Psychophysiological Assessment of Panic Disorder

Kent A. Koehn

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PSYCHOPHYSIOLOGICAL ASSESSMENT OF PANIC DISORDER

by

Kent A. Koehn

A Thesis
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Master of Arts
Department of Psychology

Western Michigan University
Kalamazoo, Michigan
August 1989
This study compared physiological and psychological measures between 9 subjects meeting the DSM-III-R criteria for panic disorder (PD) and 9 control subjects to identify psychophysiological differences that might be relevant to the etiology and maintenance of panic attacks. The subjects were assessed through a number of self-report measures and across three laboratory sessions involving five experimental conditions: baseline, role play, relaxation, mental arithmetic, and cold pressor. We measured electrodermal (EDG) and electromyographic activity (EMG), heart rate (HR), and blood volume pulse (BVP) in terms of absolute values, reactivity, and habituation. Both groups were also assessed in their ability to accurately estimate changes in HR and EMG. The results indicated that PD subjects endorsed significantly greater numbers of psychological symptoms and demonstrated greater EDG and BVP levels across all experimental conditions with the differences being significant during the baseline and relaxation tasks. The PD group did not differ from the control group in physiological reactivity nor within-task habituation. The PD group consistently evidenced EDG sensitization versus habituation among the control group across the repeated assessment sessions. Neither group was accurate in estimating EMG and HR changes but the PD group made greater numbers of errors in overestimating EMG change.
Acknowledgements

This thesis owes much to so many who have contributed in multiple ways. I would like to thank R. Wayne Fuqua for the guidance necessary to make this project come to fruition. I want to express my gratitude to M. Michele Burnette without whose inputs this study would not have gotten off the ground. I would like to acknowledge my many peers who helped at all stages of the research. I particularly would like to thank Sue Keller, Carman Stark, Michael Winter, and Rita Kenyon-Jump. I am especially thankful to my friend and wife, Denise, who has provided endless support.

Kent A. Koehn
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Psychophysiological assessment of panic disorder

Koehn, Kent Allen, M.A.
Western Michigan University, 1989
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INTRODUCTION

In recent years panic disorder (PD) has been the focus of increasing attention by health professionals and researchers. Panic is defined as "the sudden onset of intense apprehension, fear, or terror. Often there is a feeling of impending doom." (DSM-III-R; American Psychiatric Association, 1987, p. 236). The most common symptoms include palpitations, dyspnea, chest pain, trembling, choking or smothering sensations, dizziness, faintness, paresthesia, sweating, and feelings of unreality. In addition to its associated distress and the diminished life activity through patterns of learned avoidance for those with panic attacks, the disorder is believed to result in costly medical evaluations e.g., coronary arteriography, and treatments to identify or alleviate the somatic basis for the symptoms (Fackelman, 1989). Excessive mortality from suicides and cardiovascular disease associated with PD has also been reported (Coryell, Noyes, & Clancy, 1982). While precise data on the prevalence of PD is unknown, surveys indicate that PD occurs in 1% of the population (Meyers et al., 1984) with the highest frequency of occurrence among women (Ilfeld, 1979).

Despite the frequency of this disorder and the recent abundance of treatment research and theoretical analyses, little empirical evidence on the psychophysiological nature of panic exists. In an effort to understand the physiological characteristics and putative physiological mechanisms underlying PD, a number of researchers have
conducted descriptive studies of patients who experience panic attacks.

Sudden and marked increases in heart rate, skin conductance, and frontalis electromyograph (EMG) have been documented in laboratory case studies during the spontaneous occurrence of panic (Cohen, Barlow, & Blanchard, 1985; Lader & Mathews, 1970). Other researchers have monitored physiological processes while inducing panic attacks with sodium lactate (Kelly, Mitchell-Heggs, & Sherman, 1971; Pitts & McClure, 1967), hyperventilation (Rapee, 1986; Clark, Salkovskis, & Chalkey, 1985), caffeine (Uhde, Boullenger, Jimerson, & Post, 1984), and even during relaxation (Cohen, Barlow, & Blanchard, 1985; Heide & Borkovec, 1983). These studies have shown the wide spectrum of laboratory conditions that can evoke panic while offering descriptive data on the physiology of panic attacks. It is unclear the extent to which these laboratory precipitators to panic compare with the mechanisms that provoke panic in naturalistic environments.

Ambulatory monitoring of heart rate and skin temperature has provided additional assessment information on the physiology associated with self-reports of panics (Freedman, Ianni, Ettequwi, & Puthezhath, 1985; Taylor et al., 1986). Freedman et al. (1985) found substantial heart rate increases and finger temperature changes during panic attacks but not during non-panic high anxiety periods. Taylor et al. (1986) reported similar physiological changes, primarily heart rate increases, during naturally occurring panic attacks although this relationship was not consistently observed across all participants. Over one-third of the reported panics in this study occurred without
corresponding heart rate changes, supporting the hypothesis that it is not heart rate alone but a complex interplay of a number of symptoms (e.g., chest tightness, difficulty breathing, hot and cold flashes, feelings of doom, and dizziness) that lead to the self-identification of a panic attack.

Several researchers have assessed the physiological reactions of panic disorder patients to laboratory-based stressors of a cognitive nature. Barlow et al. (1984) found that when compared to patients diagnosed with Generalized Anxiety Disorder, Panic Disorder patients exhibited greater heart rate and muscle tension increases in response to laboratory stress-induction procedures involving mental arithmetic and stressful imagery tasks. Beck and Scott (1987) found that frequent panic subjects, when compared to infrequent panickers, demonstrate greater reactivity in muscle activity and skin conductance during "cognitive" imagery stressor tasks (e.g., neutral scene and hospitalization imagery) than during performance tasks (e.g., paced arithmetic and signal detection). This finding appears to support cognitive models of panic (e.g., Beck & Emery, 1985; Clark, 1986) that emphasize the role of cognitive factors as precipitating events in panic arousal.

