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**Influence of personality on learning and related processes among
nonclinical panickers**

Richman, Harvey, Ph.D.

The University of North Carolina at Greensboro, 1994

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300 N. Zeeb Rd.
Ann Arbor, MI 48106

**INFLUENCE OF PERSONALITY ON LEARNING AND RELATED
PROCESSES AMONG NONCLINICAL PANICKERS**


by

Harvey Richman

A Doctoral Dissertation Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the requirements for the Degree
Doctor of Philosophy

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Approved by



Rosemary O. Nelson-Gray, Ph.D.

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This study examined hypothesized personality-related individual differences in learning and related processes among four groups of participants who differed in presence vs. absence of nonclinical panic attacks and in high vs. low self-reported agoraphobic avoidance. Variables of interest were (a) level of audible stimuli tolerated, (b) magnitude of skin resistance (SR) responding to audible stimuli, (c) rate of habituation to an audible stimulus, and (d) evidence of conditioning of a neutral stimulus to an aversive audible stimulus.

The four groups in this study were found to occupy different locations in Eysenckian two dimensional personality space as predicted. Nonclinical panickers high on agoraphobic avoidance were highest on neuroticism and trait anxiety while nonpanickers low on agoraphobic avoidance were lowest on neuroticism and trait anxiety. The former group was lower than the latter on extroversion. Nonclinical panickers low on avoidance and nonpanickers high on avoidance were intermediate on these three measures.

No notable group differences were found in sensitivity to audible stimuli. High avoidant panickers evidenced the largest skin resistance (SR) responses while low avoidant nonpanickers evidenced the smallest responses. Low avoidance panickers and high avoidant nonpanickers were intermediate. A measure of habituation yielded statistically significant group differences in the predicted

direction, with high avoidant panickers habituating to an audible stimulus more slowly than low avoidant or high avoidant nonpanickers. No statistically significant group differences emerged for the index of "conditionability." However, group means on this measure were higher for high avoidant panickers and high avoidant nonpanickers than for the low avoidant panickers and low avoidant nonpanickers.

Stimulus sensitivity did not appear to be related to the personality variables of interest. Greater response magnitude and slowed habituation appeared to be positively related to trait anxiety and neuroticism. No strong relationships were evidenced between the index of conditionability and the personality variables being investigated

In general, the results suggest that the presence of nonclinical panic attacks and increased self-reported agoraphobic avoidance are predictive of one's location along an "anxiety continuum" in Eysenckian personality space. Those physiological measures on which notable group differences emerged (i.e., SR response magnitude and rate of habituation) appeared to be related to neuroticism and trait anxiety. Presence of panic attacks and high self-reported agoraphobic avoidance (particularly in combination) predicted high SR responsivity and slowed habituation, suggesting a role for these variables in the development and maintenance of panic attacks and agoraphobic avoidance. The results support the continued study of panic and agoraphobic avoidance in nonclinical populations.

APPROVAL PAGE

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Overview

This research arose from the assumption that certain neurological, autonomic, and behavioral differences exist among individuals and that these differences may influence what we commonly refer to as personality. Two noted personality theorists, H. J. Eysenck (1967) and Jeffrey A. Gray (1971, 1972) have suggested that these individual differences derive from a combination of genotypic and phenotypic influences and manifest themselves in observable personality differences and, in some cases, in psychopathology. It is suggested by these theorists that individual differences in "learning" or "conditionability," and in other processes (e.g., sensitivity to stimuli, autonomic responsivity, and rate of habituation to stimuli), play a role in accounting for individual differences in personality and behavior. In the present research, the hypotheses which follow from the theorizing of Eysenck and Gray were applied to panic and agoraphobic tendency, two phenomena in which learning or conditioning, and other

physiological variables, are assumed to play an important role.

Panic Disorder

Panic and Agoraphobia

The phenomenon of panic as a clinical entity came into being with publication of the third edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-III) in 1980 (American Psychiatric Association, 1980). Before considering the panic-related disorders as conceptualized in DSM-III-Revised (American Psychiatric Association, 1987), an introduction to another term is necessary. The word "agoraphobia" is derived from the Greek words agora (meaning marketplace) and phobos (meaning fear). Thus, agoraphobia literally translated means fear of the marketplace (Marks, 1969). The syndrome encompasses a complex, and at times paradoxical, spectrum of symptoms including intense and unexplainable fear or panic which occurs upon exposure to certain places or situations, an increasing pattern of phobic avoidance of these places or situations, and a tendency for these fear reactions to generalize to other similar places and situations. Common agoraphobic situations include being outside the home alone, being in a crowd or standing in a line, being on a bridge, and traveling in a bus, train, or car (American Psychiatric Association, 1987). Agoraphobic avoidance is discussed in greater detail in subsequent

sections.

DSM-III-R criteria for panic attacks and panic disorder are listed in Appendix A. DSM-III-R allows for the diagnosis of panic disorder with and without agoraphobia as well as agoraphobia "without history of panic disorder." A diagnosis of panic disorder with agoraphobia involves meeting the additional DSM-III-R criteria that the person evidence a pattern of avoiding identifiable situations, such as those described above, because exposure to the situations elicits severe anxiety and panic symptoms. An individual who experiences severe discomfort and anxiety in these situations, but does not actually avoid the situations, also meets criteria for diagnosis of panic disorder "with agoraphobia."

Prevalence, Onset, and Course

DSM-III-R suggests that panic disorder is common, with panic disorder with agoraphobia being found more often than panic disorder without agoraphobia in clinical settings. Females and males are equally likely to receive a diagnosis of panic disorder without agoraphobia. However, females are approximately twice as likely as males to receive a diagnosis of panic disorder with agoraphobia (American Psychiatric Association, 1987). DSM-III-R suggests that the average age of onset for panic disorder is in the late twenties. Marks and Lader (1973) suggest that age of onset for the great majority of anxiety states is between sixteen

and forty five. Panic disorder may run its course in a few weeks or months. However, this is rare. Panic disorder can last for years or a lifetime with alternating periods of remission and exacerbation of symptoms.

Factors Associated with Panic

Donald Klein, David Sheehan, and other supporters of the "medical model" of panic suggest that panic is related to, and in fact derives from, an innate or inborn biochemical dysfunction. Evidence for a direct biological influence in the development of panic and agoraphobia derive from findings in three areas. These are: (a) treatment outcome studies involving the differential effects of two classes of anti-anxiety medications (benzodiazepine tranquilizers such as Tranxene and tricyclic antidepressants such as Norpramine) on panic and generalized anxiety states (Klein, 1964; Klein, 1969; Klein et al., 1978; Klein, 1981); (b) studies involving the experimental induction of panic in anxious subjects (e.g., via injections of sodium lactate such as was done by Pitts & McClure, 1967) suggesting an abnormality in lactate metabolism; and (c) findings which suggest that a genetic predisposition for panic disorder is transmitted within families. Torgerson (1983), for example, reported findings which supported the specific transmission of panic disorder. Other researchers (Leckman et al., 1983; Raskin et al., 1982) have provided results suggesting that relatives of panic disorder patients were at increased risk

for several types of psychopathology including depression and alcoholism. Jaradine et al. (1984) and Kendler et al. (1987) suggested that some individuals have a non-specific predisposition for all anxiety and depressive disorders.

Several medical disorders produce symptoms similar to those observed during panic attacks and appear to have a greater than chance association with panic attacks. These include mitral valve prolapse syndrome (MVP), a usually benign condition which causes a fluttering sensation in the heart area. Evidence suggests that the association between panic and MVP may be due in part to the tendency of panickers to be more aware of, and more concerned with, bodily sensations than nonpanickers and to be more likely to seek medical attention for MVP symptoms than nonpanickers.

Hyperventilation syndrome is a condition in which an anxious individual's shallow rapid breathing leads to biological changes (e.g., autonomic arousal) which mimic panic symptoms (Ley, 1987). Because hyperventilation appears to be both a potentiator of panic and a consequence of panic, it has a prominent role in the phenomenology of panic. Ronald Ley's (1985a) suggestion of a "hyperventilation theory of panic disorder" underlines the important role of hyperventilation in the genesis of panic disorder. Anxiety-related hyperventilation syndrome might well provide our best answer to the difficult question of what initiates the first unexpected panic attack.

Hypoglycemia symptoms can mimic panic attacks. However, blood sugar levels taken during an attack clearly identify the problem and ingesting food will relieve the symptoms. Paroxysmal tachycardia produces panic-like symptoms but can be identified via electrocardiogram. Inner ear problems, which result in balance difficulties, have been associated with panic patients. However, no unequivocal evidence of a relationship between these syndromes and panic disorder has surfaced. As with mitral valve prolapse, it may be the heightened awareness of bodily sensations, and a tendency to interpret these sensations as dangerous, that accounts for the apparent association of these conditions with panic disorder.

While there are many biological correlates of panic and agoraphobia, the exact relationships between these variables and the occurrence of panic and agoraphobia are at present not fully understood.

Current writings on panic disorder suggest that "cognitions" play an important role in the development and maintenance of panic attacks. Cognitive theory of panic focuses on how individuals construe signals originating from within the body (Beck, 1988). Panickers fear that some vital body part (e.g., the heart or lungs) will stop functioning or that their mind will become dysfunctional. Beck suggests that because correct functioning of body and mind are crucial to immediate survival, it is not surprising

that vulnerable individuals will be hypersensitive to any indication of malfunction in these area. Beck suggests that there may be a neurophysiological/cognitive vulnerability in some individuals which is manifested in varying combinations of increased physiological arousal, a tendency to misinterpret somatic sensations, and an inability to reappraise events. Beck, in this 1988 writing, does not specify particular neurological substrates for this vulnerability.

The term "cognitive" has been used by the present author not because it is preferred or indisputably correct. It has been used because it is pervasive in the panic disorder literature and in the communications of applied psychologists and those in related professions. However, radical behaviorists view "cognitions" or "thoughts" as covert verbal behavior (the result of the individual's learning history) and afford these behaviors no unique causal status. See Skinner (1953,1980) and Catania and Harnad (1988) for a discussion of these issues.

Learning Theory and Panic

Behavioral approaches to panic and agoraphobia are based on the principles of classical and/or operant conditioning, though the specific roles of each type of learning are still debated (Davey, 1981). Classical conditioning approaches suggest that a number of interoceptive stimuli, such as pain (usually considered an

unconditioned stimulus or UCS) or unique situations, can come to elicit an anxiety response (Clum & Pickett, 1984). See Campbell, Sanderson, and Laverty (1964) for an excellent example of how a conditioned emotional fear response (CER) can be conditioned in one trial. Mowrer (1960) suggests that fear (conditioned emotional) responses or CERs are acquired via classical conditioning. In panic disorder, stimuli which have been associated with the initial panic attack via temporal contiguity become capable of eliciting subsequent panic attacks. The "stimulus complex" which triggers subsequent panic attacks may include (a) bodily sensations such as sweating, (b) the situation in which the previous attack occurred such as a shopping mall, and (c) cognitions or "covert verbal behaviors." Any part or combination of parts of the stimulus complex (CS) may trigger a subsequent attack. The CS acts to elicit numerous responses. These may include physiological responses (e.g., increased heart rate), behavioral responses (e.g., escape seeking), and cognitive responses or "private events" (e.g., I must be having a heart attack). These responses then serve as stimuli for further responses and the "vicious cycle" of a panic attack ensues. In the case of the initial attack, the UCS might be some type of bodily sensation (interoceptive stimulus) with the UCR being the fear responses already discussed. Hyperventilation, of which the individual is unaware, may produce many symptoms which mimic

panic symptoms and may be the starting point for an initial attack in many individuals.

Personality and Panic

The relationship between panic disorder/agoraphobia and the DSM-III-R personality disorders has been receiving increasing attention in recent years. Tyrer et al. (1983) found, in a mixed sample of 316 anxiety and depressive neurotics, that anxiety neurotics were more likely than depressive neurotics to have a coexisting personality disorder, with 48% of the anxiety neurotics, 45% of phobic neurotics, and 30% of depressive neurotics, respectively, evidencing a personality disorder. Many recent investigations approach the relationship between panic/agoraphobia and personality within the context of the DSM-III-R personality disorders. These personality disorders involve enduring patterns of relating to the environment and to oneself which are inflexible, maladaptive, and which result in significant impairment in functioning or in subjective distress. DSM-III-R recognizes 11 personality disorders which are grouped into three "clusters" (Appendix B). Cluster A (odd-eccentric) is comprised of paranoid, schizoid, and schizotypal personality disorders. Cluster B (erratic-dramatic) includes borderline, antisocial, histrionic, and narcissistic personality disorders. Cluster C (anxious-fearful) includes dependent, avoidant, passive-aggressive, and obsessive-

compulsive personality disorders (American Psychiatric Association, 1987). Friedman, Francis, and Shear (1987) reported that in a small sample of clinical panickers personality disorder symptomatology from the DSM-III-R anxious-fearful cluster (dependent, avoidant, passive aggressive, and obsessive compulsive) was particularly prevalent. Mauri, Sarno, Rossi, Armani, Zambotto, Cassano, and Akiskal (1992), using the Personality Disorder Examination, found high occurrences of Cluster C personality disorders (especially avoidant, dependent, and passive aggressive) in a sample of 40 panic disorder patients. Additionally, these researchers found a high prevalence of borderline personality disorder in this sample. Mellman, Gabriele, Leverich, Hauser, Kramlinger, Post, and Uhde (1992), using the SID-P interview (Stangle et al., 1985), also reported high occurrences of Cluster C personality disorder in 23 panic disorder patients. Green and Curtis (1988), using the SCID-II interview, reported that avoidant personality disorder was the most often diagnosed personality disorder in a sample of 25 panic disorder sufferers. The present author (Richman & Nelson-Gray; 1991, 1992) has observed that nonclinical panickers (individuals who have experienced panic attacks but without the frequency to warrant a diagnosis of panic disorder) reliably reported more personality disorder symptomatology than nonpanicking controls. Notable were differences on avoidant, dependent,

schizotypal, borderline, passive-aggressive, and self-defeating symptomatology. The present author has also observed that nonclinical panickers score higher than nonpanickers on measures of neuroticism and introversion (Eysenck, 1957) and trait anxiety (Gray, 1970). Thus, it appears that there are reliable personality differences between individuals who experience panic attacks, clinical or nonclinical, and those who do not experience such attacks. An important point to be noted here is that those personality disorder symptomatology measures on which panickers and nonpanickers differed most (e.g., avoidant, borderline) had in common high positive correlations with measures of trait anxiety, neuroticism, and to a lesser extent introversion.

Agoraphobic Avoidance

The question of who will, and will not, develop severe agoraphobic avoidance has been receiving increasing attention. Agoraphobic avoidance commonly develops in conjunction with panic attacks; however, some individuals do develop agoraphobic avoidance without having experienced panic attacks. An individual who develops extensive agoraphobic avoidance may be so incapacitated that he or she is unable to hold a job or participate in family and social activities. The primary symptoms of agoraphobia are severe phobic anxiety and phobic avoidance across a variety of related situations. Common themes among these situations

appear to be (a) perceived potential for being trapped, (b) perceived potential of not being able to reach, or be reached by, help if it is needed, (c) situations in which momentary incapacitation would be disastrous (e.g., driving a car), and (d) perceived potential for social embarrassment. Some specific examples are leaving home alone, driving, using public transportation (e.g., trains), walking in shopping malls, waiting in lines, eating in restaurants, attending concerts or sporting events, going through tunnels, crossing bridges, being in high places, riding in elevators or on escalators, and being in very open spaces.

