13.28$, $p<.01$, dizziness, $F(1,44)=6.49$, $p<.05$, and trembling, $F(1,44)=4.05$, $p<.05$.

Table 9 shows comparisons between high and low trait anxious subjects for each symptom. The high trait anxious subjects scored significantly higher than the low trait anxious subjects on only 3 symptoms including dyspnea, $F(1,44)=4.63$, $p<.05$, faintness, $F(1,44)=4.11$, $p<.05$, and fear of going crazy, $F(1,44)=4.63$, $p<.05$.

In order to investigate whether there was any stylistic difference between groups in thoughts while waiting for the electric shock, data from questions 4 and 5 of the PEQ were examined. The responses to these questions could be divided into avoidant and nonavoidant thought content. For the purposes of this experiment, avoidant content was considered

to be statements suggesting an attempt to avoid thinking about the impending shock. Such statements included: thoughts of pleasant experiences, upcoming events, and activities unrelated to the experiment. Nonavoidant content was indicated by statements in which the subjects reported actually thinking about the shock throughout phase II. Such statements included: speculation of the intensity, duration, and probability of the upcoming shock.

Two separate 2-way (groupXthought content & anxietyXthought content) chi square analyses were performed to investigate any differences in the distribution of avoidant thoughts between panicker categories and trait anxiety levels. It was found that panickers and nonpanickers did not differ significantly in avoidant vs nonavoidant thoughts, nor did the 2 groups varying in anxiety. Table 10 contains the cell frequencies for each group.

Table 8

Table of Mean Symptom Scores and ANOVAs for the Laboratory Symptom List by Group

Symptom	Panicker	Nonpanicker	F	Tail Prob.	
Dyspnea	.88	.29	6.30	.0158	*
Palpitations	1.54	.96	5.06	.0295	*
Chestpains	.75	.08	13.28	.0007	**
Choking	.17	.04	1.36	.2505	
Dizziness	1.13	.46	6.49	.0144	*
Unreality	.79	.42	2.64	.1111	
Paresthesis	1.38	1.08	.86	.3589	
Flashes	.42	.46	.02	.8815	
Sweating	.38	.25	.46	.5010	
Faintness	.42	.21	1.27	.2664	
Trembling	.92	.33	4.05	.0502	*
Fear of Dying	.33	.21	.58	.4508	
Fear of Going Crazy	.13	.04	1.16	.2878	
Fear of Being Uncontrolled	.46	.38	.18	.6744	

Note: degrees of freedom for all tests were 1 and 44.
* p <.05, ** p<.01.

Table 9

Table of Mean Symptom Scores and ANOVAs
for the Laboratory Symptom List by Anxiety

Symptom	High Anxiety	Low Anxiety	F	Tail Prob.
Dyspnea	.83	.33	4.63	.0369 *
Palpitations	1.29	1.21	.10	.7494
Chestpains	.50	.33	.83	.3672
Choking	.17	.03	1.38	.2501
Dizziness	1.00	.58	2.53	.1185
Unreality	.70	.49	2.61	.1117
Paresthesis	1.33	1.13	.44	.5113
Flashes	.58	.29	1.10	.2995
Sweating	.33	.21	.43	.5023
Faintness	.50	.13	4.11	.0488 *
Trembling	.58	.67	.18	.7750
Fear of Dying	.38	.17	1.16	.2114
Fear of Going Crazy	.17	.00	4.63	.0369 *
Fear of Being Uncontrolled	.54	.29	1.16	.2112

Note: degrees of freedom for all tests were 1 and 44.
* p <.05, ** p<.01.