A number of panic induction studies have additionally reported that patients who panic demonstrate higher levels of anxiety than normal controls at baseline (Freedman et al., 1984; Kelly et al., 1971; Liebowitz, et al., 1984; and Rainey, et al., 1984a, 1984b) and higher levels of heart rate, systolic blood pressure, and forearm blood flow (Kelly et al., 1971; Liebowitz et al., 1984, 1985). It has
also been found that high anxiety subjects habituate more slowly to auditory stimuli than do normals (Lader & Wing, 1966). Such a pattern of physiological arousal without habituation may function in the development of panic (Lader & Wing, 1966).

Somatic manifestations are common in most anxiety disorders and have also been frequently assessed for their role in panic. Panic patients more frequently report a greater number and severity of anxiety-related somatic events than do Generalized Anxiety Disorder patients (Barlow et al., 1985; Hoehn-Saric, 1981). The number of symptoms reliably experienced by an individual across a panic attack is also quite variable (Rapee, Craske, & Barlow, 1988). This variability would seem to support a non-biological model for panic. Although no consistent physiological cues for the inception of panic were identified in these studies, it has been speculated that detecting physiological changes plays an interactive role in triggering panic (Barlow, 1986; Beck & Emery, 1985; Clark, 1986; Margraf, Ehlers, & Roth, 1986; & Rachman, 1983).

The potential role of vigilance for physiological changes in triggering panics is supported by research showing that Agoraphobics with a history of panic attacks and Panic Disorder subjects demonstrate a marked sensitivity to physiological changes and bodily sensations (Chambless, 1982; Chambless & Goldstein, 1981; King, Margraf, Ehlers, & Maddock, 1986). Sensitivity to these physiological changes is thought to trigger panic attacks through an interoceptive and semantic conditioning process that results in a positive feedback loop (Ackerman & Sachar, 1974; Beck & Emery, 1985; Clark et al., 1985;
Denny, 1976; Evans, 1972; Lader, 1975; Mathews, Gelder, & Johnson, 1981; Razran, 1961). According to this model, patients may anticipate aversive events and experience physiological responses such as dyspnea, tachycardia, sweating, etc. The level of physiological arousal is thought to escalate partly as a function of the lack of habituation to anxiety-provoking stimuli and partly as a function of attending to the symptom until a full-blown panic attack occurs.

Research on the ability of PD patients to discriminate physiological changes that characterize panic attacks is inconclusive. Roy-Byrne, Uhde, Post, King, and Buchsbaum (1985) found no differences between panic disorder subjects and normals in discriminating a range of painful stimuli. Beck and Scott (1987) found that a group of subjects with panic disorder were less aware of autonomic sensations than a group of subjects who experience panic attacks but not frequent enough to meet the criteria for panic disorder. Neither panic group's somatic symptom reports were correlated with significant physiological reactivity. Additionally, Ehler, Margraf, Roth, Taylor, and Birbaumer (1988) reported that PD subjects and controls did not differ on a test of cardiac awareness. PD subjects did, however, show higher levels of arousal than normal controls to false heart rate feedback suggesting a cognitive component (e.g., cognitive labeling of the cardiac feedback) played a role in eliciting higher levels of arousal in the PD subjects. The absence of differences on the cardiac awareness task does not support contentions that panic patients are more sensitive to physical changes. Unfortunately, Ehlers et al. (1988) did not adequately address whether panic patients are differentially sensitive
to actual physiological changes. The role of cognitions in panic is also left for further research given the non-significant cardiovascular increases that were found among the panic patients who received false feedback. As an extension of these unanswered questions, assessing the ability to make discriminations of physiological changes may be more adequately determined by inducing such changes directly.

Current explanations of the physiological and psychological dimensions of panic are inconclusive. While differences have been found between panic patients and other anxiety disordered and normal subjects, the research is plagued by a number of problems. Many of the findings are based upon retrospective reports of questionable validity and through global ratings of symptoms. Laboratory anxiety-induction tests can be challenged for their artificiality, lack of control for subject baseline differences, inadequate assessment of relevant dependent variables, confounds in instructions given, and their global nature of subjective anxiety assessment. Inconsistent results have also been found across studies in both physiological and psychological measures as well as across and within subjects. Subject selection criteria have additionally been poorly defined or broadly inclusive, resulting in the findings being difficult to interpret and extend. It remains unclear the extent to which panic sufferers experience the numerous symptoms often reported, the degree of their reactivity to stress, and their ability to detect physiological changes.

The present research introduced non-pharmacological stressful situations to PD subjects and normal controls to identify physiologi-
cal and cognitive differences that characterize PD and that might be relevant to the etiology of panic attacks. This experiment examined a broader range of stressor conditions than prior studies including tasks designed to approximate those of naturalistic settings. Multiple and repeated measures were obtained across symptom self-reports, physiological variables, and estimates of physical change. Data were analyzed in absolute values and also adjusted for baseline differences as a control for individual differences. It was hypothesized that PD subjects would endorse a greater number and intensity of panic symptoms and show heightened physiological reactivity and less habituation to the stressor tasks than normals. It was further hypothesized that PD subjects would demonstrate greater accuracy in detecting such physiological changes.
METHOD