Dianne Chambless and associates at the Temple University School of Medicine Department of Psychiatry have developed a self report inventory designed to assess agoraphobic avoidance, both when the individual is alone and when he or she is accompanied by a "support person" (a person who the agoraphobic trusts and who will deal with a problem if one arises). The inventory assesses avoidance in 27 situations commonly avoided by agoraphobics. The Mobility Inventory for Agoraphobia (Chambless, Caputo, Gracely, & Williams, 1985) has been used widely in research and clinical practice. Avoidance is rated on a 5-point Likert scale ranging from "never avoid" to "always avoid." Among clinical agoraphobics, avoidance alone scores have been found to be significantly and reliably higher than

avoidance accompanied (by a spouse or other "support" person) scores. In addition to assessing agoraphobic avoidance both alone and accompanied, the inventory in its original form, also assessed agoraphobic "discomfort" (the individual experiences discomfort in situations but does not avoid them). Chambless et al. reported the discomfort alone and discomfort accompanied scales to be quite redundant with the avoidance alone and avoidance accompanied scales (with Pearson correlations ranging from +.87 to +.94). Hence, the discomfort scales are not usually used with clinical populations. The present author, and others, have used this inventory in studying agoraphobic avoidance among college populations (nonclinical panickers). In the adaptation used by the present author, the avoidance accompanied scale has been omitted as it is unlikely that college undergraduates have a significant support person to accompany them. The discomfort scale has been retained because the discomfort and avoidance scales do not appear to correlate as highly when used with nonclinical panickers as when used in clinical populations. The present author has observed Pearson correlations between the discomfort and avoidance scales to be more on the order of +.75 in samples of college undergraduates. College undergraduates who experience panic attacks consistently score higher than nonpanicking controls on both the discomfort and avoidance scales.

Factors Associated with Agoraphobic Avoidance

Clum and Knowles (1991) reviewed a considerable number of articles and concluded that frequency of panic attacks seemed to be consistently associated with increased agoraphobic avoidance.

Cognitive aspects of panic and agoraphobic avoidance are currently being given extensive attention (e.g., Rachman & Maser, 1988). Noyes et al. (1987) found catastrophic cognitions to be positively related to agoraphobic avoidance. As previously noted, the term "cognitions" is used here because of its frequent usage in the panic and agoraphobia literatures, not because the present author considers it to be particularly accurate or descriptive. Craske and Barlow (1988) pointed out the importance of anticipation of panic attacks in contributing to development of avoidance.

Based on a review of twelve studies, Clum and Knowles (1991) concluded that while females are only slightly more likely than males to have panic disorder without agoraphobia, females are significantly more likely to have panic disorder with agoraphobia and that this may be more related to gender identity than to gender per se.

Several developmental factors have been implicated in the development of agoraphobic avoidance. A history of significant maternal overprotection among agoraphobics has been reported by Terhune (1961) and others. Bowlby (1973)

suggested that a lack of maternal affection may predispose toward agoraphobia. According to Tearnan and Telch (1984), several studies provide convergent evidence for a history of poor family relations in agoraphobics. Chambless and Goldstein (1981) suggest that in such families children are punished and criticized for, or denied the opportunity to practice, independent behaviors. As a result, they may grow up viewing themselves as incompetent and unable to cope in many situations. Some researchers have suggested modeling as a component in the development of agoraphobia. Hagman (1932) found that fears reported by phobic mothers and their children correlated $+0.67$. Separation anxiety or its symptoms have been suggested by several researchers. Liebowitz and Klein (1979) reported that 20 percent of outpatient and 50 percent of adult inpatient agoraphobics had a history of separation anxiety usually manifested as difficulty in attending school.

Positive relationships between depression and agoraphobic avoidance have been reported by Barlow (1986) and several others. Clum and Knowles (1991) suggest that depression may influence development of avoidance by reducing the level of energy available for coping with panic attacks.

Generalized anxiety disorder also often co-occurs with panic and agoraphobia (Aronson & Logue, 1987; Cloninger, Martin, & Clayton, 1981). I have not noted any

investigations into the role of anxiety itself as a possible mediating variable in the development of agoraphobic avoidance. This possible specific role of anxiety may have been overlooked because the universal acceptance of a strong general association between trait anxiety and panic disorder/agoraphobia.

Learning Theory and Agoraphobic Avoidance

During the 1940s, researchers were discovering that animals such as rats and dogs could learn to make a response to a particular stimulus which would allow them to avoid shocks. Two factor theory, described by O.H. Mowrer and others, integrates Pavlovian and operant elements into a coherent theory of "avoidance" (Levis, 1989). It is important to note that Mowrer, and others, felt the term "avoidance" to be misleading and actually preferred the term "escape" in describing these phenomena (Levis, 1989). In traditional learning theory, the organism is not seen as "anticipating or expecting" and avoiding the UCS (e.g., shock). The organism makes a response which leads to "escape" of the CS (e.g., light) which has acquired its own aversive properties, not avoidance of the UCS (shock). Thus, for Mowrer, the organism learns to make a response which leads to escape of the CS and reduces the drive of fear.

In the case of agoraphobic avoidance, the UCS might be physiological sensations resulting from a spontaneous panic

attack (perhaps due to hyperventilation). The CS might be a department store (a stimulus associated with the physical sensations via temporal contiguity). The department store (CS) can now elicit autonomic arousal or CERs on its own. The agoraphobic flees the department store and in so doing escapes from the CS (the department store). The probability that avoidance will again occur is increased via reinforcement related to reduction of the fear "drive". With regard to the first avoidance response, the agoraphobic in a department store is no doubt extremely activated during a panic attack. Avoidance or escape would appear to be the most socially safe and acceptable form the activation could take.

Personality and Agoraphobic Avoidance

With regard to the DSM-III-R personality disorders already discussed, it appears that the types of personality disorder symptomatology which seem to be associated with agoraphobic avoidance are in general, the same types which appear to be associated with the occurrence of panic attacks (i.e. avoidant, dependent, passive aggressive, borderline). However, avoidant and dependent personality disorders may be most closely associated with agoraphobic avoidance. In the Friedman, Frances, and Shear (1987) research, already discussed, it was reported that avoidant personality disorder symptomatology, and Cluster C symptomatology in general, tended to be positively associated with increased

self-reported agoraphobic avoidance. Mavissakalian and Hamann (1986) reported that avoidant and dependent personality disorder symptomatology were most common in a sample of agoraphobics with panic attacks (DSM-III). As noted previously, the present author has found in samples of both nonclinical panickers and nonpanicking controls that various types of personality disorder symptomatology (e.g., dependent, passive-aggressive, self-defeating, borderline, and avoidant) were positively related to self-reported agoraphobic discomfort/avoidance. Thus, the types of personality disorder symptomatology which distinguish individuals reporting high agoraphobic discomfort/avoidance from those reporting minimal agoraphobic discomfort/avoidance appear to be similar to the types of personality disorder symptomatology which are associated with the occurrence of panic attacks. Recall that among nonclinical panickers and controls, these types of personality disorder symptomatology were found to have in common high correlations with trait anxiety and neuroticism.

In addition to being viewed as an emotional state, anxiety may be conceptualized as a "dimension" or "trait" of personality (Gray, 1970, 1971). The present author has recently collected data on a sample of 194 nonclinical panickers and 94 nonpanicking controls. In both groups, there were notably high Pearson correlations between the anxiety subscale of the Minnesota Multiphasic Personality

Inventory and the Mobility Inventory for Agoraphobia avoidance scale ($r.= +.40$) and discomfort scale ($r.= +.45$). This suggests that high trait anxiety, viewed as occupying a location in a two-dimensional space (Eysenck, 1967), or as being a primary dimension of personality in this same two-dimensional space (Gray 1970, 1971), might be predictive of increased agoraphobic avoidance. Eysenck suggests that highly anxious individuals are high on his dimension of neuroticism and low on his dimension of extroversion (i.e., introverts) The present author has found that nonclinical panickers low on self-reported agoraphobic discomfort/avoidance tended to be less neurotic and less introverted than nonclinical panickers high on self-reported agoraphobic avoidance. Nonpanickers were lower still on neuroticism and introversion. Eysenck suggested that increased cortical arousal (characteristic of introversion) and increased autonomic reactivity (characteristic of high neuroticism) predispose individuals to the development of conditioned fears and phobias. This is because they produce more frequent and more intense autonomic reactions in response to stimulation and readily associate these responses with various environmental stimuli. Gray makes similar predictions with regard to learning or conditionability for individuals who are high on his dimension of anxiety. However, these predictions are somewhat more specific and are based on slightly different

theoretical grounds. Thus, some panickers, as a result of their personality and physiology, might develop greater agoraphobic avoidance because various stimuli (e.g., a shopping mall) condition more quickly and more strongly to the aversive experience of a panic attack than would be the case with an individual not so predisposed. Additionally, these individual differences may play a role in the occurrence of panic itself. The theorizing of Eysenck and Gray will be discussed in greater detail in later sections.

Personality, Physiology, and Behavior

Given the wide acceptance of learning processes as playing a role in the development and maintenance of panic and agoraphobic avoidance, and given that there are reliable personality differences between (a) panickers and nonpanickers and (b) those who evidence high and low levels of agoraphobic avoidance (both panickers and nonpanickers), it is somewhat surprising that more attention has not been paid to the role of individual differences in learning/conditionability and related variables (i.e., stimulus sensitivity, autonomic reactivity, rate of habituation) in the development and maintenance of panic and agoraphobia.

Hans Eysenck's Theory

Hans J. Eysenck, since the 1940s, has developed and refined a "two-dimensional" theory of personality based on numerous factor analytic studies (see Eysenck, 1970c).

Eysenck (1982) suggests that an interaction of genotype and experience determines individual differences. Eysenck's first dimension (usually plotted on the x axis) is introversion-extroversion. Eysenck suggests that a number of "traits" or "primary factors" correlate highly with or comprise the "superfactor" or "dimension" of extroversion. Thus, the extrovert is sociable, lively, active, assertive, sensation-seeking, carefree, dominant, surgent, and venturesome. The introvert would display traits opposite to these. Eysenck suggests that differences in cortical arousal account for individual differences in introversion-extroversion. Eysenck implicated the ascending reticular activating system as playing a role in determining individual differences in introversion-extroversion (Eysenck, 1967a). Eysenck suggests that individuals high in cortical arousal condition readily to all types of stimuli. Additionally, high cortical arousal is assumed to result in an "augmenting" of incoming stimuli so that a stimulus of a given intensity will be perceived as having greater intensity by an introverted (highly cortically aroused) individual than by an extrovert. The second dimension in Eysenck's two dimensional space, neuroticism, is usually plotted on the y axis. This dimension refers to a general emotional instability or lability. Neuroticism, is assumed to be closely associated with the sympathetic branch of the autonomic nervous system. Thus, individuals high on

neuroticism are assumed to be characterized by strong emotional (and autonomic) reactions to all classes of stimuli. Additionally, Eysenck suggests that the autonomic reactions of highly neurotic individuals will habituate slowly relative to the reactions of those individuals who are low on this dimension.

Thus, Eysenck suggests that individuals who are emotionally reactive (neurotic) and who condition easily (introverted) will tend to become overly socialized and will be predisposed to developing "dysthymic" or "neurotic" disorders, characterized by fears, phobias, obsessions, compulsions, and reactive depression (1982). Such individuals will have developed "surplus conditioned reactions" (Eysenck, 1982). The theorizing of Eysenck suggests that the development of these surplus conditioned reactions will be influenced by individual differences in (a) stimulus sensitivity, (b) autonomic reactivity, (c) habituation, and (d) conditionability. Eysenck also makes predictions with regard to individuals who are neurotic and "extroverted;" however, these predictions are not germane to the present discussion and are not discussed in detail here.

Jeffrey Gray's Theory

Jeffrey Gray (1970, 1972, 1973, 1981) has developed a theory which is based on, and is strongly related to, Eysenck's. Gray is in agreement with Eysenck with regard to there being a personality space defined by two independent

dimensions which are predictive of behavior and psychopathology. However, Gray argues that the two primary dimensions defining the space be labeled anxiety (reflecting activity of the behavioral inhibition system) and impulsivity (reflecting activity of the behavioral activation system). Gray suggests that the "behavioral inhibition system," involving the septo-hippocampal system, noradrenergic neurons in the locus coeruleus, and serotonergic neurons in the raphe nuclei underlies the dimension of anxiety. This system responds to signals of punishment and signals of nonreward. The behavioral effects of this system include behavioral inhibition (suppression of ongoing behavior), increased arousal, and increased attention. Thus, this system would appear to govern anxious or fearful behavior. Gray predicts that a consequence of having a highly active behavioral inhibition system will be increased conditionability to aversive stimuli (i.e., tendency to learn associations involving aversive stimuli). Gray suggested that the dimension of anxiety runs from Eysenck's quadrant bounded by low neuroticism and extroversion (lower right) upward through the quadrant bounded by high neuroticism and introversion (upper left). Gray's second dimension, impulsivity (reflecting activity of the behavioral activation system), lies at right angles to the dimension of anxiety. This is often referred to as a 45 degree rotation of Eysenck's dimensions; however, Gray

actually felt that the anxiety dimension should be located closer to the neuroticism dimension than to the extroversion dimension. Measures of anxiety and neuroticism correlate very highly (apx. +.70) (Eysenck & Eysenck, 1985). Gray also makes predictions with regard to the conditionability of individuals who are high of the dimension of impulsivity; however, these are not pertinent to the present research and are not discussed here.

With regard to where individuals who experience panic attacks and agoraphobic avoidance would be located in this two dimensional personality space, Eysenck and Gray are in agreement. Eysenck would expect such "dysthymic" individuals to be neurotic introverts (located in the quadrant bounded by high neuroticism and introversion). This is so because their high autonomic reactivity and chronic cortical overarousal leads to the formation of "surplus conditioned reactions." This location corresponds to the high end of Gray's dimension of anxiety. Gray would presume this to be due to an overactive behavioral inhibition system.

Personality-Related Differences in Conditionability

As already noted, Eysenck suggests that differences in introversion-extroversion and in neuroticism influence conditionability. According to the theorizing of Eysenck (1957, 1967a) individuals who are highly cortically aroused (introverts) would tend to develop associations or

"condition" more rapidly than individuals who are less cortically aroused (extroverts). Results from studies utilizing eyeblink and GSR conditioning have generally borne out the predictions made by Eysenck (Eysenck & Levey, 1967). Additionally, it is reasonable to assume that a stimulus of a given intensity will make a more potent CS+ for an introverted individual (who augments the incoming stimulus). With regard to the manner in which high neuroticism might enhance learning, it seem reasonable to assume that physiological sensations will be stronger and more frequent in neurotic individuals because they are more autonomically responsive to various stimuli. This should generally constitute a more potent UCS and enhance conditioning. Additionally, if habituation to stimuli is slower in highly neurotic individuals, autonomic activation (unconditioned stimuli) should remain salient or potent for a longer period of time. This too might enhance conditionability.

Within Eysenck's theory, it makes no difference if the stimuli involved are aversive or appetitive. Eysenck suggests that introverts will condition more readily than extroverts in either case (given the appropriate task conditions). In contrast, Gray's theory would predict that as individuals become more anxious (neurotic introverts), aversive stimuli will produce better conditioning than appetitive stimuli because the behavioral inhibition system is involved. The theorizing of Eysenck and Gray carries

implications for operant as well as classical learning processes. However, the present research limits itself to the role of individual differences in classical conditioning processes. Some additional predictions made by Eysenckian theory are now reviewed.

Personality-Related Differences in Stimulus Sensitivity

An additional prediction made by Eysenckian theory which seems relevant to a discussion of avoidance learning involves sensitivity to stimulation. Eysenck (1967a) suggests that a consequence of the heightened cortical arousal which characterizes the introvert is that incoming signals are "augmented." This results in introverts being more sensitive to stimuli and having lower sensory thresholds than extroverts. This leads to an expectation that introverts would seek out lower levels of stimulation than extroverts. It follows that if introverts were unable to avoid a highly stimulating situation (e.g., a shopping mall), they might find it more aversive and more emotionally arousing than would extroverts. Weisen (1965), Davies, Hockey, and Taylor (1969), Gale (1969), and Hill (1975) all obtained results supporting this prediction.