Table 10

Cell Frequencies of Avoidant vs Nonavoidant Thought Content by Group and Anxiety

	Groups			
	Panicker	Nonpanicker	High Anx.	Low Anx.
Avoidant	7	10	11	8
Nonavoidant	6	12	8	8
Ambiguous	10	1	5	6
Missing Data	1	1	0	2

DISCUSSION

The results obtained in the present study confirmed the first hypothesis, that if the threat of shock was effective in increasing anxiety, within group comparisons would show greater physiological activity in the threat phase (II) than in either rest phase (I & III). A significant phase effect was found with every physiological measure, and the very large F values showed the threat of shock paradigm to have a very robust effect on the subject's physiological levels. The difference in the phase means may have been even greater if the subjects had not been anticipating the shock in the upcoming phase (phaseII) while in the first rest phase. This anticipation may be inferred from the fact that, with the exception of SCL, all measures showed higher values in the first rest phase than in the second rest phase.

The second hypothesis, that panickers would show higher resting physiological activity, was partially confirmed with HR. The panickers displayed a significantly higher HR across all three phases of the experiment.

It is interesting to note that even while stressed, the nonpanicker group did not even reach the resting levels of the panicker group. This was also found by Rabavilas et. al

(1977) in a similar paradigm using anxious patients and normal controls.

An anxiety by group interaction was also found for HR in the second rest phase. The subjects were equated at the low trait anxiety level, and once again the panickers had a higher resting HR than nonpanickers. Although this interaction did not occur in the overall ANOVA, the results do add further support for the hypothesis that panickers would show higher resting physiological activity.

Although only HR measures showed panickers to have significantly higher resting physiological activity, the data do add some support to the view that panic disorder involves a biological predisposition towards states of autonomic hyperarousal which follows a progression from early prodromal subpanic symptoms to fullscale panic disorder (Roberts, 1984). While the panickers in this study did not show elevated resting levels for all physiological measures as anxiety patients have, they did show some increased physiological activity suggesting some degree of heightened physiological functioning.

The third hypothesis, that panickers would be more physiologically reactive than nonpanickers to the threat of shock was not supported in this study. There was no significant difference in reactivity between these groups on

any measure. It is possible that the higher reactivity of anxiety patients to stress in the study by Rabavilas et. al. (1977) may have been due to increased symptom self perception resulting from their high resting physiological state. Since high physiological arousal can enhance self perception of symptoms, the disturbing sensations which are often associated with states of panic, may have prompted increased anxiety (Barlow, 1986). This further increase in anxiety would in turn result in additional bodily sensations, via a feedback loop, to further increase arousal during such states of stress.

Since the panickers in this study may be just beginning to display signs of autonomic dysregulation (HR), their overall level of arousal may not have been sufficient to affect detection of bodily sensations that would culminate in further increases in arousal.

The fourth hypothesis, that there would be no difference between high trait anxious and low trait anxious subjects in physiological reactivity to the threat of shock was confirmed in this study. These subjects did not show any differential reactivity for any measure. These results support the past research by Katkin (1965,1966) who also found no difference in reactivity between high and low anxious subjects. These results would have been particularily interesting if there had been differential

reactivity to the threat of shock between the panicker and nonpanicker groups. Had this occurred, we would have had more evidence to show that the PAQ is measuring something different from trait anxiety. As it is though, the group differences in resting HR do suggest that panickers are different from nonpanickers on a dimension that is not accounted for by trait anxiety. This may indicate that the experience of panic attacks is distinct from the experience of high trait anxiety. Since low anxious panickers have been identified, this is not an unreasonable interpretation. If panic attacks are merely another symptom of high trait anxiety, one would not expect to find low trait anxious panickers.

Another interesting finding that arose from the overall analysis of the data was an anxiety effect in SBP. It was found that low anxious subjects actually had higher SBP than high anxious subjects across all phases. This seems rather counter-intuitive, in that one would expect the high anxious subjects to have the higher SBP. However, a study by Averill and Rosenn (1972) found similar results. The authors investigated coping strategies and psychophysiological stress reactions to the threat of shock, and found that subjects who had nonvigilant coping strategies tended to have low trait anxiety and showed higher physiolgical arousal (HR,SCL) than subjects who had

vigilant coping stategies and who tended to have high trait anxiety.