Subjects

Six female and three male subjects aged 23–37 (mean = 31.1) meeting the criteria for Panic Disorder with mild or no agoraphobic avoidance and a normal control group matched for sex and age (mean = 32.4) served as participants in this research. The mean length of time for experiencing panics was 8.8 years (range = 1.5 to 20 years) among the panic subjects. Both groups were interviewed with the Anxiety Disorders Interview Schedule-Revised (ADIS-R; Di Nardo, O'Brien, Barlow, Waddell, and Blanchard, 1983). Normal control subjects were excluded if any psychopathology was evident from the interview or if any first degree relative had a history of panic attacks. Subjects meeting criteria for additional diagnoses of depression, agoraphobia, generalized anxiety disorder, or social phobia were excluded. Subjects were excluded if they were taking tricyclic or phenothiazine medication, were pregnant, or had active cardiovascular, renal, endocrine, or neurologic disease. Two panic disorder subjects were infrequent users of alprazolam (Xanax) at a low dosage and documented this information throughout the study. Laboratory sessions were held after medication-free periods of 48 hours or more. (The mean elimination half-life of alprazolam is 12–15 hours, Medical Economics Company, 1988, p. 2150). Participants were recruited through newspaper article and advertisements requesting
persons experiencing panic attacks to participate in assessment and treatment research for panic disorder. All subjects were required to give written informed consent after the assessments and procedures had been fully explained. All subjects received $10.00 for participating.

Setting

Screening interviews were held individually in an office. All sessions were conducted in a small laboratory room equipped with a comfortable chair for the subject and a chair for the experimenter or role play actor. The room was equipped with an intercom system for communicating instructions and an observation window. Recording equipment was located behind the subject in an adjacent room.

Apparatus

Electromyographic activity (EMG) and skin conductance (EDG) signals were measured through attachment of silver-silver chloride electrodes and measured with a J & J Electronic component system (EMG Model M-52; EDG Model R-72; and Digital Integrator Model D-200). Blood volume pulse (BVP) and heart rate (HR) were measured with a photoelectric plethysmographic transducer (Grass Model PPS) and amplified by a Grass Model 7P1 preamplifier. The measures were recorded on a Grass oscillograph at a chart speed of 3mm/sec.

Forehead EMG was measured through attachment of electrodes to alcohol-cleansed skin on the forehead directly over each pupil at a distance approximately 2.5cm above each eyebrow.
Electrodermal activity was measured through placement of elec-
trodes on the distal phalanxes of the middle two fingers of the non-
dominant hand.

Heart rate and blood volume pulse were measured through place-
ment of a single infra-red light photocell plethysomograph attached
with a Velcro strip to the distal phalanx of the non-dominant index
finger.

Procedure

General Procedure and Instruction

The subjects were comfortably seated in the laboratory and the
recording equipment attached. The experimenter read instructions for
each phase and then monitored and recorded the physiological measures
from the adjacent room. The experimental conditions, their duration,
and specific instructions given to each subject were as follows:

1. Adaptation—approximately 10 minutes—"Please sit quietly."

2. Baseline—3 minutes—"Please sit quietly with your eyes closed
for the next few minutes."

3. Stressor tasks—(a) mental arithmetic—3 minutes—
"Keeping your eyes closed, please count backwards from 30 to 0 by
3's." then "Keeping your eyes closed, please start at 300 and count
backwards by 7's." (b) cold pressor—1–7 minutes—"When I say start,
I'd like you to place your right hand up to the wrist in the ice
water and keep it there until it hurts so badly you want to remove it
or until I tell you to remove it. Tell me as soon as you remove your
hand if you do so. Start." (c) stressful role plays-3 minutes-
"During this phase I will read you a description of a potentially stressful event. An actor will briefly role play this situation with you. I would like you to respond to this person as you normally would in such a situation. You are not being evaluated on your acting ability and there is no right or wrong way of responding. Please continue your interaction with the actor until I say stop."

4. Relaxation phase-5 minutes. The subject listened to a tape recording of relaxation exercise derived from Bernstein and Borkovec's (1973) manual for progressive muscle relaxation. The tape asked the subject to achieve relaxation by the recall method where four major muscle groups were described sequentially without the subject exerting muscle tension.

Role plays

A combination of standardized and individualized role plays constructed from each subject's response to the Daily Hassles Scale (Kanner, Coyne, Schaefer, & Lazarus, 1971) were employed. The standardized role plays consisted of an approximately 3-minute interaction of a stressful situation that commonly is encountered in everyday life. The individualized role plays had the same format but approximated idiosyncratic stressors identified on the Daily Hassles Scale. The subjects were encouraged to respond to the situation as if it were real. For example, one role play required the subject to interview for a new job; another required the subject to confront a landlord about a recent rent increase.
Role play actors were varied across subjects and sessions. The actors received a written scenario of each role play clarifying the manner in which they should act and suggesting specific verbal responses to be made in the role play. Actors were instructed to arouse the subject by progressively obstructing and challenging the subjects' statements. Actors were trained by observing a video tape and reading scenarios demonstrating various role play situations and actor responses. They were then required to rehearse the role plays prior to the study and were directed to consistently present each scenario across all subjects. Subjects rated each role play at the end of the session on a Likert scale according to its stressfulness, the degree the role play was typical of life situations encountered and their reaction to them, and the degree to which the role play actor's responses to the subject were typical of how others generally respond to them in similar situations.

Experimental Design

During each assessment session, three sequences of stressor tasks were employed and alternated across three experimental sessions with each subject receiving all three arrangements with intervening baselines. The three sequences were the following: (a) role play, relaxation, mental arithmetic, and cold pressor; (b) mental arithmetic, role play, relaxation, and cold pressor; and (c) relaxation, mental arithmetic, role play, and cold pressor. Each subject participated in three assessment sessions to evaluate the effect of repeated exposure to the experimental stressors.
Dependent Variables

Physiological Measures

The physiological measures (EMG, EDG, HR, & BVP) were monitored for all subjects. Values for EMG and EDG were obtained every 15 sec. Heart rate and blood volume pulse were monitored continuously. Heart rate was calculated by counting 3 random ten-second intervals per minute and averaging these values into beats-per-minute (BPM). Blood volume pulse was scored by calculating the average pen deflection (in millimeters) across 6 randomly selected spikes per minute. Artifact was defined as any deflections exceeding twice the measured height of the previous deflection. When this occurred the subsequent blood flow measures were discarded for 20 sec. All subjects' BVP data were mathematically converted to equal microvolt sensitivities to permit absolute comparisons. Each of the four physiological measures were further averaged into one value per condition.