Personality-Related Differences in Autonomic Responsivity

Recall that Eysenck suggests that individuals high on his dimension of neuroticism will be highly autonomically reactive. It seem reasonable to assume that physiological sensations will be stronger and more frequent in such

individuals. This should generally constitute a more potent UCS and enhance conditioning. Additionally, increased autonomic reactivity might result in various situations (e.g., concerts, noisy places such as carnivals) being aversive to such individuals.

Personality-Related Differences in Habituation to Activating Stimuli

Additionally, if autonomic habituation to stimuli is slower in highly neurotic individuals, then a given internal UCS (e.g., muscle tightness, shortness of breath, heart palpitations) should remain salient or potent for a longer period of time. This would result in more frequent and/or longer presentations of unpleasant UCS which could serve as (a) a UCS to which environmental stimuli such as the department store become conditioned) and/or (b) a UCS which elicits catastrophic cognitions (e.g., I must be dying!) via an association (palpitations = heart failure) which is part of the individual's learning history. Thus, slower habituation might enhance conditioning of agoraphobic stimuli to bodily sensations and might lead to more frequent elicitation of catastrophic cognitions (which, as noted previously, have their own stimulus properties).

Personality, Avoidance Learning, and Panic

The theorizing of Eysenck and Gray converge to suggest that personality-related individual differences in (a) conditionability to aversive stimuli, (b) stimulus

sensitivity, (c) autonomic reactivity, and (d) habituation may influence the development of panic and agoraphobic avoidance.

What then is the mechanism through which these individual differences might influence development of panic and agoraphobic avoidance? In anxious (neurotic introverted) individuals, more frequent and stronger autonomic responses will combine with increased conditionability to produce more and stronger CS+s (both bodily sensations and external stimuli) for fear responses. Thus, we would expect that in anxious individuals there will be more frequent and stronger elicitation of conditioned emotional response (CERs) in response to stimuli (e.g., bodily sensations or the shopping mall) which were present during previous panic attacks. Consider the following example. Chronic autonomic overarousal, and perhaps hyperventilation, in a predisposed individual, leads to a spontaneous panic attack in a shopping mall (a highly stimulating environment). Via the potent UCS (bodily sensations) and heightened conditionability, a strong connection is made between the panic-related bodily sensations and the shopping mall. A Pavlovian connection has been established between the sensations of a panic attack and the shopping mall. The shopping mall (CS) is now capable of "eliciting" panic sensations (UCR). Exposure to the shopping mall now produces a heightened "drive state."

The panicker, who is now highly activated, makes a socially acceptable response and flees to someplace less stimulating (home is the favored sanctuary) and the attack eventually subsides. The response of escaping lowers the aversive drive state elicited by the now aversive shopping mall (CS). Both Eysenck and Gray predict that our predisposed anxious (neurotic introverted) individual will learn to "emit" this escape response more readily than an individual not so predisposed.

The present author has recently administered the Panic Attack Questionnaire, the Mobility Inventory for Agoraphobia, the Eysenck Personality Inventory, and two measures of trait anxiety to a sample of nonclinical panickers and nonpanickers. Nonclinical panickers appeared to occupy a location higher on the dimensions of anxiety, neuroticism, and introversion (the inverse of extroversion) than nonpanickers. Additionally, nonclinical panickers high on self-reported agoraphobic avoidance appeared to be more trait anxious, neurotic, and introverted than nonclinical panickers low on self-reported agoraphobic avoidance. These findings suggest a relationship between the phenomena of panic and agoraphobic avoidance and the Eysenck/Gray two-dimensional personality space. Assuming that the Eysenck/Gray predictions are accurate, then these findings are also supportive of the concept of individual differences in learning and related processes, as predicted by Eysenck

and Gray, as having an influential role in development of both panic and agoraphobic avoidance.

The predictions regarding individual differences in conditionability which follow from the theorizing of Eysenck and Gray have been empirically supported. Welch and Kubis (1947a) found that the skin conductance response was conditioned more rapidly in highly anxious individuals than mildly anxious individuals and that mildly anxious individuals conditioned more rapidly than non-anxious individuals. Welch and Kubis (1947b) found that the conditioned skin conductance response was both obtained more rapidly and was more resistant to extinction in highly anxious individuals when compared with non-anxious controls. Vogel (1960b) found that in a sample of alcoholics, introversion and neuroticism were predictive of rapid acquisition and slowed extinction of the conditioned skin conductance response. Vogel (1961) obtained similar results. Bitterman and Holtzman (1952) found that the conditioned skin conductance response was developed more quickly, and was more resistant to extinction, in highly anxious university students than in non-anxious students. There are numerous other examples (see Eysenck, 1965). To date, such work has not been carried out by dividing subjects on status as a panicker or nonpanicker or by level of self-reported agoraphobic avoidance.

Nonclinical Panickers: A population Within Which to Study
Study Personality, Learning and Related Processes

The occurrence of panic attacks is not limited to panic disorder or agoraphobia. Panic attacks have been observed in a number of psychiatric disorders (Barlow, 1985), alcoholics (Cox, Norton, Swinson, & Endler, 1990), and in first order relatives of panic disorder patients (Crowe, Noyes, Pauls, & Slymen, 1983). Additionally, it has been estimated that as many as 42 percent of major depressives may experience panic attacks (Norton, Cox, & Malan, 1992). Panic attacks, of course, also occur in phobic disorders such as simple phobia and social phobia; however, in contrast to panic disorder the attacks in simple and social phobia occur only when triggered by the feared stimuli.

These observations, and the notoriety accorded panic-related disorders during the early 1980s, likely provided impetus for the study of nonclinical panic. The term nonclinical or infrequent panicker is generally used to describe an individual who has experienced, or is experiencing, panic attacks but without the frequency to qualify for a diagnosis of panic disorder. Norton, Harrison, Hauch, and Rhodes (1985) were responsible for the first published study on nonclinical panickers. Additionally, Norton et al. (1986) developed a self-report questionnaire, the Panic Attack Questionnaire, to assess for panic attacks and panic disorder in terms of DSM-III-R

criteria. To date, over 30 studies have focused on nonclinical panickers.

One of the first phenomena to attract attention was the unexpectedly high prevalence of nonclinical panic. The average prevalence for questionnaire format studies appears to be on the order of 20 to 30 percent reporting one or more attacks during the past year. In several samples of university students, the present author has found the prevalence of nonclinical panic to be on the order of 25 percent (Richman & Nelson-Gray, 1991; Richman & Nelson-Gray, 1992).

Nonclinical panickers consistently score higher than normals on measures of anxiety and depression (Norton et al., 1985, 1986) and on measures of personality disorder symptomatology and agoraphobic avoidance (Richman & Nelson-Gray, 1991, 1992). Nonclinical panickers appear to be similar to clinical panickers on a number of dimensions and, as a group, would appear to be intermediate between nonpanickers and clinical panickers on an "anxiety continuum." I believe that I have argued convincingly for the utility of this population for studying phenomenology and correlates of panic and agoraphobic tendency such as conditionability and processes related to it.

Statement of Purpose

The goal of this research was to test empirically within a population of nonclinical panickers several

hypotheses which follow from the theorizing of Hans Eysenck and Jeffrey Gray. Specifically, it was suggested that it would be possible to observe group differences among four groups of subjects on the following variables: (a) sensitivity to audible stimuli, (b) magnitude of autonomic (electrodermal) responsivity to an aversive stimulus, (c) rate of habituation of the electrodermal response to an aversive stimulus, and (d) conditionability (susceptibility to conditioning of a previously neutral stimulus to an aversive unconditioned stimulus). Eysenck and Eysenck (1985) note that the obstacles to demonstrating individual differences on these variables are numerous. Nevertheless, the important role that learning processes are assumed to play in the development and maintenance of panic/agoraphobic avoidance makes apparent the value of attempting to identify individual differences which might impact on such learning.

Group membership in this experiment was based on (a) presence or absence of panic attacks (panicker or nonpanicker) and, within each of the above mentioned categories, (b) status on self-reported agoraphobic avoidance (high vs. low avoidance). Thus, there were four groups: nonpanickers low on self-reported agoraphobic avoidance (low avoidant nonpanickers or LAN), nonpanickers high on self-reported agoraphobic avoidance (high avoidant nonpanickers or HAN), panickers low on self reported agoraphobic avoidance (low avoidant panickers or LAP), and

panickers high on self-reported agoraphobic avoidance (high avoidant panickers or HAP). These four groups were assumed to lie along Gray's dimension of anxiety (i.e., on a continuum moving from Eysenck's stable extrovert quadrant through his neurotic introvert quadrant), in the order: (a) LAN, (b) HAN and LAP, and (c) HAP. In other words, the HAP group was assumed to be most anxious while the LAN group was assumed to be least anxious. The LAP and HAN groups were assumed to occupy the same middle location.

The following hypotheses were suggested.

1. While previous research by the present author suggested the above-described locations of these groups in Eysenckian two-dimensional personality space, group differences on these dimensions were not tested statistically. In the present research, the following hypotheses were tested: on neuroticism and trait anxiety it was predicted that $HAP > LAP$ and $HAN > LAN$. Panic and agoraphobic avoidance are assumed to be inversely related to extroversion by the present author. Therefore, predictions with regard to the extroversion dimension are given in terms of introversion (the inverse of extroversion). It was hypothesized that on introversion $HAP > LAP$ and $HAN > LAN$. No hypotheses were made with regard to impulsivity.
2. With regard to stimulus sensitivity (inability to tolerate increasing intensities of a stimulus), it was predicted that $HAP > LAP$ and $HAN >$ than LAN . In other

words, while high avoidant panickers would find relatively low amplitude tones to be unpleasant, higher amplitude would be required for low avoidant nonpanickers to find the tones unpleasant. Low avoidant panickers and high avoidant nonpanickers would be intermediate.

3. With regard to autonomic responsivity (skin resistance decrease in response to aversive loud tones), it was predicted that $HAP > LAP$ and $HAN > LAN$. In other words, high avoidant panickers would evidence the largest skin resistance responses while low avoidant nonpanickers would evidence the smallest skin resistance responses. Low avoidant panickers and high avoidant nonpanickers would be intermediate.

4. In addition to examining magnitude of the SR response, the form of the SR response or "response slope," a novel measure was also examined. No specific group differences were hypothesized with regard to this additional variable as it has not been previously addressed in the personality - electrodermal responding literature.

5. With regard to rate of habituation to loud white noise stimuli (steepness of slope reflecting the rate at which the skin resistance response to an aversive tone decreases over repeated presentations), it was predicted that $HAP < LAP$ and $HAN < LAN$. In other words, high avoidant panickers would habituate most slowly (flattest slope) while low avoidant nonpanickers will habituate most quickly (steepest slope).

High avoidant nonpanickers and low avoidant panickers were predicted to be intermediate.

6. With regard to conditionability (of a neutral nonsense syllable stimulus to an aversive loud tone), it was predicted that HAP > LAP and HAN > LAN. In other words, high avoidant panickers would evidence greatest conditionability (larger/more frequent conditioned skin resistance responses) and low avoidant nonpanickers would evidence the poorest conditionability (smaller/less frequent conditioned responses), with low avoidant panickers and high avoidant nonpanickers being intermediate.

HAN and LAP were not expected to differ from each other on any of these measures. Previous research by the present author suggests that they occupy nearly the same location in the Eysenck-Gray two-dimensional personality space (i.e., they do not appear to differ on measures of anxiety, neuroticism, or introversion-extroversion).

On a given dependent variable (e.g., response magnitude), it was predicted that the HAP group would evidence larger SR responses than the LAP group and that the HAN group would evidence larger SR responses than the LAN group. However, there was no reason to assume that these group difference would be additive (i.e., no interaction) or multiplicative (i.e., interaction). Thus, no predictions were made with regard to the expectation of a panicker status * MIA avoidance interaction. However, it seemed

reasonable to test for the possibility that group differences on a given dependent variable (e.g., response magnitude) would be predicted by a panic status * MIA avoidance interaction (i.e., was the difference between HAP and LAP significantly greater or smaller than the difference between HAN and LAN).

CHAPTER II

THE PRESENT RESEARCH

Method

Participants

Potential participants were selected from a large pool of undergraduate students attending introductory psychology courses at the University of North Carolina at Greensboro during two consecutive semesters. Nearly 1,250 students completed the Panic Attack Questionnaire (Norton et al., 1986) as part of a "mass screening" to help fulfill the research requirement of their introductory psychology class. The Panic Attack Questionnaire was placed in random order in packets containing additional questionnaires unrelated to the present research and distributed to students at "mass screening" sessions.

Students who reported experiencing at least one panic attack which met DSM-III-R criteria for a panic attack (Appendix A) during the past year on the Panic Attack Questionnaire (described later) were classified as "nonclinical panickers." For brevity and clarity, nonclinical panickers will subsequently be referred to as "panickers." To have been classified as a panicker, a student: (a) must have responded positively to Question 1 of

the Panic Attack Questionnaire indicating that they felt they had experienced at least one attack, similar to the one described in the questionnaire, during the past year; and (b) have responded positively (a rating of 1 or greater) to at least four of the first thirteen items (a through m) in Question 13 of the Panic Attack Questionnaire. These items correspond to the criteria for a panic attack specified in Part C of the DSM-III-R criteria for panic disorder (Appendix A). Students who met DSM-III-R criterion B for panic disorder (four or more attacks during a four week period or one or more attacks followed by at least a month of persistent fear of having another attack) would not have been requested to participate further in the research. No student met this criterion. Students who did not report experiencing a panic attack on the Panic Attack Questionnaire were classified as "nonpanickers." Of the nearly 1,250 students who completed the Panic Attack Questionnaire, 238 (19%) were classified as panickers. This percentage is consistent with that observed in previous research by the present author (Richman & Nelson-Gray; 1991, 1992) and with percentages from several studies reviewed by Norton et al. (1992). Of these 238 panickers, 162 were female and 76 were male. This gender difference would appear to reflect the demographics of the undergraduate population (about 2/3 female) rather than a gender difference in the occurrence of nonclinical panic.

Students identified as panickers (N=238) and an approximately equal number of randomly selected nonpanickers were contacted and asked to complete a packet containing the Mobility Inventory for Agoraphobia and the questionnaires assessing location on the Eysenck and Gray dimensions. These students received additional credit towards their introductory psychology course research requirement for completing the additional questionnaires.

Mobility Inventory Avoidance upper and lower quartile cutoff points were established separately for panickers and nonpanickers. These cutoffs were based on a large data set that had been collected by the present author during three earlier semesters of screening undergraduate students (N for panickers = 407, and N for nonpanickers = 469). Each student's Mobility Inventory for Agoraphobia score represents the average of that student's responses to the 27 items (scored on a 1 to 5 Likert scale) on the Mobility Inventory for Agoraphobia. Upper quartile cutoffs were 1.67 for panickers and 1.44 for nonpanickers respectively. Lower quartile cutoffs were 1.11 for panickers and 1.00 for nonpanickers. While the range of Mobility Inventory scores observed in college populations tends to be rather restricted, the present author has observed panicker-nonpanicker differences on this measure to be very consistent across several samples (Richman & Nelson-Gray, 1991, 1992).