Perhaps the low anxious subjects in the present study also tended to have a nonvigilant coping strategy, such as avoidant thoughts or denial, and thus demonstrated the higher arousal level as seen in the Averill and Rosenn (1972) study. The analysis of the thought content of subjects awaiting the electric shock in the present study does not support this hypothesis. However, coping style was not the focus of this study, and may not have been adequately assessed. The PEQ was formulated mainly for the purpose of assessing the subject's belief in the threat of shock, and thus may be an inadequate tool for formulating an accurate coping profile.

Finally, the analysis of the state anxiety scores for panickers and nonpanickers supported the hypothesis that panickers would be more sensitive to threat than nonpanickers, and thus report higher state anxiety than nonpanickers at the start of the shock anticipation period (phase II). This may indicate that panickers in this study, like the anxiety patients in Raskin's (1973) study, are more sensitive to danger or threat related events.

The HSCL-90 also revealed interesting differences between panickers and nonpanickers paralleling the differences

between panic disorder patients and normal controls. The panickers in this study had significantly higher scores than nonpanickers on subscales that are descriptive of actual anxiety patients (Anxiety, Somatization, Depression, & Interpersonal Sensitivity).

A difference between high anxious and low anxious subjects was also found with the HSCL-90. High trait anxiety subjects scored higher than low trait anxiety subjects on all of the 9 subscales. This result does not negate the group difference between panickers and nonpanickers since these groups did not differ in trait anxiety level.

The results of this study contribute some support to the view that panic disorder involves a biological predisposition toward states of autonomic dysregulation. Although significant effects were only found in the resting levels of HR, the panickers in this study were actually only infrequent panickers and autonomic hyperarousal may be just awakening in these individuals.

In addition to the resting level HR differences, the panicker group can also be differentiated from nonpanickers in psychological attributes that are also characteristic of anxiety patients. From this data one can see that panickers can be viewed as distinct from nonpanickers and further research with these individuals is needed to illuminate the underlying mechanisms.

As habituation deficits, in conjunction with high arousal, are said to contribute to the experience of panic attacks,it would also be fruitful to conduct a habituation study with infrequent panickers. Since the present study demonstrated that infrequent panickers may show elevated HR levels, these individuals might also show a deficit in habituation of orienting response of this measure as well. This data would add further support to the arousal-habituation deficit theory of chronic anxiety states.

Barlow's current treatment strategy of panic disorder actually follows the principles of this theory. The protocol involves lowering arousal through relaxation, and subsequent habituation to the alarming internal sensations that accompany panic attacks through repeated exposure to these stimuli.

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APPENDICES

Appendix A

Panic Attack Questionnaire (PAQ)

Dear Introductory Psychology Student:

We would like to thank you for agreeing to participate in our research project. This is an exploratory study- we are investigating the incidence of panic attacks in a university population. We want to learn more about your experience of anxiety and whether you have ever had a panic attack. A panic attack has been defined as "the sudden onset of intense apprehension, fear, or terror, often associated with feelings of impending doom. Some of the most common symptoms experienced during an attack are: dizziness, shortness of breath, chest pain or discomfort and trembling or shaking."

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

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

We are intending to do some follow-up studies at a later date. If you are still willing to help us in this subsequent phase of our research program, please indicate your willingness to be contacted by filling in your name and telephone number in the appropriate spaces on page 1 of the questionnaire. This will provide you with additional research credit hours or financial remuneration.

Thank you for your cooperation.

Sincerely,

L. Sandler, Ph.D.

K. Wilson, Ph.D.

Appendix II: Panic Attack Inventory

If you are willing to participate in a follow-up study, please fill in your name and telephone number. Doing so, does not commit you to taking part in a further study.

NAME

TELEPHONE NUMBER

Please complete all questions. Your responses will be kept confidential.

AGE

SEX

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

A. Here is a brief description of one woman's experience with a panic attack.

And then her heart struck the inside of her chest hard, once, twice, again and again, gathering speed, smiting her blindly as though pursued and frantic, in its terror attacking her.