Percentage change scores from baseline as a measure of physiological reactivity were calculated for each subject to correct for individual differences in baseline levels on all data thereby providing relative as opposed to absolute change scores. Baseline values were obtained by averaging the values of the three baselines immediately preceding each stressor task. The percentage change scores were then derived by subtracting the 3-session average baseline value from the 3-session average task measure and dividing by the baseline value. Positive values represented an arousal above baseline levels for EMG, EDG, and HR. Negative values for BVP
represented reactivity.

Two measures of habituation to the experimental conditions were determined. Within-session habituation was assessed by comparing the level of each physiological measure taken during the first minute of the 3-minute sampling period with the level during the third minute of that sampling period. This measure was calculated for each task (e.g., role play, relaxation, mental arithmetic, and cold pressor) by taking the average value of each physiological variable from minute 3 minus the average value from minute 1 and dividing by the minute 1 average (minute 3 - minute 1 / minute 1). These were averaged across all three repeated assessment sessions for each subject. With this calculation, a positive number represents sensitization (increased physiological arousal) during each task across EDG, EMG, and HR, and a negative number represents habituation during the 3-minute sampling periods. Habituation, as measured by BVP, is inversely represented, i.e., negative values represent sensitization and positive represent habituation. Second, a measure of across-session habituation was obtained by calculating percentage change across each physiological variable per condition from session 1 to session 3 (session 3 average - session 1 average / session 1 average).

Heart rate and blood volume pulse measures were independently scored by two assistants and interrater reliabilities calculated across a random sample of 25% of the polygraph tracings using Pearson product-moment correlations. This resulted in correlational coefficients of .99 for heart rate and .97 for blood volume pulse.
Self-Report Measures

Questionnaire and Daily Diary Recordings

All subjects completed a battery of self-report measures at the initial assessment session. Subjects also monitored daily panics and anxiety throughout the study. These included:

1. State-Trait Anxiety Inventory (STAI, Speilberger, Gorsuch, & Lushene, 1970). This inventory measures the severity of anxiety symptoms presently and across time.

2. Hopkins Symptom Checklist (SCL-90-R, Derogatis, 1983). This is a standardized multiple-scaled instrument that measures physical and psychological symptomatology.

3. Beck Depression Inventory (BDI, Beck, 1972; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). This questionnaire has been demonstrated to be sensitive in assessment of depressive moods and was used to estimate the severity of depressive symptoms.

4. Psychosomatic Symptom Checklist (PSC, Cox, Freundlich, & Meyer, 1975). This checklist surveys the frequency and severity of 17 physical symptoms.


6. Daily self-monitoring diaries. A pocket-sized self-monitoring diary was developed for subjects to record their subjective anxiety and panic attacks throughout the study. Subjects rated their anxiety at four time periods in the day on a 0 to 8 scale (0 = calm,
2 = slight disturbance, 4 = moderate disturbance, 6 = marked disturbance, and 8 = very severe disturbance, worst panic ever). If they experienced moderate anxiety (4 or greater) at times other than the four scheduled recordings, they recorded its onset and offset, where it occurred, what was transpiring, who was present, their thoughts and feelings, and what resulted.

**Laboratory Symptom Reports**

Subjects were given a record sheet following the initial 10-minute adaptation period. At this point and after all subsequent phases, participants were asked to estimate changes in their current level of heart rate and muscle tension. Subjects circled values from -4 to +4, with -4 representing extreme decreases and +4 representing extreme increases. In addition, each subject estimated their current anxiety level on a 0-8 subjective units of disturbance scale (SUDS; 0 = no anxiety 8 = highest level imaginable). As a supplementary index of PD symptoms, subjects checked-off the presence and intensity of any of the 15 DSM-III-R symptoms of PD on a written checklist after each task.
RESULTS

Self-Report Measures

Table 1 summarizes the group means on the self-report measures. The self-report data were analyzed by using one factor analysis of variance. The PD group scored significantly higher on the STAI trait scale than the control group \( F(1,16 = 12.141), p = .0031 \). Analysis of the BDI yielded significant differences with the PD group again scoring higher \( F(1,16 = 8.515), p = .01 \). On the Psychosomatic Symptom Checklist, the panic group's scores were significantly elevated over that of the control group \( F(1,16 = 5.659), p = .0302 \). Significantly higher scores were also found on both scales of the CSAQ for the PD group: cognitive \( F(1,16 = 7.165), p = .0165 \); somatic \( F(1,16 = 17.562), p = .0007 \). The panic group scored significantly higher than the non-panic controls on each of the SCL-90-R indices: global severity index \( F(1,16 = 11.016), p = .0043 \); positive symptom index \( F(1,16 = 10.066), p = .0059 \); positive symptom distress index \( F(1,16 = 8.767), p = .0092 \).