Student (panicker and nonpanicker) whose Mobility Inventory for agoraphobia scores fell in the upper or lower quartiles were classified as "potential participants" for the experimental paradigm. Additional potential participants were solicited from among students who did not participate in mass screening. A poster asked students to fill out a questionnaire packet, containing all questionnaires needed for participation, if they had experienced an "episode of anxiety" during the past year which included four of the DSM-III-R symptoms listed in the Panic Attack Questionnaire. Those "solicited" students whose responses to the Panic Attack Questionnaire and the Mobility Inventory would place them in one of the four experimental groups being formed were added to the pool of potential participants to be contacted. Potential participants were then contacted in random fashion and were asked to participate in the experimental paradigm.

Potential participants were contacted and experimental data collected until there were 20 participants per group (total N = 80). Participants who agreed to take part in the experiment received one additional credit towards their Psy 221 research requirement. Eight of those potential participants contacted had already earned all of their research credits and were instead paid \$10.00 for participation. Seventy seven of the participants were students who took part in mass screening. The remaining

three participants were solicited. Mean age of the 80 participants was 20 1/4 years. Fifty nine of the participants were female and 21 were male. Gender breakdown by group was as follows: for HAP 16 females and 4 males, for LAP 14 females and 6 males, for HAN 13 females and 7 males, and for LAN 16 females and 4 males.

As the experiment involved response to visual and audible stimuli, individuals having deficiencies in vision (despite use of corrective lenses) or in hearing determined to be severe enough so as to invalidate their data were not asked to participate. This was determined by researcher inquiry and observation at time of initial contact with each participant. Only one potential participant, a male panicker, was excluded based on a hearing impairment.

Questionnaires/Measures

In addition to completing the Panic Attack Questionnaire (PAQ; Norton et al., 1986) (Appendix C) and the Mobility Inventory for Agoraphobia (MIA; Chambless et al., 1985) (Appendix D) to form the four groups of participants, potential participants also completed the Eysenck Personality Inventory, Form A (EPI; Eysenck & Eysenck, 1968) (Appendix E), the State-Trait Anxiety Inventory, Form Y2 (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) (Appendix F), the Minnesota Multiphasic Inventory-2 anxiety subscale (MMPI-A; Graham, 1990) (Appendix G), the "impulsiveness narrow" subscale of

the Impulsiveness Questionnaire, Seventh Edition (I.7; S.G.B. Eysenck et al., 1985) (Appendix H), and the Barratt Impulsiveness Scale, Version 10 (BIS; Barratt, unpublished) (Appendix I).

The Panic Attack Questionnaire (DSM-III-R version) is a comprehensive assessment tool which can be used to ascertain frequency, duration, symptomatology, severity, and history of panic attacks using DSM-III-R criteria (American Psychiatric Association, 1987). The Panic Attack Questionnaire has been shown to have adequate test-retest reliability ($\kappa = .65-1.00$) for almost all items (reliability for onset to peak severity, duration, and reports of unexpected attacks was lower) (Margraf & Ehlers, in press).

The Mobility Inventory for Agoraphobia contains 29 items which specify situations or places commonly avoided by agoraphobics. On the original version of the questionnaire, each situation was rated for both the extent to which entering it caused "discomfort" and the extent to which each situation was actually "avoided." Chambless et al. eliminated the discomfort scale as responses to them tended to be redundant with responses to the avoidance scale when using the questionnaire with clinical populations. Chambless et al. reported correlations between avoidance and discomfort scales ranging from .87 to .94 in populations of clinical panickers and agoraphobics. The present author,

however, has found the discomfort and avoidance scales to be less redundant when the questionnaire is used in a college population (correlations on the order of $+0.70$). The discomfort scale has been retained in the version of the questionnaire which the present author has used in previous studies and which was used in the present study (Appendix D) as it may provide useful information. Group membership in the present study was, however, based on the avoidance scale only. Convergent validity for the avoidance scale was demonstrated by a correlation of $.68$ ($n=42$) with the agoraphobia factor of the Fear Questionnaire (Marks & Matthews, 1979). Chambless et al. (1985) reported good test retest reliability with correlations for the avoidance scale ranging from $.75$ to $.90$. The original questionnaire also assessed discomfort and avoidance when the individual was accompanied by a "support person" (usually a spouse). As this measure did not seem applicable to a college population, and because it would have made the questionnaire unnecessarily long, it has been omitted from the version of the questionnaire used by the present author.

The remaining measures utilized the 2-dimensional personality conceptualizations of Eysenck (1967a) and Gray (1972, 1973). The Eysenck Personality Inventory contains 57 items. The neuroticism and introversion-extroversion scales of the EPI each contain 24 true-false items. One year test-retest reliabilities of $.82$ for the introversion-

extroversion scale and .84 for the neuroticism scale have been reported. Eysenck and S. B. G. Eysenck (1968) have reviewed validity work which has been done on the Eysenck Personality Inventory. No questionnaire has been specifically developed to assess individuals on Gray's dimensions of anxiety and impulsivity. In an attempt to obtain a broad sampling of items relating to each of these dimensions, two independent questionnaires were utilized to assess individuals on each dimension.

An anxiety index was derived by extracting a standardized principal component from scores on the STAI and the MMPI-2 anxiety scale. The form of the STAI used in this study contains 20 items which are rated on a 4-point Likert scale to assess trait anxiety. Spielberger et al. (1983) reported alpha coefficients of .90 and .91 for the STAI Form Y. Sixty day test-retest reliabilities of .68 for male and .65 for female high school students were reported. The MMPI anxiety subscale is comprised of 39 true-false items. Welch (1965), based on research using college undergraduates, reported split half reliability of .88 for the MMPI anxiety subscale. Gocka (1965) reported Kuder Richardson 21 (internal consistency) to be .94 in a sample of Veterans' Administration psychiatric patients. Welch (1965) reported 4-month test-retest reliability of .70.

In like manner, an impulsiveness index was derived by extracting a standardized principal component from scores on

the Eysenck I.7 scale and the Barratt Impulsiveness Scale. The Eysenck I.7 impulsiveness scale contains 19 yes-no questions. Reliabilities of .84 for males and .83 for females have been reported for the I.7. Validity information for the I.7 scale is reported by S. B. G. Eysenck et al. (1985). The BIS-10 contains 34 statements which are rated on a 4-point Likert scale. Though unpublished, the BIS-10 has been used in a number of studies and has been found to be a valid index of impulsiveness (Barratt, 1985a, 1987; Barratt, Pritchard, Faulk, & Brandt, 1987). Farmer (1992) noted that Barratt reported alpha reliabilities for the three aspects of impulsiveness tapped by the BIS-10 to be .87 for motor impulsiveness, .91 for cognitive impulsiveness, and .86 for non-planning.

SAS Proc Princomp was utilized for deriving anxiety and impulsivity principal components.

Design

This study utilized a two (presence or absence of panic) x two (high vs. low self-reported avoidance) design. Thus, there were four groups (as described earlier): LAN (nonpanickers low on self-reported agoraphobic avoidance), HAN (nonpanickers high on self-reported agoraphobic avoidance), LAP (panickers low on self reported agoraphobic avoidance), and HAP (panickers high on self-reported agoraphobic avoidance). The experiment was conducted in three phases.

Experimental Tasks

Phase 1 involved each participant listening to three series of tones of increasing intensity in order to establish a level of "stimulus sensitivity" for that participant. In each series, the participant indicated at what loudness the tones became "clearly unpleasant." The levels that the participant indicated in each series were averaged to arrive at that participant's "db level," the dependent variable of stimulus sensitivity. In addition to serving as one of the dependent variables in the study, this measure of stimulus sensitivity was used to adjust the volume level of white noise presentations during Phase 2 and UCS (tone) presentations during Phase 3 to a level that the participant found unpleasant but tolerable.

Phase 2 involved presentation of a series of eight white noise stimuli which were used to assess group differences in rate of habituation to repeated presentations of a novel stimulus. The slope of a regression line fitted to the magnitude of the SR responses to the eight white noise stimuli served as the Phase 2 dependent variable.

Phase 3 consisted of a Pavlovian conditioning paradigm adapted from Welch and Kubis (1947). A series of nonsense syllables, with one syllable serving as the CS, were presented to participants. The CS was paired with an unpleasant tone. Difference between skin resistance (SR) change in response to the CS syllable and skin resistance

(SR) in response to buffer syllables was the measure of conditioning. Skin resistance responding is described in detail in a subsequent section.

Stimuli and Apparatus

Phase 1 tone stimuli were produced by a Hewlett Packard Model 200 AB audio oscillator. In order to establish the loudness level at which each participant was willing to listen to white noise presentations (during Phase 2) and UCS tone presentations (during Phase 3), three series of tones were presented. In each series, tones were presented at increasing loudness (in 5 db increments starting at 65 db and continuing to a maximum level was 100db). Tone presentations were one second in duration. Because SR response to the first two tones in each series was used in computing the response magnitude measure (described later), tone presentations were variably spaced (in a random manner) during recording of the stimulus tape to minimize the likelihood of SR responses caused by expectancy of the next tone presentation. Intervals between tone presentations ranged from 8 to 18 seconds. The participant indicated, by pressing a button, when the volume level of the tones had reached a level that was "clearly unpleasant" but that he or she could tolerate during subsequent exposures. The participant's acceptable level of loudness, for three series of tones, at frequencies of 2,000, 3,000, and 4,000 hertz respectively, was averaged to establish a "db (decibel)

level" or stimulus sensitivity for that individual. Established sound contours (Robinson & Dadson, 1956) indicate that within this frequency range audible stimuli will be perceived as being of equivalent loudness regardless of the frequency.

The goal of Phase 2 was to examine rate of habituation to a novel stimulus. Because white noise is made up of sound waves of numerous frequencies, it is typically perceived as louder than tones of a single frequency. Level of white noise presentation for each participant was established by a conversion formula which was created in the following manner. The researcher and five other volunteers went through the procedure described for establishing db level. Each of these individuals then listened to one second exposures of the 3,000 hertz tone at their db level through one ear and adjusted the volume of alternately presented white noise exposures to the other ear for "equal loudness." The difference for each volunteer (e.g., tone at 85 db and white noise at 75 db = a difference of 10 db) was averaged with the differences obtained from the other volunteers. The actual average of these differences was -3 db. Thus, white noise stimuli were presented at a level 3 db less than the participant's db level established during Phase 1. White noise for use during Phase 2 was produced by a Grasson-Stadler Model 901 Noise Generator.

Phase 2 consisted of eight white noise presentations of

one second duration. The first of these presentations occurred one minute after the last tone of the last of the three Phase 1 tone series, regardless of whether the participant had heard that tone or had stopped the series earlier. As with Phase 1 tone presentations, the white noise presentations were spaced variably (intervals ranging from 10 to 20 seconds) to avoid SR responses resulting from expectancy of the next presentation.

A good deal of consideration was given to the audible stimulus which was to serve as the UCS during Phase 3. During pilot work for this study, the 2,000, 3,000, and 4,000 hz pure tone stimuli from Phase 1 were used as the UCS. It was noted that a great deal of preexposure, and possibly habituation, to these tones were taking place. For example, a participant with a db level of 90 would have heard 18 of the pure tone presentations during Phase 1. For this reason, a new audible stimulus was sought. A high pitched (830 hz) automobile horn, recorded onto the videotape, was chosen because its unpleasant timbre would likely make the UCS presentations more unpleasant at a given volume level. The previously discussed loudness contours established by Robinson and Dadson (1956) indicated that the tones produced by the 830 hz horn should be presented 5 db above the participants db level established in Phase 1 to maintain equal perceived loudness.

In the Welch and Kubis (1947) study and in other

studies which used this paradigm, a new nonsense syllable appeared every six seconds. The UCS began .5 seconds after CS (syllable) onset and remained on until CS offset. Thus, the interstimulus interval (ISI) was .5 seconds and UCS duration was 5.5 seconds. In developing the adaptation of the Welch and Kubis (1947) paradigm to be used in this study, it was posited that the non-occurrence of the UCS (tone) following .5 seconds after presentation of the CS (syllable) might serve as a "safety signal" indicating that no UCS was to come. Therefore, the ISI was extended to four seconds. This allowed for a longer period of uncertainty which, it was hoped, would increase the probability of eliciting UCRs (unconditioned responses). To accommodate this increased ISI, duration of UCS (tone) presentation was reduced to 1 second. This did not appear to make the UCS less effective and may have actually reduced the extent to which participants habituated to the tone.

Onset and duration of tones and white noise was controlled by a circuit consisting of a Lafayette Model 58010 Electronic Timer and a Hunter 111-C electronic timer. Tones and white noise were recorded onto the audio channel of a videotape cassette on an RCA CC017 Video Color Camera. Tone and white noise stimuli were reproduced from videotape on a Symphonic Model 7700Z video cassette deck. The audio signal was amplified through an Allied Model 395 receiver/amplifier and was attenuated to each participant's

"db level" via a Hewlett Packard 350D Attenuator Set. Loudness of audible stimuli (in db) was measured at the headphones with a Ballantine Laboratories Model 320A RMS Voltmeter. Participants heard audible stimuli binaurally over a set of Telephonics TDH49P headphones.

The series of visual stimuli for conditioning (Phase 3) were derived from 40 different 3-letter pronounceable nonsense syllables of low meaningfulness (Appendix j). These syllables were selected from the list of 2,019 nonsense syllables compiled by, and categorized with regard to meaningfulness or association value, by Glaze (1928). Glaze "values" range, in increments of seven, from 0 (none of his subjects had an association to the syllable, e.g., ZIL) to 100 (all of his subjects had an association to the syllable, e.g., PIL). Two syllables beginning with each "consonant" were randomly chosen from syllables with Glaze values of 20 or less to insure that the syllables would not have any "meaning" for the participants beyond the stimulus properties that they acquired during the experiment. The letter "x" was omitted as syllables beginning with this letter tend to be difficult to pronounce. This provided a pool of 40 syllables from which the final series of 140 presentations was developed.

The series of nonsense syllables seen by all participants (Appendix K) was constructed as follows. Each participant saw the same series of 140 nonsense syllable

exposures during Phase 3. One syllable, chosen at random from the pool of 40 syllables (GAX), served as the CS. The 39 remaining syllables (Appendix J) served as potential "buffer syllables." The CS appeared 20 times with the UCS (acquisition trials) and 20 times without the UCS (test trials). To assure that CS presentations were well distributed throughout Phase 3, the conditioning series was constructed in four "blocks" with each block containing 5 acquisition trials, 5 test trials, and 20 buffer syllables. This "block" construction was used only to aid in creating the sequence of syllables and was not perceived by the subject. The actual sequence was created as follows. The buffer syllables to appear in each block (20 syllables) were taken from the list of 40 "Glaze" syllables discussed earlier using a random number table. The selected syllables were written on small pieces of paper. A syllable could have occurred more than once in a block or not at all. For each block, the following were placed in a box: 20 pieces of paper containing the selected buffer syllables, 5 pieces of paper marked GAX + (for acquisition trials), and 5 pieces of paper marked GAX (for test trials). The pieces of paper were drawn from the box at random. The exception to the random selection was that at no time would the syllable GAX (acquisition or test trial) appear twice without at least one buffer syllable intervening. In such a case, the syllable that violated this was replaced in the box and

another syllable drawn.

Syllables were recorded onto videotape via the RCA video camera. Syllables were reproduced via the Symphonic 7700Z video recorder and presented to participants on a Zenith 19" diagonal color television monitor. Syllables appeared as being black on a gray background. Syllables were approximately five inches high x eight inches wide. With participants seated 48 inches from the monitor the syllables subtended a visual angle of approximately 5.8 degrees. A new syllable appeared every 6 seconds. Thus, the Phase 3 series took approximately 14 minutes to complete (6 seconds x 140 syllables).

Exact time of onset of audible and visual stimuli were fed to two channels of a Grass Model 79 EEG and Polygraph Data recording system via electronic relays. This, in turn, produced pen deflections on a Grass Model 7H-25-60 Chart Drive, thus recording the events. These recordings served as reference points for measuring SR changes.