Oh Christ oh God, she thought, I'm having a heart attack.

It pounded inside her chest, panicked and out of control, trying to batter its way out; the blood would be shooting through her veins in a torrent, her ribs would be broken, her heart would burst.

Oh my God, she thought, and pushed her chair away from the table.

The waitress hurried over and beamed a smile upon her. "Will that be all?"

Oh Jesus, thought Leona. It doesn't show.

The waitress scribbled a total on the bill and laid it face down on the table. "Have a good evening", she said.

The beat of her heart was faster and louder, increasing in intensity, but there was no pain. That must come next,

thought Leona and she stood up. I can still stand, she thought. Oh God, she thought, just let me get out of here.

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

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

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

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

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

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

_____ months _____ years

4. Have the panic attacks become more frequent recently?

Yes No

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

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

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

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

b) _____ when receiving injections or minor surgery

c) _____ eating or drinking with other people

d) _____ hospitals or visits to doctor

e) _____ travelling alone by bus or coach

f) _____ walking alone in busy streets

g) _____ being watched or stared at

h) _____ going into crowded shops

i) _____ talking to people in authority

j) _____ sight of blood

k) _____ being criticized

l) _____ going alone far from home

m) _____ thought of injury or illness

n) _____ speaking or acting to an audience

o) _____ large open spaces

p) _____ going to the dentist

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

r) _____ during or following relaxation

s) _____ during or following exercise

t) _____ sleeping

u) _____ while under the influence of alcohol or drugs

v) _____ prior to or during tests or exams

w) _____ while driving a car

x) _____ walking alone at night

y) _____ while experiencing high levels of stress

z) _____ during sexually intimate situations

aa) _____ during a family crisis

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

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

dd) _____ while meeting stranger(s)

ee) _____ being in an enclosed area

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

gg) _____ other (please explain)

7. When a panic attack occurs, generally what is the time period between the onset of the attack and when the panic is most intense?
   a. almost immediate
   b. very rapid (less than 10 minutes)
   c. moderately rapid (10-30 minutes)
   d. moderately slow (30 minutes-1 hour)
   e. slowly (more than one hour)

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

	Does not Occur	Mild	Moderate	Severe	Very Severe
a. dyspnea (difficulty breathing)	0	1	2	3	4
b. palpitations (heart pounding)	0	1	2	3	4
c. chest pain or discomfort	0	1	2	3	4
d. choking or smothering sensations	0	1	2	3	4
e. dizziness, vertigo, or unsteady feelings	0	1	2	3	4
f. feelings of unreality	0	1	2	3	4
g. paresthesias (tingling in hands or feet)	0	1	2	3	4
h. hot and cold flashes	0	1	2	3	4
i. sweating	0	1	2	3	4
j. faintness	0	1	2	3	4
k. trembling or shaking	0	1	2	3	4
l. fear of dying (or being seriously ill)	0	1	2	3	4
m. fear of going crazy	0	1	2	3	4
n. fear of doing something uncontrolled	0	1	2	3	4

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

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

   b. 10 - 30 minutes

c. 30 minutes to one hour

d. several hours

e. more than one day

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

YES _____ NO _____

If yes, please explain:

_____

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

YES _____ NO _____

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

YES ___ NO ___ difficulties at work

YES ___ NO ___ loss of a loved one

YES ___ NO ___ birth of a child

YES ___ NO ___ marital/family problems

YES ___ NO ___ life-threatening situation

YES ___ NO ___ other stressful event (please specify)

Instructions Below are a list of problems and complaints that people sometimes hav
Please read each one carefully.
After you have done so, please circle one of the numbers to the right that best describes
HOW MUCH THAT PROBLEM HAS BOTHERED YOU DURING THE PAST WEEK INCLUDIN TODAY
Circle one number for each problem and do not skip any items.
If you change your mind, erase your first choice completely.
Please read the example below before beginning.