Analysis of the average daily diary anxiety ratings showed significantly higher ratings for the PD group \( F(1,16 = 6.181), p = .0252 \). The PD subjects recorded 75 panics across 258 days of diary recording compared to 9 during 189 days of recording for the control group, which was highly significant \( F(1,16 = 19.64), p = .0004 \). The PD group reported greater numbers of symptoms across the laboratory
Table 1

Questionnaire, Daily Diary, and Session Symptom Data for Panic Disorder and Control Subjects

<table>
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<tr>
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<tr>
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<tr>
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<td>Somatic</td>
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<td>8.5</td>
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<tr>
<td>SCL-90-R</td>
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<td>Global Severity</td>
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<tr>
<td>Positive Symptoms</td>
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<tr>
<td>Symptom Distress</td>
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<tr>
<td>Daily Diary</td>
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<tr>
<td>Panics per days</td>
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<tr>
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<tr>
<td>Average anxiety</td>
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** Significant at p < .01

* Significant at p < .05

Note. STAI = State Trait Anxiety Inventory
Beck = Beck Depression Inventory
PSC = Psychosomatic Symptom Checklist
CSAQ = Cognitive Somatic Anxiety Questionnaire
SCL-90-R = Hopkins Symptom Checklist-Revised

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sessions than the control group, but this failed to reach statistical
significance $F(1,16 = 4.235), p = .0563$. Both groups rated each of
the tasks similarly on the SUDS anxiety scales. By task, these
ratings were: role play 2.2, 2.8; relaxation .8, .4; mental arithme-
tic 1.9, 3; cold pressor 1.6, 3; for panic and control group respect-
ively. On the separate (1-5) scale for rating the stressfulness of
the roleplay, the ratings were similar for both groups: 2.63 for
panic subjects and 2.73 for controls.

Untransformed Physiological Data Analyses

All individual session raw data for each experimental condition
were averaged across the three laboratory sessions to result in one
value per task for each group. These absolute values were then
analyzed separately by means of one factor analysis of variance and
Wilcoxon-Mann nonparametric analysis of variance to compare the two
groups. Panic Disorder subjects demonstrated a higher pattern of
arousal than the control group across every task and physiological
measure but these differences reached statistical significance only in
EDG and BVP. A summary of these results is displayed in Figures 1 and
2. Panic Disorder subjects showed significantly higher levels of EDG
during the Baseline and Relaxation conditions. Additionally, PD
subjects exhibited greater BVP constriction during Baseline, Roleplay,
and Relaxation tasks than the control subjects.

Baseline Conditions

Analysis of variance across the baseline conditions revealed
Figure 1. Physiological Assessment of Panic Disorder and Normal Control Subjects: EDG Values.

Figure 2. Physiological Assessment of Panic Disorder and Normal Control Subjects: BVP Values.

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significantly higher arousal among the PD group in EDG, $F(1,16 = 8.236), p = .01$ and blood volume pulse $F(1,16 = 16.246), p = .001$. All other comparisons of physiological measures were not significant although the PD subjects showed higher levels of physiological arousal.

Task Conditions

The relaxation condition resulted in significantly greater arousal for the panic subjects than the control subjects in EDG, $F(1,16 = 5.2), p = .0366$ and BVP, $Z = 2.4725, p = .0134$. Finger blood volume pulse during the role play was significantly less for the PD subjects as analyzed by the Wilcoxon test $Z = 2.1193, p = .0341$. The panic group also approached statistically significant higher elevations in EDG over the control group on the roleplay $Z = -1.766, p = .0774$ and during the mental arithmetic task $Z = -1.8543, p = .0637$.

Percentage of Change Data Analyses

Percentage of change from baseline analyses were conducted with both one factor analysis of variance and the Wilcoxon-Mann nonparametric test. For both groups, physiological reactivity was evident on all measures and across each of the stressor tasks while reductions occurred across the relaxation task. Group differences in reactivity, though, were significant for only one task and measure. The PD group showed significantly greater reduction in EMG during the relaxation task $F(1,16 = 7.10), p = .017$. During this task, the panic group exhibited greater percentage reductions in muscle tension than the
control group, 50% versus 21%, respectively. It is noteworthy that both groups exhibited similar reactivity on the stressor tasks, but the panic group displayed less percentage change than the control group on every measure. These differences approached statistical significance in EDG during role play \( Z = 1.766, p = .0774 \) and in BVP reactivity during mental arithmetic \( F(1,16 = 3.79), p = .0692 \).

Habituation

**Within-task**

Percentage of change analyses comparing minute 1 to minute 3 for each physiological response served as a measure of habituation to stressor stimuli. This measure was calculated by subtracting the mean of each physiological value of minute 1 from minute 3 and then dividing by minute 3 (minute 3 - minute 1 / minute 3). Comparisons of minute 1 to minute 3 habituation averages for each group were in the direction of decreasing arousal suggesting habituation to each of the experimental conditions. However, the only minute 1 to minute 3 change approaching statistical significance was EMG during the relaxation task, \( F(1,16 = 3.20), p = .089 \), where the PD group demonstrated no change in EMG on this task while the control group demonstrated a 14% reduction. A comparison of habituation across all physiological measures for the PD and control groups revealed no statistically significant differences in habituation across the two groups.

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Habituation to the experimental stressors across sessions was calculated by comparing average physiological levels during session 1 with average levels during session 3 for each physiological measure. Percentage change comparisons of each physiological variable and experimental task from session 1 to session 3 served as an across-session measure of habituation i.e., (session 3 - session 1 / session 3). These comparisons were analyzed in two ways. First, the average degree of habituation for each measure per task per group were compared against the hypothesis of no change across sessions using a one sample t-test. This provided an evaluation of whether rates of sensitization or habituation were significantly different from no change. This resulted in 20 analyses per group (4 physiological measures x 5 conditions). Second, one way ANOVAs were calculated comparing the two group means on each variable and task.