Dependent Measures

Stimulus Sensitivity (db Level)

To determine the level at which audible stimuli became unpleasant to each participant, a series of tones (three series at 2,000, 3,000, and 4,000 hertz respectively) were presented. Tones were of one second duration and were presented at intervals ranging from 8 to 18 seconds. Sound pressure level began at 65 db and increased in increments of

5 db until a maximum of 100 db was reached. The participant indicated (by pressing a button during the interval following a tone presentation) that the volume level that he or she had just experienced was at "the level at which the tone has become clearly unpleasant but at which he or she would be willing to hear sounds during the remainder of the experiment." The levels that the participant indicated on the three tone series were averaged to arrive at that participant's "db level" or stimulus sensitivity.

Response Magnitude

Skin resistance (SR) response refers to a change in the resistance (or its reciprocal, conductance) offered by the skin to a small externally applied current (10 microamperes in the present research) which flows between two electrodes which are attached to the skin. Electrodermal responding is assumed to be closely tied to, and therefore may be taken as an indicant of, activity of the sympathetic branch of the autonomic nervous system. The studies upon which this paradigm was based used skin conductance (SC) (measured in micromhos) for their dependent measures. The instrumentation utilized in the present study, however, dictated that skin resistance (SR), the reciprocal of conductance, be utilized for the dependent measures in the present research. The unit of resistance is the ohm (reciprocal of the mho) and responses will generally be measured as a change in K ohms (1,000 ohms). Note that

because an autonomic "response" results in a decrease in skin resistance (increase in conductance) larger responses are indicated by a greater negative change in resistance (i.e., -25K is a larger response than -2K). Changes in skin resistance (SR) were recorded in the following manner. Two rectangular electrodes (each approximately 1 cm² in size) were attached to the medial phalanx (area between the first and second joint) (Andreassi, 1989) of the index and middle fingers of the subject's non-preferred hand (i.e., the left hand for a right handed person). Skin resistance data were recorded on a Grass Model 79 EEG and Polygraph Data Recording System. The skin resistance signal was fed through a Grass Model 7P1F Low Level DC Preamp to a Grass Model 7DAF DC Driver Amplifier. A record of the subject's skin resistance responses during the session was recorded on Channel 1 of the Grass Model 7H 25-60 Chart Drive. Pen deflection indicated the magnitude of the response. Amount of pen deflection was converted to change in skin resistance in K omhs.

The measure of response magnitude for each subject was the average of his or her skin resistance responses to ten audible stimuli: (a) the first two tones during each of the three Phase 1 series, (b) the first two white noise presentations during Phase 2, and (c) the first two tone (UCS) presentations during Phase 3. It was felt that the best estimate of a participant's SR responsivity could be

obtained by averaging responses to various "novel" stimuli throughout the procedure. Responses consisted of the greatest change in skin resistance (in K ohms) during the interval beginning with stimulus (tone) onset and extending to 6 seconds after stimulus onset (interval of 6 seconds).

Response Slope

During planning of the present research, it was thought that it would be of interest to assess group differences in the "form" or shape of the SR response. While SR recovery (speed of return to baseline following an SR response) has been examined, the present author is not aware of any research in the personality - electrodermal responding literature that has examined "response slope" (i.e., slow change in skin resistance versus rapid change in skin resistance in response to a stimulus). It was thought that group differences here might suggest an additional aspect of autonomic/electrodermal responding which might influence individual differences in the variables (e.g., conditionability) being examined in the present study. The measure of response slope was the steepness of the line running from (a) the subject's SR level (on the Grass chart recorder) at the time the SR response begins to (b) the point of maximum height on the chart recorder during the 6 second interval following stimulus onset. Response slope was computed as rise (the measure of response magnitude in K ohms) over run (the time between the start of the response

and peak of the response). As with response magnitude, response slope was averaged for (a) the first two tones during each of the three Phase 1 series, (b) the first two white noise presentations during Phase 2, and (c) the first two tone (UCS) presentations during Phase 3 (total of 10 responses).

Rate of Habituation

Individual differences in rate of habituation were based on the change in skin resistance in response to a series of eight white noise presentations during Phase 2. Skin resistance response (greatest SR change occurring during the 6 seconds following stimulus onset) was recorded for each of the eight white noise presentations. The eight data points for each participant were utilized to derive a regression coefficient. This coefficient served as the measure of rate of habituation for further analyses. Note that because the SR responses are recorded as negative changes in skin resistance, greater negative coefficient values indicate slower habituation.

Index of Conditionability

The criteria for determining individual differences in conditioning used by Welch and Kubis (1947a, 1947b) and Vogel (1960b, 1961) was "rate of conditioning" or number of CS-UCS pairings occurring before a specific criterion was met. This criterion was the occurrence of two SC responses to the CS larger than any response to any syllable

intervening between the two CS presentations. As the paradigm to be used in the present research was being developed, it was noted that the criterion of conditioning used in the Welch and Kubis (1947a, 1947b) and Vogel (1960b, 1961) studies could well be met by chance rather than by actual evidence of conditioning. This criterion would be especially easy to meet if only one or two buffer syllables separated two CS test trials. Additionally, the present author has noted that participants differed dramatically in the way in which they evidenced a conditioned response. A given participant might, for example, (a) evidence many large SC responses to the CS throughout the paradigm, (b) evidence many small SC responses to the CS throughout the paradigm, (c) evidence several large responses to the CS with the CR fading away quickly or (d) evidence several small SC responses to the CS which fade away quickly. The idiosyncratic nature of SR responding prompted the present author to adopt an "index of conditioning" that would take into account the numerous individual differences in SR responding. The "index of conditioning" utilized in the present study was a t statistic arrived at by comparing each participant's SR change during 40 randomly selected buffer syllables (10 from each block) with the participant's SR change during the 20 CS test trials. In this way, individual differences in both magnitude and frequency of the CR were taken into account. As this t statistic was

being utilized as an indicant of effect size and not a statistical test, failure to meet the assumptions inherent in the use of parametric statistics (e.g., independence of the observations in the trial and random by sampled populations) did not present a problem.

A segment from the latter part of an actual record is depicted in Figure 1. Each downward pen deflection at the top of the record indicates a new nonsense syllable appearing every six seconds. The darker, wider downward deflections indicate the tone UCS. Test trials are marked with an *. It can be seen that this participant was quite responsive to the tone UCS. This participant evidenced particularly robust conditioning to the CS syllable "GAX." This is apparent not only on the test trial (marked with an *) but is also apparent on two of the three conditioning trials shown in the record excerpt. It is also interesting to note that this participant displayed a relatively long "response latency" (time between stimulus onset and beginning of the SR response).

Procedure

Potential participants, after being contacted by the researcher by phone, met with the researcher on the third floor of the Eberhart Building at a scheduled time. At this time, the researcher provided the potential participant with a written explanation of the procedures involved in the experiment, approximately how long the experiment would

take, and what discomforts (if any) the procedure might entail (Appendix L). After the potential participant read the written presentation, the researcher answered any questions the potential participant had, as long as answering these questions did not bias the potential participant's responding in the paradigm. Participants were told that they would be participating in a task which was assessing the influence of various stimuli on the sweat activity of their skin during relaxation. If the potential participant elected to proceed with the research, he or she then signed the consent form (Appendix M) and accompanied the researcher to a room on the third floor in the Eberhart building where the research took place.

Phase 1 involved the participant listening to three series of tones to establish a db level (stimulus sensitivity) for that participant as well as for establishing an index for setting levels of white noise and tone stimuli during Phases 2 and 3 of the research. The participant was seated upright in a comfortable chair and the SR electrodes were attached to the participant's non-dominant hand. The researcher explained that the participant would hear several series of tones which would gradually increase in loudness. The subject was instructed to press a button by his or her dominant hand to indicate "the level at which the tone has become clearly unpleasant but at which he or she would be willing to hear sounds

during the remainder of the research" (Appendix N). The participant was instructed that following the third series of tones, the television monitor would turn off. This served as a reminder that the participant should no longer use the button and should simply "close his or her eyes and listen to the sounds which followed" (Phase 2). The researcher then provided the participant with a pair of headphones, told the participant to relax and listen to the background music which was playing, and moved to an adjoining room which contained the polygraph recording equipment. When available, an undergraduate student majoring in psychology assisted the researcher in recording data. Five minutes was allowed for the subject to become accustomed to the situation and for the SR readings to stabilize. A "baseline" measure of skin resistance was taken during the last minute prior to the beginning of the first series of Phase 1 tones (average of six randomly spaced readings during the last minute prior to the start of Series 1). This baseline served as a covariate in the various analyses which were performed. The first series of tones (2,000 hertz), starting at 65 db and increasing in 5 db increments through 100 db, was played back on the videotape. The television monitor indicated which series of tones were being heard (e.g., "SERIES 1") during Phase 1 to help the participant keep track of the procedure. When the subject pressed the button, the researcher or assistant

switched off the audio signal and recorded the db level at which the button was pressed. The researcher or assistant switched the audio signal back on when the next series of tones was about to begin. The sequence was repeated three times at frequencies of 2,000, 3,000, and 4,000 hertz to establish that individual's stimulus sensitivity over a wide range of frequencies.

The first Phase 2 white noise presentation followed approximately one minute after the final tone presentation of the third Phase 1 series (regardless of whether or not the participant tolerated the full series). Phase 2 consisted of eight 1 second presentations of white noise, variably spaced between 10 and 20 seconds. Prior to Phase 2, the television monitor had been shut off (by the researcher or assistant from the adjoining room). This, as the participant had been told, was a reminder that he or she should not use the button and should close his or her eyes and just listen to the sounds that came over the headphones.

After Phases 1 and 2 were completed (approximately 15 minutes including the 5 minute adaptation period), the participant took a break of approximately ten minutes. The goal of this break was to relieve fatigue and reduce habituation of the SR response. The SR electrodes and headphones were removed and the participant returned to the waiting room for approximately 10 minutes.

Prior to beginning Phase 3, the researcher brought the

participant back to the research area, asked the participant to again be seated, attached the SR electrodes, assisted the participant in positioning the headphones, and read additional instructions for Phase 3 (Appendix N) to the participant. The researcher again moved to the adjoining room, set the sound level for that participant, and started the videotape after at least three minutes of adaptation. The participant was instructed to say each of 120 nonsense syllables out loud one time as he or she saw each one appear on the television monitor. Phase 3 took approximately 16 minutes to complete including time for SR readings to stabilize.

At the end of Phase 3, the researcher entered the room, removed the headphones, and removed the SR electrodes. The participant was then given a brief questionnaire to respond to (Appendix O). The questionnaire asked if the participant (a) was aware of a relationship between the tones and any of the nonsense syllables, and (b) what the syllable(s) was(were) if there was a relationship. The participant was then given a printed debriefing to read (Appendix P). After this, the participant was free to ask the researcher or assistant any additional questions that he or she may have had about the research. The participant was asked not to discuss the nature of the research with other students who might also be participating in the experiment.

CHAPTER III

RESULTS

Overview of Statistical Methods

Descriptive group statistics were computed for (a) participant demographics (Table 1), (b) personality data, (Table 2), and (c) dependent variable data (Table 2). Anxiety and impulsivity indices were derived from the anxiety and impulsivity questionnaire data collected via the SAS Proc Princomp procedure (SAS Institute, 1985). In each case the first principal component was utilized. Pearson correlations among the dependent variables were computed using the SAS Proc Corr procedures (SAS Institute, 1985) (see Table 3).

Questionnaire and experimental data to be analyzed were examined to determine the extent to which the assumptions (e.g., normality) of the parametric statistical procedures to be used were met by the data. In general, the data appeared to meet the assumptions made when using parametric statistics. However, the distributions of data for two of the skin resistance dependent variables, SR baseline and SR response magnitude, were somewhat skewed. Several transformations that are often used in such situations (log, square root, and reciprocal) were applied to the SR baseline

and SR response magnitude data in an attempt to better normalize the distributions. The log transformation did reduce skewness in the distribution of SR baseline data. However, the improvement in normality was modest and had no impact on the various analyses performed. Therefore, the SR baseline data were left in their original form. None of the transformations tried reduced the skewness of the SR response magnitude data. Removal of several "outliers" (high SR responders) reduced skewness somewhat in the SR response magnitude data. However, results of the various analyses were not altered by removing these data points. Consequently, it was decided that removal of these data points was not advantageous.

When dealing with several variables, such as the dependent variables in the present research, which are assumed to be related to each other, a multivariate analysis of variance (MANOVA) is often performed. While some of the dependent variables in the present research are related or correlated (e.g. response magnitude and habituation), it was the independent relationships of the dependent variables to the independent variables (i.e., panicker status and MIA avoidance status) that were of primary interest. Additionally, a canonical correlation was used to assess relationships between and among the dependent and independent variables. It was determined that the individual analyses of variance/covariance and the canonical

correlation analysis would provide the same information that the MANOVA would have provided. Thus, a MANOVA was not performed.

For each of the dimensional personality measures, and for each of the dependent measures, a two way analysis of variance was performed assessing the influence of the independent variables (a) panicker status, (b) MIA avoidance status, and (c) the panic status * MIA avoidance status interaction on the personality or dependent variable of interest. SR baseline was included as a covariate in each analysis involving SR data. The SAS Proc Gln procedure (SAS Institute, 1985) was used for each of these analyses. Each of these analyses was followed with a posteriori Fisher's LSD multiple comparisons. To assess for relationships between and among the personality variables and dependent variables, a canonical correlation was performed using the SAS Cancorr procedure (SAS Institute, 1985). Throughout these analyses, the present author has adopted the generally accepted probability level of .05 as the criterion for rejecting the null hypothesis. However, because this study involved what was essentially an "analogue" population, results which closely approached the .05 level were noted and discussed.

Group Differences on the Personality Variables

To assess the extent to which the four experimental groups were located in Eysenckian two-dimensional space as

predicted (i.e., the extent to which panicker and MIA (Mobility Inventory for Agoraphobia) avoidance status were predictive of location on the Eysenck and Gray Dimensions), four separate two way analyses of variance were performed. In each analysis, the dependent variable was one of the personality dimensions of interest: neuroticism, extroversion, anxiety, or impulsivity. The independent variables in each analysis were status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction. Each of the four analyses of variance was followed with a posteriori Fisher's LSD (least significant difference) group comparisons.

As can be seen in Table 4, there was a significant main effect for the relationship between panicker status and neuroticism, $F(1,76) = 19.37, p < .0001$. There was also a significant main effect for MIA avoidance status, $F(1,76) = 23.26, p < .0001$. The test for the interaction did not reach statistical significance. A posteriori group comparisons on neuroticism reached statistical significance with the exception of the comparison of LAP and HAN for which a difference was not predicted. As can be seen in Table 4, group means on neuroticism were ordered as predicted (HAP > LAP and HAN > LAN).

In the analysis of variance examining extroversion (Table 5), the main effect of panicker status reached

statistical significance, $F(1,76) = 4.01$, $p=.0488$. The MIA avoidance main effect also was significant, $F(1,76) = 6.70$, $p=.0116$. The interactive effect was negligible. A posteriori Fisher's LSD group comparisons indicated that only one of the predicted group differences reached the .05 level of statistical significance. This was the comparison of HAP vs. LAN. The groups were, however, ordered in accordance with the hypotheses on extroversion (i.e., HAP < LAP and HAN < LAN). The relative locations of the four experimental groups in Eysenck's two-dimensional personality space are plotted as a function of standardized group means on the neuroticism and extroversion scales of the Eysenck Personality Inventory in Figure 2.

Tables 6 and 7 assess the extent to which the experimental groups were located in Gray's modification of Eysenck's personality space. Recall that the Gray dimensions (anxiety and impulsivity) represent a counterclockwise rotation of the Eysenck dimensions.