Example:

HOW MUCH WERE YOU BOTHERED BY:

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
1. Backaches	0	1	2	3	4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
1. Headaches	0	1	2	3	4
2. Nervousness or shakiness inside	0	1	2	3	4
3. Unwanted thoughts, words, or ideas that won't leave your mind	0	1	2	3	4
4. Faintness or dizziness	0	1	2	3	4
5. Loss of sexual interest or pleasure	0	1	2	3	4
6. Feeling critical of others	0	1	2	3	4
7. The idea that someone else can control your thoughts	0	1	2	3	4
8. Feeling others are to blame for most of your troubles	0	1	2	3	4
9. Trouble remembering things	0	1	2	3	4
10. Worried about sloppiness or carelessness	0	1	2	3	4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
11. Feeling easily annoyed or irritated	0	1	2	3	4
12. Pains in heart or chest	0	1	2	3	4
13. Feeling afraid in open spaces or on the streets	0	1	2	3	4
14. Feeling low in energy or slowed down	0	1	2	3	4
15. Thoughts of ending your life	0	1	2	3	4
16. Hearing voices that other people do not hear	0	1	2	3	4
17. Trembling	0	1	2	3	4
18. Feeling that most people cannot be trusted	0	1	2	3	4
19. Poor appetite	0	1	2	3	4
20. Crying easily	0	1	2	3	4
21. Feeling shy or uneasy with the opposite sex	0	1	2	3	4
22. Feeling of being trapped or caught	0	1	2	3	4
23. Suddenly scared for no reason	0	1	2	3	4
24. Temper outbursts that you could not control	0	1	2	3	4
25. Feeling afraid to go out of your house alone	0	1	2	3	4
26. Blaming yourself for things	0	1	2	3	4
27. Pains in lower back	0	1	2	3	4
28. Feeling blocked in getting things done	0	1	2	3	4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
29. Feeling lonely	0	1	2	3	4
30. Feeling blue	0	1	2	3	4
31. Worrying too much about things	0	1	2	3	4
32. Feeling no interest in things	0	1	2	3	4
33. Feeling fearful	0	1	2	3	4
34. Your feelings being easily hurt	0	1	2	3	4
35. Other people being aware of your private thoughts	0	1	2	3	4
36. Feeling others do not understand you or are unsympathetic	0	1	2	3	4
37. Feeling that people are unfriendly or dislike you	0	1	2	3	4
38. Having to do things very slowly to insure correctness	0	1	2	3	4
39. Heart pounding or racing	0	1	2	3	4
40. Nausea or upset stomach	0	1	2	3	4
41. Feeling inferior to others	0	1	2	3	4
42. Soreness of your muscles	0	1	2	3	4
43. Feeling that you are watched or talked about by others	0	1	2	3	4
44. Trouble falling asleep	0	1	2	3	4
45. Having to check and doublecheck what you do	0	1	2	3	4
46. Difficulty making decisions	0	1	2	3	4
47. Feeling afraid to travel on buses, subways, or trains	0	1	2	3	4