Some evidence of habituation occurred for the PD group on 7 of the 20 analyses i.e., lower physiological levels on session 3 than session 1. More specifically, analyses of the PD group revealed habituation on BVP across each of the 5 experimental conditions and on EMG across the mental arithmetic and cold pressor conditions. Only BVP during relaxation differed significantly from no change across sessions (M = .95), t(8) = 2.73, p = .0257. On this task, the PD group had a substantial increase (95.2%) in blood flow from session 1 to session 3. The control group showed habituation on 9 of the 20 measures. Decreases in physiological arousal from session 1 to session 3 occurred on BVP and EDG across each of the stressor tasks i.e., role play, mental arithmetic, and cold pressor. EDG habituation
also occurred across baselines and EMG habituation was evident across the baseline and mental arithmetic conditions. Several of these comparisons reached statistical significance. The control group habituated significantly from no change in BVP during baseline \((M = .109), t(8) = 3.00, p = .017;\) mental arithmetic \((M = .490), t(8) = 2.30, p = .049;\) and cold pressor \((M = .769), t(8) = 3.01, p = .016.\) The control subjects also showed significant habituation in EDG during mental arithmetic \((M = -.31), t(8) = -5.38, p = .003;\) and cold pressor \((M = -.16), t(8) = -2.55, p = .033.\)

The second method of analyzing habituation patterns revealed habituation differences on EDG and BVP between the two groups from session 1 to session 3. Figure 3 displays the group comparisons of EDG percentage change across each of the experimental conditions. Sensitization occurred among the PD group and habituation within the control group across each condition with these differences being significant during the role play \(F(1,16 = 4.729), p = .045\) and mental arithmetic \(F(1,16 = 10.179), p = .005.\) Additionally, the PD group evidenced greater increases in BVP during the relaxation task \(F(1,16 = 8.174), p = .011\) than the control group.

Physiological Response Awareness and Arousal

Heart rate and muscle tension change estimates for each subject were compared against the direction of actual physiological change for each task and session to assess the subjects' ability to accurately estimate physiological changes. A correct estimate of change was defined by the subject reporting increases or decreases in HR or EMG...
Figure 3. EDG Across-session Habituation/Sensitization Across Each Experimental Condition for Panic and Normal Control Subjects.
that matched the direction of actual physiological change compared to the previous baseline during that task. For example, a subject reporting increases in their heart rate across each of the 4 experimental tasks when the actual physiological measures for the tasks consisted of two increases and two decreases would result in a 50% accuracy rating. For each physiological measure, 108 estimates were obtained from each group across all tasks and sessions and used to calculate group averages. The PD group was correct across 55% and 54% of the tasks, respectively, for reporting EMG and HR changes versus 72% and 59% for the control subjects. Differences in accuracy between the 2 groups did not reach statistical significance.

In a finer-grained analysis of the inaccuracy of these estimates of physical change, it was found that of the PD groups' errors, 79.2% were errors in reporting EMG increases when actual decreases occurred. This compared with 56% for the control group. The majority of these errors occurred during the relaxation task, where out of a total of 27 EMG estimates, the PD group committed 11 errors of reporting increases in EMG when the actual reductions ranged from 46% to 65%. Three of the 8 errors made by the control group were similarly inaccurate.

Session Symptom Reports

Average panic symptom reports per experimental phase were determined for each subject and averaged across groups. These values were then compared for each task by one way analysis of variance and Wilcoxon nonparametric measures. Group means are displayed in Table 2. The panic group reported more dizziness, feelings of unreality and

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<table>
<thead>
<tr>
<th>Symptom</th>
<th>Roleplay PD</th>
<th>Roleplay C</th>
<th>Relaxation PD</th>
<th>Relaxation C</th>
<th>Arithmetic PD</th>
<th>Arithmetic C</th>
<th>Cold Pressor PD</th>
<th>Cold Pressor C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain</td>
<td>2.3</td>
<td>1.0</td>
<td>1.3 * 0.0</td>
<td>2.8</td>
<td>1.4</td>
<td>2.0</td>
<td>1.2</td>
<td></td>
</tr>
<tr>
<td>Dyspnea</td>
<td>1.7</td>
<td>1.2</td>
<td>1.7 * 0.0</td>
<td>2.8</td>
<td>0.9</td>
<td>3.0</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Palpitations</td>
<td>5.1</td>
<td>5.0</td>
<td>2.6 0.7</td>
<td>3.3</td>
<td>2.8</td>
<td>2.6</td>
<td>2.1</td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>0.6</td>
<td>0.1</td>
<td>1.3 0.0</td>
<td>0.3</td>
<td>0.0</td>
<td>1.6</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>Fear of dying</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0 0.0</td>
<td>0.1</td>
<td>0.0</td>
<td>0.3</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td>2.2 * 0.1</td>
<td>1.6 0.2</td>
<td>2.2 0.6</td>
<td>1.7</td>
<td>0.1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trembling</td>
<td>2.2</td>
<td>1.3</td>
<td>0.8 0.0</td>
<td>1.6 * 0.0</td>
<td>1.2</td>
<td>0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feelings of unreality</td>
<td>1.1</td>
<td>0.1</td>
<td>2.4 * 0.3</td>
<td>1.3</td>
<td>0.6</td>
<td>1.0</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>Choking</td>
<td>1.0</td>
<td>0.0</td>
<td>1.4 0.0</td>
<td>1.4 * 0.0</td>
<td>1.3</td>
<td>0.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Faintness</td>
<td>0.3</td>
<td>0.0</td>
<td>0.2 0.0</td>
<td>1.1</td>
<td>0.0</td>
<td>1.8</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Going crazy/uncontrolled</td>
<td>0.2</td>
<td>0.0</td>
<td>0.2 0.0</td>
<td>1.0 * 0.0</td>
<td>0.8</td>
<td>0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paresthesias</td>
<td>1.4</td>
<td>0.6</td>
<td>0.3 0.4</td>
<td>0.6</td>
<td>0.3</td>
<td>1.4</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>Hot or cold flashes</td>
<td>3.7</td>
<td>1.3</td>
<td>0.7 0.2</td>
<td>2.0</td>
<td>1.7</td>
<td>3.8</td>
<td>1.8</td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td>2.4</td>
<td>1.7</td>
<td>0.1 0.0</td>
<td>1.7</td>
<td>1.6</td>
<td>4.1 * 0.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. * ANOVA/Wilcoxon-Mann p < .05

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choking during the role play condition although only dizziness reached significance $F(1, 16 = 7.56), p = .014$. During the relaxation task, the panic group reported greater chest pain $Z = -2.12, p = .033$, and dyspnea $Z = -2.12, p = .033$. The mental arithmetic condition resulted in a higher number of panic group reports of trembling $Z = -2.46, p = .013$, choking $Z = -2.80, p = .005$, and feelings of going crazy or doing something uncontrolled $Z = -2.12, p = .033$. The panic groups' symptom reports across the cold pressor task were significantly higher for sweating $Z = -2.46, p = .011$. 