Table 6 presents the results of the analysis of variance examining the relationship between anxiety and panicker/MIA avoidance status. As can be seen in Table 6, there was a significant main effect for panicker status, $F(1,76) = 32.50$, $p<.0001$. There was also a significant main effect for MIA avoidance status, $F(1,76) = 19.63$, $p<.0001$. The panicker status * MIA avoidance status interactive effect was negligible. The results of the Fisher's LSD

group comparisons on anxiety closely paralleled the results of the group comparisons on neuroticism. A posteriori group comparisons on anxiety reached statistical significance with the exception of the comparison of LAP and HAN for which a difference was not predicted. As can be seen in Table 6, group means on anxiety were ordered as predicted (HAP > LAP and HAN > LAN).

The analysis of variance examining relationships between panicker/MIA avoidance status and impulsivity (Table 7) indicated that neither the main effects of panicker status and MIA avoidance status nor the interactive effect of panicker * MIA avoidance status approached statistical significance. None of the a posteriori group comparisons on impulsivity reached statistical significance. While the group means on impulsivity were ordered HAP > LAP and HAN > LAN, the very small magnitude of these differences suggests that this ordering was likely to chance. In general, this variable has not proved to be predictive of panicker or MIA avoidance status in previous research by the present author.

The relative locations of the four experimental groups in Gray's modification of Eysenck's two-dimensional personality space are plotted as a function of standardized group means on the derived anxiety and impulsivity indices in Figure 3. Note that Gray's dimensions are "rotated" counterclockwise from Eysenck's dimensions.

Group Differences on Stimulus Sensitivity (db level)

To assess for group differences in stimulus sensitivity, a two-way analysis of variance was performed with the dependent variable being the "db level" for each participant established during Phase 1, and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * avoidant interaction (Table 8). As can be seen from Table 8, neither the independent variable main effects nor the panicker * MIA avoidance interaction approached statistical significance in accounting for group differences in stimulus sensitivity. Group means and a posteriori group comparisons (Table 8) indicate that group means on stimulus sensitivity were virtually identical.

Group Differences on Response Magnitude

To assess for group differences in response magnitude, a two-way analysis of covariance was performed with the dependent variable being the index of response magnitude described earlier, and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction (Table 9). In this analysis, and in subsequent analyses involving SR data, SR baseline was included as a covariate. This was done to take into account the possibility that SR baseline

might influence SR responding. As can be seen from Table 9, there was a statistically significant main effect for panicker status, $F(1,75) = 6.64$, $p=.0120$. The panicker status * MIA avoidance status interactive effect was negligible. The SR baseline effect was also negligible. Group means and a posteriori group comparisons (Table 9) indicate that group means on response magnitude were ordered in the predicted direction. As hypothesized, HAP > LAP and HAN > LAP. The predicted differences between HAP and LAP and between HAN and LAN were not observed at a statistically significant level, though that trend was clearly apparent. These results suggest that presence of panic attacks, and possibly high MIA avoidance are predictive of increased SR response magnitude (i.e., greater negative change in skin resistance in response to an audible stimulus).

Group Differences on Response Slope

To assess for group differences in response slope, a two-way analysis of covariance was performed with the dependent variable being the index of response slope described earlier, and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction. SR baseline was included as a covariate. The initial analysis of covariance for this variable (Table 10) indicated that there was a statistically significant main effect for

panicker status, $F(1,75) = 7.89$, $p = .0063$. The effects of MIA avoidance, the panicker status * MIA avoidance interaction, and the SR baseline covariate were nonsignificant. The group means and respective Fisher's LSD tests for response slope listed in Table 10 indicate that all four groups not only differed from each other at a statistically significant level ($p \leq .05$), but were ordered as predicted with the LAN group having the smallest (negative) resistance change to time ratio and the HAP group having the greatest (negative) resistance change to time ratio. This variable was computed as the ratio: response (resistance change)/time (from onset of response to peak of response, maximum of 6 seconds) This would appear to suggest that the more neurotic/anxious participants (HAP and LAP groups) had more rapid SR responses than the less neurotic/anxious (HAN and LAN) participants.

However, the similarity of the ordering between the response magnitude index and the response slope index suggested an examination of the relationship between these two variables. It was found that the two variables correlated extremely highly ($r = .93$, $p < .0001$). When response magnitude was added to the response slope analysis of covariance (Table 11), it was found that response magnitude accounted for nearly all of the variance (87%) in response slope. The effect of response magnitude in the response slope analysis of covariance reached a high level

of statistical significance, $F(1,74) = 428.67, p < .0001$. Additionally, the panicker status main effect became non-significant. Thus, the apparent group differences in response slope were likely a reflection of group differences in response magnitude. For this reason, response slope was not utilized in subsequent analyses.

Group Differences on Rate of Habituation (habituation slope)

To assess for group differences in habituation slope, a two-way analysis of covariance was performed with the dependent variable being the index of habituation slope described earlier (coefficient of a regression line fit to the decreasing magnitude of SR responses to eight white noise presentations), and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction (Table 12). SR baseline was included as a covariate. The initial habituation slope analysis of variance (Table 12) indicated that neither the main effects nor the panicker * MIA avoidance interaction reached statistical significance. The SR baseline effect was negligible. Surprisingly, group means on habituation slope (Table 12) were ordered in a direction opposite to that which had been predicted (LAN < HAN and LAP < HAP). The group comparison between the HAP group and the LAN group reached statistical significance

($p \leq .05$). This suggested that members of the HAP group actually habituated more rapidly (had steeper, more negative regression coefficients) than the LAN group.

Because the group ordering on habituation slope closely resembled the group ordering on response magnitude and response slope (HAP < LAP and HAN < LAN), the relationship between the habituation slope index and the response magnitude index was examined. These two variables were found to correlate highly ($r = .58$, $p < .0001$). For this reason, response magnitude was added to the habituation slope analysis of covariance (Table 13). As can be seen from Table 13, the response magnitude effect reached a high level of statistical significance, $F(1,74) = 34.45$, $p < .0001$ in accounting for group differences in habituation slope. Response magnitude accounted for more than one third (35%) of the variance in habituation slope, suggesting that the habituation slope data were to a large extent reflecting group differences in response magnitude.

A review of the SR response habituation literature shed some light on this surprising finding, indicating that SR response and SR habituation are indeed not independent. In numerous studies that have yielded statistically significant group differences in SR habituation, the addition of a covariate measure of response magnitude to the model was found to render group differences non-significant (Stern & Janes, 1973). While statistically partialing out group

variance due to response magnitude seems intuitive, Stern and Janes question the use of this approach as a solution to the habituation slope-response magnitude confound.

An alternative method to covarying for response magnitude as was done in the analysis summarized in Table 13, is to express each of the habituation responses as a proportion of the original response (i.e., for habituation responses 1 through n, $\text{response} = \text{response}_n / \text{response}_1$). This is frequently done with data in which a change from initial magnitude is expected at subsequent data points (e.g., in animal learning research). The reanalysis of the habituation slope data as a proportion of initial response is presented in Table 14. It can be seen that response magnitude is no longer significant in the analysis of covariance and accounts for virtually none of the variance in habituation slope. In addition, there are now no statistically significant results among the Fisher's LSD a posteriori comparisons and no comparisons that approach statistical significance. Group means are no longer ordered as they were previously. The correlation between habituation slope and response magnitude was originally $+0.59$, $p < .0001$. After converting habituation data to a proportion of initial response, the correlation dropped to $+0.07$, $p = .4899$. Thus, it appears that the confound between habituation slope and response magnitude has been removed. The results obtained utilizing proportion of initial

response data further suggest that the apparent group differences in habituation slope were reflecting differences in response magnitude.

Though apparently free of the influence of response magnitude, the use of proportion of initial response data is not without difficulties. It has been found in practice that different corrections for SR response magnitude (e.g., initial response magnitude vs. range correction) produce differing results (Venables & Christie, 1973).

Stern and Janes, in their review of the SR habituation literature, noted an additional method of approaching SR habituation data analysis. Stearn and Janes reported the use of "number of responses" until habituation (a non-response) as an index of habituation in a study by Stewart, Winokur, Stern, Guze, Pfeiffer, and Horenung (1959). This suggested to the present author that using the number of white noise presentations to which a response (negative SR change) occurred, without the occurrence of a non-response (0 or positive SR change), would provide an index of habituation that was relatively free of the influence of response magnitude and did not require a response magnitude correction. The data were reanalyzed in terms of this new variable, "habituation count."

Group Differences on Rate of Habituation (Habituation Count)

To assess for group differences in habituation count, a two-way analysis of covariance was performed with the

dependent variable being habituation count (number of responses to the eight white noise presentations before a presentation yielded a "non-response") and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction. The SR baseline covariate was also included. As can be seen in Table 15, there was a statistically significant main effect for panicker status, $F(1,75) = 4.78$, $p=.0318$. Additionally, the MIA avoidance main effect approached statistical significance, $F(1,75) = 3.30$, $p=.0733$. As can be seen in Table 15, group means were ordered in the predicted direction. A posteriori Fisher's LSD group comparisons indicated that the HAP group differed from the HAN and LAN groups, but not from the LAP group, at a statistically significant level ($p\leq.05$).

The habituation count data suggest that habituation of the SR response to repeated white noise presentations was slower for the HAP group than for the HAN and LAN, and possibly LAP, groups.

The habituation count data appeared to be more independent of response magnitude than were the habituation slope data (habituation count was found to correlate only $-.32$ with response magnitude as opposed to $+.59$ for habituation slope), but not as free from response magnitude as the proportion of initial response data (which correlated

+.07 with response magnitude).

Results of the various analyses performed on the habituation data herein, and reports from the SR habituation literature, suggest that each method of analyzing habituation data carries its own benefits and disadvantages. It was decided that in the present research habituation count data would be used in subsequent analyses.

Group Differences on Index of Conditionability

To assess for group differences in conditionability, a two-way analysis of covariance was performed with the dependent variable being each participant's "Student's t statistic" (comparison of responses to the 20 CS test trials to responses to 40 randomly selected buffer words) and the independent variables being status as a panicker or nonpanicker, level of self-reported MIA avoidance (upper or lower quartile within panicker status), and the panicker * MIA avoidance interaction. As can be seen in Table 16, none of the main effects or interactive panicker * MIA avoidant effect reached statistical significance. However, the main effect of MIA avoidance approached statistical significance, $F(1,75) = 3.07$, $p=.0841$. Fisher's LSD group comparisons (Table 16) indicated that group differences on conditionability did not reach statistical significance. However, a trend in the predicted direction was suggested, with the HAP group being higher on this variable than the other groups. Additionally, the HAN group was higher on

this measure than the LAN and LAP groups. Thus, means for the two high MIA avoidance groups were higher on conditionability than means for the two low MIA avoidance groups. Results of the analysis of covariance and group means suggest a relationship, though not a statistically significant one, between conditionability and MIA avoidance status.

The index of conditionability (Student's t statistic) used in the conditionability analysis of covariance served as a continuous variable, reflecting (quantitatively) "the extent to which the conditioned response was evidenced by each participant." One might, however, want to examine individual differences in conditionability "qualitatively" (i.e., which participant's conditioned and which did not). For this reason, Kolmogorov-Smirnov statistics and associated probability values for each participant (Table 17) have been provided. The same procedure was used here as was used for deriving the Student's t statistic, comparing a given participant's SR responses on the 20 test trials to that participant's SR responses to 40 randomly selected buffer syllables. The Kolmogorov-Smirnov statistic is a nonparametric equivalent to the t test. It was used in place of the t test here because it is less susceptible than the t test to being rendered invalid by violations of the assumptions of normally distributed errors and independence of observations made when using parametric tests.

The author will leave it to the reader to arrive at a cutoff point for determining who did and did not "condition." If, for example, a cutoff of $p < .10$ is used, the number of individuals in each group who conditioned is as follows: 13 in the LAN group, 15 in the HAN group, 13 in the LAP group, and 15 in the HAP group. These results appear to be in agreement with (a) results from the conditionability analysis of covariance in which the MIA avoidance effect approached statistical significance and (b) group means on the Student's t statistic which were higher for the HAN and HAP groups than for the LAN and LAP groups, though not at a statistically significant level.

Canonical Correlation of Personality Variables and Dependent Variables

In order to examine relationships between the personality variables of interest (neuroticism, extroversion, anxiety, and impulsivity) and the dependent variables (SR baseline, stimulus sensitivity, response magnitude, habituation count, and conditionability) a canonical correlation was performed with the personality variables forming one side of the equation and the dependent variables forming the other side of the equation. Habituation count rather than habituation slope or proportion of initial response was used in the canonical correlation for reasons previously noted. The SAS Cancorr procedure (SAS Institute, 1985) extracts, from each of two

sets of variables (personality variables and dependent variables in this case), a linear combination such that the correlation between the first canonical variable of the personality variables and the first canonical variable of the dependent variables is maximized. Second, third, and fourth canonical variables are created which are uncorrelated with the other canonical variables (except the corresponding canonical variable in the other set of variables with which the correlation is maximized). Rao's F statistic is used to test the hypotheses that the correlation between each set of canonical variables = 0.

Table 18 indicates that none of the F tests for the individual canonical correlations (1 through 4) reached statistical significance. A multivariate test for overall relationship between the personality canonical variables and the dependent canonical variables did reach statistical significance, Roy's Greatest Root $F(5,74) = 2.66, p=.03$. It should, however, be noted that Roy's Greatest Root is a liberal test statistic. Table 18 presents correlations between (a) the personality measures and their canonical variables, (b) the dependent measures and their canonical variables, and C) the dependent measures and the canonical variables of the personality variables. Given the small effect sizes found among the dependent variables utilized in this study in previous analyses, it is not surprising that the canonical correlation did not yield an easily

interpretable "simple structure."

As can be seen from Table 18, the first personality variables, "per 1" appears to represent upward movement along Gray's dimension of anxiety and upward/left movement through Eysenck's upper left quadrant (bounded by high neuroticism and low extroversion). It can be seen that per1 correlates +.79 with anxiety, +.60 with neuroticism, and -.89 with extroversion. Neuroticism loads highly ($r = +.76$) on the second personality canonical variable "per2." The third personality variable "per3" correlates highly with impulsivity ($r = +.90$).

The canonical variables of the dependent measures are somewhat difficult to interpret. The first canonical variable "dep1" correlates highly with habituation count ($r = +.79$). Dep1 correlates moderately with SR baseline ($r = +.41$). Because baseline did not appear to strongly related to any of the other variables under study, this does not appear to be a significant finding. Dep1 was found to correlate moderately ($r = -.45$) with response magnitude (i.e., with greater responsivity). The dependent variable which correlated most highly with the second canonical variable of the dependent variables was conditionability ($r = +.81$). Stimulus sensitivity, and to a lesser extent, response magnitude also evidenced some relationship to dep2 correlating +.50 and -.27 respectively. The third and fourth canonical variables did not appear to be

interpretable.

In summary, the first canonical variable of the dependent variables "dep1" appears to represent some aspect of activity of the autonomic nervous system. Dep1 correlated moderately (-.45) with SR responding (higher on dep1 is associated with larger responses) and correlated highly (+.79) with habituation count (higher on dep1 is associated with more responses to repeated stimulus presentations before a non-response occurs). Dep1 was found to correlate +.23 with neuroticism, +.31 with anxiety, and -.35 with extroversion. Though small, the correlations between dep1 and the variables of neuroticism, anxiety, and extroversion do suggest an association between location in the Eysenck/Gray personality space and some aspects of activity of the autonomic nervous system. Specifically, as a participant's location in this space moves from Eysenck's stable extrovert quadrant (lower right) through his neurotic introvert quadrant (upper left), along Gray's dimension of anxiety, increased autonomic (SR) responsivity and slowed autonomic SR habituation would be expected. There did not appear to be any additional relationships of import in the canonical correlation.