48. Trouble getting your breath 0 1 2 3 4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
49. Hot or cold spells	0	1	2	3	4
50. Having to avoid certain things, places, or activities because they frighten you	0	1	2	3	4
51. Your mind going blank	0	1	2	3	4
52. Numbness or tingling in parts of your body	0	1	2	3	4
53. A lump in your throat	0	1	2	3	4
54. Feeling hopeless about the future	0	1	2	3	4
55. Trouble concentrating	0	1	2	3	4
56. Feeling weak in parts of your body	0	1	2	3	4
57. Feeling tense or keyed up	0	1	2	3	4
58. Heavy feelings in your arms or legs	0	1	2	3	4
59. Thoughts of death or dying	0	1	2	3	4
60. Overeating	0	1	2	3	4
61. Feeling uneasy when people are watching or talking about you	0	1	2	3	4
62. Having thoughts that are not your own	0	1	2	3	4
63. Having urges to beat, injure or harm someone	0	1	2	3	4
64. Awakening in the early morning	0	1	2	3	4
65. Having to repeat the same actions such as touching, counting, washing	0	1	2	3	4
66. Sleep that is restless or disturbed	0	1	2	3	4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
67. Having urges to break or smash things	0	1	2	3	4
68. Having ideas or beliefs that others do not share	0	1	2	3	4
69. Feeling very self-conscious with others	0	1	2	3	4
70. Feeling uneasy in crowds, such as shopping or at a movie	0	1	2	3	4
71. Feeling everything is an effort	0	1	2	3	4
72. Spells of terror or panic	0	1	2	3	4
73. Feeling uncomfortable about eating or drinking in public	0	1	2	3	4
74. Getting into frequent arguments	0	1	2	3	4
75. Feeling nervous when you are left alone	0	1	2	3	4
76. Others not giving you proper credit for your achievements	0	1	2	3	4
77. Feeling lonely even when you are with people	0	1	2	3	4
78. Feeling so restless you couldn't sit still	0	1	2	3	4
79. Feelings of worthlessness	0	1	2	3	4
80. Feeling that familiar things are strange or unreal	0	1	2	3	4
81. Shouting or throwing things	0	1	2	3	4
82. Feeling afraid you will faint in public	0	1	2	3	4

	Not at All	A Little Bit	Moderately	Quite A Bit	Extremely
83. Feeling that people will take advantage of you if you let them	0	1	2	3	4
84. Having thoughts about sex that bother you a lot	0	1	2	3	4
85. The idea that you should be punished for your sins	0	1	2	3	4
86. Feeling pushed to get things done	0	1	2	3	4
87. The idea that something serious is wrong with your body	0	1	2	3	4
88. Never feeling close to another person	0	1	2	3	4
89. Feelings of guilt	0	1	2	3	4
90. The idea that something is wrong with your mind	0	1	2	3	4

DIRECTIONS: A number of statements which people have used to describe themselves are given below.
Read each statement and then circle the number to the right of the statement that indicates how you GENERALLY FEEL
There are no right or wrong answers.
Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	almost never	sometimes	often	almost always
1. I feel pleasant	1	2	3	4
2. I tire quickly	1	2	3	4
3. I feel like crying	1	2	3	4
4. I wish I could be as happy as others seem to be	1	2	3	4
5. I am losing out on things because I can't make up my mind soon enough	1	2	3	4
6. I feel rested	1	2	3	4
7. I am "calm, cool, and collected"	1	2	3	4
8. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9. I worry too much over something that really doesn't matter	1	2	3	4
10. I am happy	1	2	3	4
11. I am inclined to take things hard	1	2	3	4
12. I lack self confidence	1	2	3	4
13. I feel secure	1	2	3	4
14. I try to avoid facing a crisis or difficulty	1	2	3	4
15. I feel blue	1	2	3	4
16. I am content	1	2	3	4
17. Some unimportant thoughts runs through my mind and bothers me	1	2	3	4
18. I take disappointments so keenly that I	1	2	3	4

can't put them out of my mind				
19. I am a steady person	1	2	3	4
20. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Appendix B

Instructions to the Subjects

In this study we are investigating the effect of a strong but brief electric shock on anxiety level. The first phase of the experiment will be a rest period, where you will merely relax for 15 minutes. The second phase will last 10 minutes, at the end of which you will receive one strong brief finger shock that will not be harmful. The third phase will last 10 minutes and is another relaxation phase.

Throughout these phases, we will be measuring your heart rate and skin conductance.

You will receive one credit for your participation.

Appendix C

Consent form

Date _____

I have been advised that in this experiment, I will be given one electric shock, and agree to be a participant.