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DISCUSSION

The present study assessed psychological and physiological responding in PD and normal control subjects across daily diary recordings and five laboratory conditions. Consistent with several of our hypotheses, PD subjects when compared to a normal control group have higher baseline and relaxation task EDG and BVP levels, demonstrate less EDG habituation across experimental sessions, and report greater numbers of panic symptoms and psychological distress. The results of the study do not support the predictions that the PD subjects demonstrate increased reactivity to the experimental tasks and are more accurate in detecting actual EMG and HR changes relative to normal controls.

One obvious finding is that PD subjects differed significantly from the normal control group on nearly every self-report measure. These results are consistent with existing literature (e.g., Barlow et al., 1984). The results of these pre-laboratory assessments support the hypothesis that PD subjects endorse more numerous and severe physical and psychological complaints than normal controls. This is most evident by higher scores on the SCL-90-R and by more than double the number of symptom reports during the laboratory sessions by the PD group. Moreover, these subjects report a moderate amount of daily anxiety as indicated by their average daily diary rating of 3.74 on the 0-8 subjective anxiety rating scale, an anxiety level that was significantly higher than the control group's 1.3.
average. Although the PD group reported greater numbers of panic symptoms during the laboratory sessions, this trend was inconsistent across the tasks. The PD group reported a significantly greater number of panic symptoms relative to their controls (e.g., chest pain, dyspnea, dizziness, trembling, feelings of unreality and going crazy, choking, and sweating), but none of these occurred uniformly across the tasks.

A particularly salient difference was observed in two of the physiological measures, EDG and BVP, in comparisons of the two groups on their absolute physiological levels. Panic disorder subjects were significantly more aroused as measured by EDG and BVP than the normal control subjects at baseline and during the relaxation conditions and on BVP measures during the role play tasks. The PD group also had higher "resting" levels of EMG than the control but this difference was not maintained across each of the other four experimental phases. These results are consistent with other studies which have reported baseline physiological differences between PD and/or agoraphobic subjects. The physiological differences observed in this study occurred in EDG and BVP in contrast to elevated heart rate and/or electromyographic activity differences previously reported (e.g., Barlow et al., 1984; Freedman, et al., 1984; Liebowitz et al., 1984; 1985). In each of these studies, though, EDG and BVP were not assessed, and the HR and EMG differences either failed to reach statistical significance or were difficult to interpret based on relatively short sampling intervals. This study's results are similar with Freedman et al. (1985) who found no differences between patients
with PD and control subjects on ambulatory measures of heart rate and ambient skin temperature. The significantly elevated initial baseline and relaxation condition values suggests that the non-significant differences between the two groups across the experimental tasks represent a contamination of the basal elevations. Panic Disorder subjects compared to normal controls come into the sessions at higher levels of arousal and may then be less likely to exhibit differentially greater physiological arousal across the laboratory tasks.

Percentage change from baseline was used as a method to control for the initial physiological values that may confound interpretation of differences between these subjects. This method provided a "relative" measure of change for each subject. Both groups exhibited reactivity across all physiological measures and stressor tasks and reductions during the relaxation task. The two groups, however, did not differ remarkably in their degree of arousal to the stressor tasks. In fact, the PD group exhibited less reactivity than the control group. The panic subjects, despite having higher EDG and BVP absolute values as a group, were not more reactive than their normal controls to the stressor tasks in this experiment. This study supports observations that HR reactivity does not differ significantly between panic subjects and normal controls (Ehlers et al. 1986; and Freedman et al. 1984) and contrasts with significant blood pressure reactivity differences found in two studies of panic patients and normal controls (Ehlers et al., 1986; Liebowitz et al., 1984). Perhaps these differences can be explained in terms of different regulatory functions during activation of the autonomic nervous

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system. Although this measure was not studied in this experiment, blood pressure's relationship to BVP would suggest that these measures of vasomotor activity are the more valuable discriminator variables. It appears that PD subjects are more aroused in absolute terms but they do not exhibit greater physiological increases during stressor tasks than normal subjects. This is not consistent with the positive feedback loop theory (Beck & Emery, 1985; Clark, et al., 1985; Evans, 1972; Lader & Mathews, 1968; Razran, 1961) that posits greater physiological reactivity and diminished habituation among panic sufferers.

In addition to the lack of significant differences in reactivity, within-session task analysis of habituation as measured by percentage change from minute 1 to minute 3 to the stressor tasks also revealed no differences between the two groups. Both groups evidenced decreases in their arousal during each task, but the group differences were not significant. If panic patients are more reactive and do not exhibit habituation to stressful events as has been hypothesized by (e.g., Beck & Emery, 1979; & Clark, 1986; Lader & Mathews, 1968) it was not observed in this study. It may be that this hypothesis is true only for extreme degrees and durations of induced anxiety. Despite the fact that this research involved only moderately anxiety-provoking stimuli, this theory maintains that even subtle physical changes, as evoked in this experiment, would result in similar patterns of arousal without subsequent habituation. Such a pattern was not observed across any physiological variable. Although physiological changes readily return to baseline levels, more subjective
estimates of anxiety and anxiety-related cognitive self-statements may not. While this study did not assess this possibility, the function of these components has been frequently identified as the mediating mechanism in panic attacks (e.g., Beck, Emery, & Greenberg, 1985; Clark, 1986; Mathews, Gelder, & Johnston, 1981).