Self Report of Awareness of the CS-UCS Relationship

After completion of Phase 3, participants responded to a brief self report (Appendix O) asking if they were aware of a tone-syllable relationship and what the relationship

was (i.e., which syllable). All but four participants said they knew that the correct CS syllable (Gax) was followed by the tone some of the time. Of the four participants who did not discern the relationship, one was a HAP, one was a LAP, and two were HANs. Given these findings, it seemed apparent that no further analysis was necessary to determine that, with few exceptions, participants were "aware" of CS-UCS relationship and that statistical procedures would not yield group differences with regard to awareness of the CS-UCS relationship.

Summary of Results

Group differences on the personality variables were ordered as predicted, with all of the predicted differences reaching statistical significance. On neuroticism, anxiety, and introversion HAP > LAP and HAN > LAN. As predicted, the groups did not differ on impulsivity. No group differences were observed on stimulus sensitivity. The experimental groups were ordered as predicted on response magnitude with HAP being most responsive, LAN being least responsive and LAP and HAN being intermediate. The following comparisons reached statistical significance: HAP vs. LAN and HAP vs. HAN. Though all group comparisons (direction not predicted) on the novel measure of response slope reached statistical significance, it was concluded that these differences were merely reflecting group differences in response magnitude. As a consequence, it was concluded that response slope did

not warrant further analysis. Group means on the original measure of habituation (habituation slope) were ordered in a direction opposite to that which had been predicted. With regard to the regression line fitted to decreasing SR responses, HAP > LAP and HAN > LAN (with > indicating a steeper slope or more negative coefficient and faster habituation). However, these group differences were found to be confounded with response magnitude and were, as a result, deemed not to be a valid measure of SR habituation. No group differences in habituation were indicated in the analysis utilizing proportion of initial response data. The group means were ordered as predicted on the habituation count measure. In other words, with regard to number of white noise presentations before a non-response occurred HAP > LAP and HAN > LAN. The HAP vs. LAN and HAP vs. HAN comparisons reached statistical significance. Group differences did not reach statistical significance on the index of conditionability (t statistic). However, means for the two high avoidance groups (HAP and HAN) were higher than for the two low avoidance groups (LAP and LAN). In the canonical correlation comparing the personality and dependent variables, only the overall multivariate test for any personality-dependent correlation reached statistical significance. SR responding and habituation count did appear to be related to neuroticism, anxiety, and extroversion.

CHAPTER IV

DISCUSSION

Group Membership and Eysenck/Gray Two-Dimensional Space

The results of the present research indicate that these groups do in fact occupy specific locations in the Eysenck/Gray personality space, and that these locations appear to lie along an "anxiety continuum." These findings taken together with previous findings by the present author (Richman & Nelson-Gray, 1992) indicate that nonclinical panickers, and particularly those high on self reported agoraphobic avoidance, occupy a location higher than nonpanicking controls on this anxiety continuum. While group assignment was made on the basis of the Panic Attack Questionnaire and the Mobility Inventory for Agoraphobia, group membership was found to be highly predictive of individual levels of neuroticism and trait anxiety, supporting the suggestion of a relationship between these variables and the phenomena of panic and agoraphobic avoidance. Group membership was less predictive of individual levels of extroversion, with only the two extreme groups (LAN and HAP) differing to a significant extent on this variable. In agreement with previous research by the present author (Richman & Nelson-Gray, 1992), impulsivity

did not appear to be significantly related to the phenomena of panic and agoraphobic avoidance in this nonclinical population.

It is somewhat surprising that the Eysenck and Gray conceptualizations of personality have received as little attention as they have in the panic and agoraphobia literature, especially in light of the predictions these theories make about physiological variables such as autonomic responsivity, habituation, and conditionability. Most current writings on panic disorder and agoraphobia acknowledge the role of learning processes in the development and maintenance of panic attacks and agoraphobic avoidance. For example, it is well accepted that agoraphobics develop avoidance of places where they have experienced panic attacks. Eysenck predicts that if an individual is highly neurotic, he or she will be very autonomically responsive and will tend to experience very intense bodily sensations (unconditioned stimuli or UCSs). If habituation is slowed in a highly neurotic individual, as Eysenck predicts, then these bodily UCSs will be more persistent and frequent as well. Couple these larger and more frequent UCSs with enhanced conditionability in a highly introverted/neurotic person (Eysenck's prediction) or in a highly anxious person (Gray's prediction), and increased agoraphobic avoidance is likely. Results of previous research (Richman & Nelson-Gray, 1992) as well as

results from the present research point to the usefulness of the Eysenck and Gray personality conceptualizations in examining the relationship between personality, the phenomena of panic and agoraphobic avoidance, and related physiological variables. This would seem particularly true for the dimensions of neuroticism and trait anxiety.

Findings on the Dependent Variables

Stimulus Sensitivity (db level)

Of the results obtained in the present research, those obtained for stimulus sensitivity (db level) were probably of least interest. Group differences on this measure were virtually nonexistent. The hypotheses made concerning stimulus sensitivity derive from Eysenck's theory, with particular reference to one's location on the dimension of introversion/extroversion. Eysenck assumes that introverted individuals "augment" incoming signals and would, as a consequence, experience stimulation in all modalities (e.g., tones, light, temperature) as becoming "unpleasant" at lower levels than would extroverts. Recall that group differences were much smaller for extroversion than for neuroticism or trait anxiety with only the two extreme groups (HAP and LAN) differing. Thus, the absence of group differences on the stimulus sensitivity measure were not entirely surprising. Had we been dealing with a clinical population, in which we might have found more highly introverted participants, we might have observed notable differences on the measure of

stimulus sensitivity. Even among studies that have selected participants at the high and low extremes on extroversion for the purpose of examining the influence of extroversion on stimulus sensitivity, results have been very mixed (Eysenck & Eysenck, 1985). The stimulus qualities of various situations (e.g., the bright noisy shopping mall) appear to be an important factor in determining what situations tend to trigger panic attacks, and what situations lead to the development of agoraphobic avoidance. It has been suggested that an individual who is particularly sensitive to environmental stimuli (an introvert according to Eysenck) may perceive relatively common stimuli as unpleasant and that these stimuli could elicit unpleasant autonomic responses. This, in and of itself, could contribute to the development and maintenance of agoraphobic avoidance. Though no group differences emerged on stimulus sensitivity in the present research, it is still possible that introversion may be a relevant variable for some individuals who experience panic attacks. Perhaps, those individuals who are severely agoraphobic are this way partly because they are highly sensitive to various environmental stimuli. It is, therefore, suggested that we continue investigating the role of individual differences in stimulus sensitivity.

Response Magnitude

Of the dependent variables examined in this study, the

findings for the response magnitude measure were far and away the most robust. Group differences in the predicted direction were evidenced for virtually all predictions. It seems likely that taking multiple measurements at several points in time when the responses were relatively free of habituation or other disrupting effects helped to achieve these results. Recall that this measure was the average of ten measurements of response magnitude to various stimuli.

The canonical correlation indicated a modest relationship between response magnitude and the first canonical variable (per1) of the personality variables. Per1 appears to primarily reflect upward/left movement through Eysenck's personality space (through the neurotic introvert quadrant) and upward movement on the trait anxiety dimension. Thus, response magnitude would appear to be, at least in part, a function of one's location along the neuroticism and trait anxiety dimensions. Eysenck has suggested that increased neuroticism would be associated with increased autonomic responsivity. A review of studies into this relationship (Eysenck & Eysenck, 1985) suggests mixed findings. Other researchers (e.g., Dureman & Saaren Seppalia, 1963)) have investigated the relationship between trait anxiety and response magnitude. Results have also been mixed.

The implications of greater autonomic responsivity in the development and maintenance of panic and agoraphobia are

evident. Environmental stimuli may elicit unpleasant or disturbing physiological responses in an individual possessing a particularly responsive nervous system. This can lead to a chronic state of overarousal (discussed in Chapter I) and may "set the stage" for an initial panic attack. This overarousal may contribute to the occurrence of subsequent attacks as well. From a therapeutic standpoint, an understanding of this phenomenon can have beneficial effects. If an individual can come to understand that he or she may experience various everyday stimuli as unpleasant due to a highly responsive nervous system, then he or she may experience less guilt and self blame for the disorder and may be more able to accept and work realistically with the problem.

Response Slope

To the best of the present author's knowledge, studies examining the relationship between SR responding and personality have not assessed response slope as did the present study. Group differences on this measure were at first apparent. It later became evident that differences on response slope were essentially a manifestation of group differences in response magnitude. Response magnitude was in fact found to account for nearly all the variance on the measure of response slope. Thus, the usefulness of this novel measure does not appear to be supported by the present findings.

Habituation (Slope and Count)

Examining the coefficients of regression lines fitted to increasingly smaller responses to repeated stimulus presentations has been a popular way of examining SR habituation (Stern & Janes, 1973). In the present research, the independent variables of interest, panicker status, MIA avoidance, and their interaction, did not account for group differences on this variable. Group means on habituation slope were, surprisingly, ordered in a direction opposite to that which had been predicted. Despite the fact that these differences were small, and only one comparison (HAP vs. LAN) reached statistical significance, this was a puzzling finding. It was, however, found that response magnitude accounted for a considerable portion of the variance in habituation slope (approximately one third). But why were the groups ordered in a direction opposite to that which had been predicted? The answer is actually quite straightforward. Imagine two individuals whom we know possess the same pattern of habituation: each succeeding response is half the magnitude of the previous one. However, the responses of the second individual are much larger (e.g., ten times larger) than those of the first individual. The coefficient of the regression line fitted to the decreasing responses will be a much greater negative value (suggesting faster habituation) for the second individual than for the first, despite the fact that the two

individuals actually are identical in their pattern of habituation.

This appears to account for the surprising trend in the habituation slope data in the present research. This habituation slope-response magnitude confounding may have been particularly problematic in the present research because the response magnitude effect was so robust. The revised habituation slope analysis of covariance table (response magnitude included) and associated group comparisons demonstrated that while the inclusion of response magnitude as a covariate might partial out its effect on the dependent variable before examining main and interactive effects, the group means would still reflect the habituation-response magnitude confound. Any group comparisons would, of course, also reflect this confound. Stearn and Janes (1973) note that the greatest difficulty with this approach is a greatly decreased probability of detecting group differences if they exist.

Taking a count of responses to habituation stimuli, as reported by Stearn and Janes (1973), provided another approach to analyzing habituation data. Findings on the habituation count variable were in general agreement with the hypotheses. The group differences were ordered as predicted, with the group located highest on the neuroticism dimension (i.e., the HAP group) emitting a greater number of SR responses to the white noise stimuli before failing to

emit a response to a white noise stimulus. The difference between the two extreme groups (HAP and LAN) did reach statistical significance.

The findings on habituation count appear to support Eysenck's prediction of slowed habituation in highly neurotic subjects. Other researchers (e.g., Epstein & Fenz, 1970) have found slowed habituation in highly anxious subjects. Habituation count was found to be modestly related to the canonical variable representing upward movement on the neuroticism trait/anxiety dimensions (per1), the same canonical variable to which response magnitude was related. This would appear to support the concept of both heightened response magnitude and slowed habituation as being (a) functions of the autonomic nervous system and (b) associated positively with neuroticism and trait anxiety in this population. With regard to the phenomena of panic and agoraphobia, if an individual habituates slowly, then arousal of the autonomic nervous system in response to various stimuli (both external such as a loud noise and internal such as lightheadedness) will be persistent. This could lead to the chronic state of overarousal which is believed by some researchers to set the stage for the first panic attack. This sustained autonomic arousal would, of course, also make subsequent attacks more likely.

While the habituation count measure at first appeared to provide a simple solution to the habituation-response

magnitude confound, further scrutiny suggested that this measure was not completely free of the influence of response magnitude.

Expressing each habituation data point as a proportion of initial response provides another approach to analyzing habituation data. This approach does appear to control well for response magnitude. A difficulty with such corrections, however, is that in practice results may vary depending upon the particular correction used (Venables & Christie, 1973). To determine whether a regression coefficient based on proportion of initial response or a count of responses is a more appropriate indicant of habituation, it will likely take a number of additional studies involving (a) participants varying widely on the anxiety/neuroticism dimensions (i.e., nonanxious individuals and clinically anxious individuals, to increase effect size) and (b) large numbers of participants (in order to detect small effect sizes).

Conditionability

A majority of the participants in this research evidenced conditioning of the skin resistance response to the paired nonsense syllable. However, statistically significant group differences in conditionability were absent. The groups did differ, with the HAP group being highest on this variable. The HAP and HAN groups were higher on this variable than the two low avoidance groups

(LAP and LAN) suggesting some relationship between conditionability and MIA avoidance. The MIA avoidance main effect did approach significance in the conditionability analysis of variance.

It might be the case that the conditioning processes involved in the development of agoraphobic avoidance differ in some way from those conditioning processes which are involved in the development of panic attacks. As noted in Chapter 1, the role of conditioning in agoraphobic avoidance seems, at least on the surface, to be more straightforward than in panic attacks. The former involves an association developing between some tangible environmental stimulus (e.g., the department store, the interstate, the shopping mall) and the panic state while the latter involves a complex chain of associations involving external stimuli, bodily states, behaviors (e.g., hyperventilating, muscle tightening), and cognitions or "covert/verbal behaviors." Of course, the observed relationship between MIA avoidance and conditionability could quite easily be due to chance and any interpretation of this relationship is speculative.

It had been hypothesized that stimulus sensitivity, response magnitude and habituation might have an influence on conditioning. These hypotheses were not put forward by either Eysenck or Gray but they do have intuitive appeal. It seems reasonable to assume that conditioning, (e.g., of an external stimulus such as a department store to bodily

sensations accompanying a panic attack) would be enhanced if the UCS bodily sensations (a) occurred more frequently (due to increased stimulus sensitivity (b) were more intense (due to increased responsivity), and (c) were more persistent and frequent (due to slowed habituation). It is important to note here that the above-mentioned relationships are not assumed to operate in a simple one to one manner. For example, high stimulus sensitivity might combine with high autonomic responsivity to contribute to producing more intense physiological responses. The hypothesized relationships between conditionability and the other dependent variables were assessed via the canonical correlation. Stimulus sensitivity did load moderately on the second canonical variable of the dependent variables (dep2) in the canonical correlation. This is the same canonical variable on which the index of conditionability (Student's *t* statistic) loaded heavily, suggesting that greater UCS intensity might have enhanced conditioning. Response magnitude loaded modestly on dep2, suggesting a possible influence of response magnitude on conditionability. Beyond this, there was little evidence of the influence of the above-mentioned variables on conditioning. It might be that the relationships do not in fact exist. It might, however, be the case that the effect sizes in the present research were simply too small for these relationships to emerge.

Awareness of the CS-UCS Relationship

Whether or not each participant was aware, and correctly so, of the CS-UCS relationship was assessed. This was done because it was possible that a significant amount of the variance in conditionability might have been accounted for by this variable had there been significant numbers of participants who were not aware of the relationship. Some researchers maintain that conditioning does not occur in humans without some awareness of the CS-UCS relationship. As it turned out, however, virtually all of the participants reported being aware of the CS-UCS relationship. It is interesting to note that of the four participants who said they were unaware of the CS-UCS relationship, two evidenced conditioning of the SR response. Thus, while awareness of the CS-UCS relationship may make conditioning more likely to occur, the present research demonstrated that it is possible to observe conditioning without such awareness.

Success of the Paradigm and Suggestions for Future Research

It appears that this experimental paradigm was fairly successful in achieving its goals despite the small effect sizes obtained and the absence of statistically significant differences for some of the predictions made. Having the stimuli for the entire procedure recorded on video tape, time locked to the polygraph, provided a high level of consistency in the manner in which the procedure was

experienced by the various participants and a good deal of accuracy in recording responses.