Signature _____

Appendix D

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

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

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

Appendix E

The results of an experiment are more meaningful to us if we know what your ideas, thoughts, and understandings of the experiment were. Please answer each of the questions on the following pages frankly and honestly. Please answer them in their numbered order, and DO NOT go on to the next question until you have given an answer to the previous question. Do not go back to a question once you have started on the next one. Remember, we want you to answer the questions as accurately as you can.

1. What do you think was the purpose of this experiment?

2. Why do you think you were threatened with an electric shock?

3. What do you think was the purpose of the 10 minute waiting period before the expected shock?

4. What did you think about during the 10 minute period before the expected shock?

5. Did you do anything to avoid thinking about the upcoming shock? If so, what?

6. If you tried to avoid thinking about the shock, how successful were you in doing this?

not at all					very successful
1	2	3	4	5	6

7. Did it ever occur to you that you would not actually receive a shock?

7b. If so, at what point did you seriously doubt the occurence of the shock? Please check one of the following.

At the beginning of the experiment _____

During the first rest phase _____

During the shock phase (phase 2) _____

At the end of the shock phase _____

At the completion of the experiment _____

Other (please indicate) _____

Appendix F

Did you experience any of the following symptoms while you waited for the electric shock.

	Does not Occur	Mild	Moderate	Severe	Very Severe
a. dyspnea (difficulty breathing)	0	1	2	3	4
b. palpitations (heart pounding)	0	1	2	3	4
c. chest pain or discomfort	0	1	2	3	4
d. choking or smothering sensations	0	1	2	3	4
e. dizziness, vertigo, or unsteady feelings	0	1	2	3	4
f. feelings of unreality	0	1	2	3	4
g. paresthesias (tingling in hands or feet)	0	1	2	3	4
h. hot and cold flashes	0	1	2	3	4
i. sweating	0	1	2	3	4
j. faintness	0	1	2	3	4
k. trembling or shaking	0	1	2	3	4
l. fear of dying (or being seriously ill)	0	1	2	3	4
m. fear of going crazy	0	1	2	3	4
n. fear of doing something uncontrolled	0	1	2	3	4

Appendix G

Summary ANOVA Table for the Within Subjects Factor for Each Physiological Variable

Measure	Source	SS	df	MS	F	Prob	
HR	phase	3073.2	2	1536.6	35.85	.0000	.44
	error	3772.1	88	42.9			
MAP	phase	844.0	2	422.0	13.28	.0000	.89
	error	2795.7	88	29.2			
SYST	phase	2009.4	2	1004.7	23.59	.0000	.34
	error	3747.4	88	42.6			
DBP	phase	402.6	2	201.3	8.21	.0005	.15
	error	2158.9	88	24.5			
SCL	phase	557.2	2	278.6	37.19	.0000	.45
	error	659.1	88	7.5			
SCF	phase	4006.6	2	2003.3	59.94	.0000	.44
	error	2941.0	88	33.4			

Appendix H

Summary ANOVA Table for Between Subjects Effects for Each Physiological Variable

Measure	Source	SS	df	MS	F	Prob.	
HR	group	15355.0	1	15355.0	10.59	.0022	.17
	anxiety	3046.3	1	3046.3	2.10	.1542	.02
	error	63771.4	44	1449.3			
MAP	group	460.8	1	460.8	.98	.3276	.00
	anxiety	1269.4	1	1269.4	2.70	.1100	.04
	error	20689.9	44	470.2			
SYST	group	224.5	1	224.5	.29	.5936	.00
	anxiety	4351.3	1	4351.3	5.60	.0224	.09
	error	34185.4	44	776.9			
DBP	group	680.6	1	680.6	1.10	.3001	.00
	anxiety	101.3	1	101.3	.16	.6878	.00
	error	27232.2	44	618.9			
SCL	group	30.9	1	30.9	.18	.6719	.00
	anxiety	262.3	1	262.3	1.54	.2208	.01
	error	7479.5	44	170.0			
SCF	group	145.8	1	145.8	.50	.4847	.00
	anxiety	594.1	1	594.1	2.70	.1100	.04
	error	20689.9	44	293.6			