Habituation measures obtained across assessment sessions did reveal differences between the two groups. PD subjects demonstrated no declines in their EDG arousal from session 1 to session 3 across any task. By contrast, the control group evidenced declines in EDG across sessions in each of the three stressor tasks. In addition to the lack of decline in arousal among the PD group, it was observed that EDG sensitization occurred among the PD subjects compared to habituation in the controls during the two performance tasks: role play and mental arithmetic. These data suggest that PD subjects are more aroused and that this arousal is maintained across repeated exposures to stressful tasks. Such a chronic state of EDG and HR arousal was also observed in agoraphobics with panic attacks (Roth et al., 1986).

Several interpretations of the non-significant differences in reactivity and within-session habituation are possible. This study employed a small number of subjects, and dramatic variability (some subjects of each group approached panic levels of heart rate) was observed across each group which confounds the generalization of our results. As previously mentioned, the experimental tasks used may not be sufficiently intense enough to produce physiological reactions that would serve as physiological markers for panic. This was evident...
in both groups who rated the various tasks mild to moderately anxiety-provoking. Each task with the exception of the relaxation condition also lasted less than 3 minutes. It is possible that a more sustained distressing event coupled with the elevated baseline level of arousal is necessary to produce the proposed effects that can lead to panic anxiety. The personally relevant role play task appeared to approximate such arousal. A ceiling effect may be responsible for the results as well. Panic subjects come into the session at a higher arousal level than the controls and are therefore less likely to demonstrate greater changes. This is consistent with the finding that these subjects demonstrated smaller percentage changes across the two most arousing tasks, the role play and mental arithmetic, while having larger percent reductions during the relaxation task.

It also appears that while within-session measures of habituation and reactivity are not indicative of group differences, the repeated exposures to the experimental stressors results in sensitization among PD subjects versus habituation in the controls. In addition to the higher EDG and BVP at baseline, the lack of across-session habituation seems to indicate a chronic pattern of autonomic arousal in panic sufferers.

The hypothesis that panic patients are more sensitive to (or at least able to report) subtle physiological changes (Chambless, 1982; Chambless & Goldstein, 1981), particularly cardiovascular changes, is also not supported by this study. Neither the panic nor control group were accurate in reporting changes much beyond what would be expected by chance alone. This may be accounted for by the mildly stressful
conditions of the experiment and the relatively modest EMG and HR reactions to these stressors. For example, the heart rate changes were generally 10% to 15% which may be too small to be discriminated or accurately reported. It was found that although both groups were inaccurate in their estimates of change, the PD subjects' errors occurred in overestimating of actual changes, particularly in EMG.

There may be a subgroup of panic patients that "misinterpret" or make poor discriminations of their physiological functioning. Several of the panic subjects reported dramatic increases in HR and EMG when either no change or reductions in arousal actually occurred. Some support for the role of such misperception of physiological events in evoking panic has been presented by Ehlers et al. (1988). These researchers found that panic patients who were given false heart rate feedback demonstrated increases in subjective anxiety and physiological arousal. In the present study, it was further observed that although as a group the panic patients did not rate the stressor tasks as more stressful or anxiety-provoking than the control group, they did report far more panic symptoms. It appears that they are focused upon and prone to report symptoms that in the past have been identified with panic anxiety regardless of their current level of arousal. Such "report" of panic symptoms can be independent of any distinctive, identifiable physiological correlate. It has long been known that dysynchrony exists across verbal, behavioral, and physiological response dimensions. At least in some cases, it seems that panic patients disproportionately focus on and overreport bodily changes. Panic disorder may then be interpreted as a behavioral problem.
consisting of maladaptive behavior involving the person's verbal report, what he or she does, how he or she reacts physiologically to environmental events, or a combination of these, all of which are explained by his idiosyncratic learning history.

This research suggests that some form of arousal reduction strategy (e.g., biofeedback training) paired with altering anxiety associated self-statements would benefit many panic sufferers. It was found in this study that PD subjects exhibited significantly higher levels of arousal and less habituation across sessions as measured by EDG and BVP. A biofeedback-assisted relaxation training employing EDG or BVP feedback appears to be a logical approach instead of the more common relaxation treatments focused on muscle tension reduction.

Given the wide variability and modest differences in arousal patterns among these subjects, it also seems valuable to assess individuals across each response system and physiological measure to design the most effective treatment as has been suggested by Barlow and Cerny (1988). Assessments would need to identify the predominate response systems involved within each individual. This would involve identifying abnormal self-statements or misperceptions such as those we observed in association with physiological changes. Physiological assessments seem appropriate for a portion of this population who clearly demonstrate high levels of arousal particularly across skin conductance and blood flow measures. Personalized role play tasks employed in this study provided a fruitful method for measuring both of these cognitive and physiological factors implicated in panic. Behavioral factors although not addressed in this study may be
valuable measures to be assessed as well for designing effective
treatment interventions. Given the length of time that many PD
subjects have experienced panic it seems likely that environmental
consequences for the symptomatology may play a role in maintaining PD.
For example, panic-related behaviors may be negatively reinforced as a
socially acceptable way to avoid responsibilities or unpleasant
events. Alternately, the symptoms may be positively reinforced by
evoking concern from significant others and health care providers.

Large individual differences in each of the physiological and
self-report measures across subjects in this study suggests that a
variety of factors are involved in panic disorder. In order to make
more definitive statements regarding the etiology and maintenance of
symptoms for individual panic sufferers, research utilizing a func­tional analytic approach that identifies antecedent events and
response consequences for the range of behavioral, physiological, and
cognitive symptoms reported by individual panic sufferers would
provide a valuable addition to the current conceptualization of panic
disorder.


Psychiatry, 42, 244-248.


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Razran, G. (1961). The observable unconscious and the inferable