In future paradigms of this type, one might start the stimulus sensitivity series at a level higher than sixty five db as all participants tolerated at least eighty decibels. The stimulus sensitivity series might, however, be changed to go beyond one hundred decibels as a number of subjects continued all the way through one hundred decibels in each of the three series. It is possible that they would have tolerated louder tones had the series gone beyond 100 db. Tones at decibel levels up to one hundred and twenty db have been used in this type of research (Prokasy & Kumpfer, 1973). A modification to the method employed to assess stimulus sensitivity in the present research would be to present tones at the various levels in random order, having the subject indicate yes or no to whether each tone was aversive or not. This would eliminate participants from stopping the series prematurely because they anticipated that the next tone would be too loud for them. Some participants in the present research may have done this and may have evidenced poorer conditioning as a result. This modification would, of course, give participants less control over the sound levels they heard than they had in the present research.

The habituation series stimuli of eight white noise bursts of one second duration used in the present research

appeared to be relatively effective in eliciting SR responses which decreased in magnitude over time. Data analyses, however, were complicated by the confound between habituation and response magnitude. The results obtained in the present research make clear the difficulties in analyzing SR habituation data. In future studies, SR habituation to repeated stimulus presentations might be analyzed both in terms of (a) a regression coefficient derived from proportion of initial response data and (b) a count of responses to habituation stimuli. In this way, a better understanding of the benefits and drawbacks of each approach might be obtained. Examining decreasing SR responses to repeated presentations of a stimulus is one way to examine habituation. Another way to approach the phenomenon of habituation, and one which might be incorporated in a similar study, would be to present an audible stimulus such as white noise which comes on and remains on for several seconds. One could then examine (a) the slope of a line fit to the rate at which the response returns to baseline or (b) the time until the SR level returns to baseline. Of course, the habituation-response magnitude confound observed in the present research would still exist and would have to be addressed in some manner.

Given that subjects were permitted to select their own level of a tone that was "clearly unpleasant" and given that the stimulus intensities used in this study were relatively

mild, the classical conditioning procedure was reasonably effective in producing some evidence of the conditioned skin response in most participants. Because individuals vary so widely with regard to SR responding and the way in which the conditioned skin response is evidenced, the type of analysis used in the present research may well be superior to that used in the Welsh & Kubis (1947a, 1947b) and Vogel (1960b, 1961) studies upon which this paradigm was based. As noted previously, relatively weak stimulus intensities were used in the present research. It is suggested that in future research higher stimulus levels be utilized if possible. Some studies have used fixed levels of up to 120 db (Prokasy & Kumpfer, 1973). Of course, the greater the stimulus intensities, the greater the discomfort experienced by the participant. This dilemma will, unfortunately, be with us as long as research is done. Participants in the present research might have selected a relatively low stimulus intensity level because they knew they would be committing to that level for the rest of the study. A solution might be to employ some type of procedure where the participant, while instructed to keep his or her level of stimulus intensity at an unpleasant level, be permitted to self-adjust the intensity of the CS during the paradigm. This, of course, adds other variables (e.g., differences in motivation of participants) to an already complicated situation.

In the final version of the stimulus tapes used in this study there was a four second ISI with a one second UCS. If that interval were lengthened slightly more, perhaps to six or seven seconds, then conditioning trials could themselves serve as test trials and the procedure could be shortened considerably.

Eysenck and Gray both predict personality-related individual differences for operant as well as classical conditioning. While data collection for the present research was in progress, it was noted that a few of the participants, before they correctly discerned the nature of the CS-UCS relationship, were trying out different strategies in an apparent effort to "turn off" the UCS tone. These included not saying the CS syllable out loud, refraining from saying any syllables out loud for a given period of time, and varying the pronunciation of the CS syllable. This clearly demonstrates how this paradigm could easily be altered to include an operant task, giving the participants an opportunity to turn off the aversive tone.

Finally, results of the present research indicate that some individuals are more responsive than others with regard to SR responding. Activity of the autonomic nervous system can be assessed through "channels" or "modalities" other than SR responding (e.g., heartrate, respiration, blood pressure, muscle tension, etc.). A given individual may be relatively nonresponsive in a given channel (e.g., SR

responding) but may respond vigorously through another channel (e.g., heartrate). This suggests recording responses in several channels simultaneously. Because one entity, the autonomic nervous system, is assumed to underlie all of these response modalities, analysis of these multiple measures would appear to be well suited to multivariate methods. Such an approach would involve additional expense and time. However, the information obtained in this manner might be well worth the added time and expense.

Summary and Conclusions

To summarize, the present author believes that the present research succeeded on two levels. Panicker status and Mobility Inventory avoidance status were shown to be related to the Eysenck and Gray dimensions. This adds support to the case for applying these personality conceptualizations to the phenomena of panic and agoraphobic avoidance. The present author is not aware of a study examining the relationship between the Eysenck/Gray dimensions and panic/MIA Avoidance status in a clinical population. It would be of interest to compare the locations of groups of clinical panic/agoraphobia patients with the locations of the nonclinical panickers studied herein in the Eysenck/Gray personality space.

Second, differences were demonstrated between panickers and nonpanickers and those high and low on MIA avoidance in the extent to which they evidenced skin resistance

responding to various stimuli and in the rate at which they habituated to a novel stimulus. Additionally, these differences appear to be associated with Eysenck's dimension of neuroticism and Gray's dimension of trait anxiety. This suggests that these physiological variables may play a role in determining severity, and persistence of panic symptoms. As suggested previously, increased autonomic responsivity could result in increased physiological arousal in response to various stimuli. Slowed habituation would tend to keep the highly neurotic/anxious individual responding (e.g., with autonomic arousal) to a stimulus (e.g., loud noise) long after a less predisposed individual has come to ignore the stimulus. As noted previously, these variables, working together, could lead to a chronic state of overarousal which may set the stage for an initial panic attack and contribute to the occurrence of subsequent attacks and the development of agoraphobic avoidance.

The results would appear to support the continued study of nonclinical panickers and would also support the study of self reported agoraphobic avoidance among nonclinical panickers and controls. The latter has received little attention in the nonclinical panicker literature. Researchers involved in the nonclinical panicker literature, have noted that the criteria for status as a nonclinical panicker needs to become more stringent as we are hoping to be able to identify those individuals who may actually go on

to have significant difficulties with panic and agoraphobic avoidance in the future. In the present research, individuals in the high avoidant panicker group (HAP) differed from nonclinical panickers in the low avoidant group (LAP) on responsivity and slowed habituation. Nonclinical panickers high on agoraphobic discomfort/avoidance may represent a smaller more homogenous group of individuals who are particularly predisposed to developing panic disorder/agoraphobia. It is suggested that the phenomenon of nonclinical panic (in some individuals) may represent an early stage of development in the genesis of panic disorder/agoraphobia. There is evidence in support of this conceptualization of nonclinical panic.

Results from a number of studies suggest that nonclinical panickers are similar to clinical panic/agoraphobia patients with regard to (a) personality disorder symptomatology, (b) frequency and severity of panic symptoms, and (c) location in the Eysenck/Gray personality space.

First, there is similarity between nonclinical and clinical panickers in the types of DSM-III-R personality disorder symptomatology that they evidence. This was discussed in some detail in Chapter I (e.g., Friedman, Frances & Shear, 1987; Richman & Nelson-Gray, 1991).

Second, there is evidence that clinical panickers and college undergraduate nonclinical panickers are similar with

regard to the frequency and severity with which various panic symptoms are experienced. Cox, Endler, and Swinson (1991) administered the Panic Attack Questionnaire (Norton et al., 1985) to undergraduate nonclinical panickers and clinical panickers. These authors reported that the groups were similar with regard to which panic symptoms were experienced most frequently and most severely.

Third, there appears to be an overlap between clinical panickers/agoraphobics and the nonclinical panickers assessed in the present research on neuroticism, extroversion, and anxiety. Mavissakalian and Hamann (1986) divided 60 panic/agoraphobia patients into high and low trait groups based on number of DSM-III-R traits endorsed on the Personality diagnostic Questionnaire (Hyer, Rider, & Spitzer, 1978). High avoidant nonclinical panickers (HAP) in the present research were higher on neuroticism (mean=16.45) than Mavissakalian and Hamann's low trait group (mean=11.6) and just slightly lower on neuroticism than the high trait group (mean=17.4). High avoidant nonclinical panickers (HAP) were less extroverted (mean on extroversion=9.20) than the low trait group (mean=11.0), but more extroverted than the high trait group (mean=8.40). With regard to trait anxiety, Norton et al. (1986) reported a mean of 46.8 (sd=10.7) for 58 undergraduate nonclinical panickers on the STAI-T, while the mean on this measure for a sample of 93 anxiety disorder with panic outpatients

reported by Taylor, Koch, and Crockett (1991) was 49.8 (sd=9.2).

There appears to be somewhat less of an overlap between nonclinical panickers and clinical panickers/agoraphobics on MIA avoidance. Craske, Rachman, and Tallman (1986) reported that the mean on MIA avoidance for 30 agoraphobics with panic was 3.54 with a standard deviation of .82. The mean on MIA avoidance for the HAP group in the present research was 2.03 with a standard deviation of .36. This suggests that personality variables such as neuroticism and extroversion might predispose to the occurrence of panic attacks while severe agoraphobic avoidance might be a consequence of panic attacks.

Taken as a whole, the similarities between nonclinical and clinical panickers would appear to support the suggestion by Cox, Endler, and Swinson that nonclinical panickers occupy a location on an "anxiety continuum" intermediate to nonanxious controls and clinical panic disorder/agoraphobia patients. These similarities support the continued investigation of panic and related phenomena in nonclinical populations.

Research on nonclinical panickers may have implications for clinical applications such as early identification of those at high risk for developing panic disorder/agoraphobia. Despite the rapid growth in knowledge and awareness of panic disorder during the past decade, many

people who begin experiencing panic attacks will go to a number of physicians and specialists before finding their way to a psychologist or psychiatrist and being diagnosed with panic disorder. A greater understanding of the nonclinical panicker, including knowledge of personality variables, symptom profile, and physiological variables could be utilized in developing some type of brief screening procedure for individuals who present with symptoms that are suggestive of panic disorder. Such a screening could be used by general practitioners, school counselors, personnel directors and others who might encounter people with these complaints. In this way, a great deal of time and money could be saved. More importantly, an individual suffering from this disorder could be diagnosed and treated much earlier. The present author strongly believes that nonclinical panickers afford us a look at panic disorder and agoraphobia during their development. The development of a brief screening device would appear to be a worthwhile endeavor. A complete understanding of the nonclinical panicker would be invaluable in this effort.

With regard to treatment of clinical panickers/agoraphobics, the Eysenck/Gray conceptualizations can provide a viable framework within which the panic disorder/agoraphobia sufferer may better understand the biological precursors (e.g., high autonomic responsivity) and environmental influences (e.g., learning experiences)

which have contributed to his or her difficulties. The present author believes that providing a comprehensive understanding of the problem is one of the most important aspects of therapy. Many individuals who develop these problems embrace the "out of the blue" notion of panic and agoraphobia supported by Klein, Sheehan and other proponents of the "medical model." Such a belief leads to the view that medication alone can solve the problem. Further, such a view leaves many patients simply waiting for the disorder to "disappear" and "go back the way it came." The present author suggests that panic and agoraphobia do not develop out of the blue and are not linked to a specific "panic gene" which suddenly comes to life after age 17. Rather, the roots of panic and agoraphobia likely develop over a long period of time in an individual predisposed (i.e., neurotic/anxious and possibly introverted) to the development of these problems. Of those individuals with these problems that the present author has encountered, those who believe that their problems are due in part to their "nature" and believe that dealing with these problems will be an ongoing learning and coping process, tend to fare much better than those who do not take this position.

The Eysenck/Gray position appears to be compatible with this approach to panic/agoraphobia and warrants greater attention in basic and applied research in both nonclinical and clinical populations. It is suggested that we extend

the present personality/physiological variable research to clinical populations to (a) add support to the application of the Eysenck/Gray conceptualizations to the phenomena of panic and agoraphobic avoidance and (b) add support to the conceptualization of nonclinical panic as a precursor of the clinical disorder.

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Appendix A

DSM-III-R criteria for panic attacks and panic disorder

Diagnostic criteria for Panic Disorder

- A. At some time during the disturbance, one or more panic attacks (discrete periods of intense fear or discomfort have occurred that were
- (1) unexpected, i.e., did not occur immediately before or on exposure to a situation that almost always cause anxiety, and
 - (2) not triggered by situations in which the person was the focus of others' attention.
- B. Either four attacks, as defined in criterion A, have occurred within a four-week period, or one or more attacks have been followed by a period of at least a month of persistent fear of having another attack.
- C. At least four of the following symptoms developed during at least one of the attacks:
- (1) shortness of breath (dyspnea) or smothering sensations
 - (2) dizziness, unsteady feelings, or faintness
 - (3) palpitations or accelerated heart rate (tachycardia)
 - (4) trembling or shaking
 - (5) sweating
 - (6) choking
 - (7) nausea or abdominal distress
 - (8) depersonalization or derealization
 - (9) numbness or tingling sensations (paresthesias)
 - (10) flushes (hot flashes) or chills
 - (11) chest pain or discomfort
 - (12) fear of dying
 - (13) fear of going crazy or doing something uncontrolled

Note: Attacks involving four or more symptoms are panic attacks; attacks involving fewer than four symptoms are limited symptom attacks (see Agoraphobia without History of Panic Disorder, p.241).

**DSM-III-R criteria for panic attacks and panic disorder
(cont.)**

- D. During at least some of the attacks, at least four of the C symptoms developed suddenly and increased in intensity within ten minutes of the beginning of the first C symptom noticed in the attack.**
- E. It cannot be established that an organic factor initiated and maintained the disturbance, e.g., amphetamine or Caffeine Intoxication, hyperthyroidism.**

Note: Mitral valve prolapse may be an associated condition, but does not preclude a diagnosis of panic disorder

Appendix B**DSM-III-R Personality Disorder Clusters****Cluster A (odd/eccentric)**

1. paranoid personality disorder
2. schizoid personality disorder
3. schizotypal personality disorder

Cluster B (erratic/dramatic)

1. antisocial personality disorder
2. borderline personality disorder
3. histrionic personality disorder
4. narcissistic personality disorder

Cluster C (anxious/fearful)

1. avoidant personality disorder
2. dependent personality disorder
3. obsessive compulsive personality disorder
4. passive aggressive personality disorder

Appendix C

Panic Attack Questionnaire

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

If you have experienced one or more panic attacks in the past year please answer all of the remaining questions by CHECKING, CIRCLING, or FILLING IN the appropriate answer. If you have not had a panic attack in the past year please skip questions 1 to 23.

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 four weeks how many panic attacks have you had?
0 1 2 3 4 5 6 7 8 9 10 or more
 3. What is the greatest number of panic attacks you have had during any four week period in your life?
0 1 2 3 4 5 6 7 8 9 10 or more
 4. For how many months or years (approximately) have you been experiencing panic attacks?
_____years _____months
 5. How long ago was your worst attack?
_____years _____months _____weeks _____days
 6. Have you ever had a panic attack that was unexpected ("out of the blue")?
_____no _____yes
 7. If you answered "yes" to question number 6, please indicate the proportion of your panic attacks that are unexpected:
_____All _____Most _____Some _____Few _____None
 8. If you recall your first panic attack, please describe briefly the circumstances surrounding the attack (e.g., where you were, what you were doing).
-
-

9. How disturbing or distressing are your panic attacks?
 ___Not at all ___Mildly ___Moderately ___Very ___Extremely
10. To what degree have your panic attacks restricted or changed your lifestyle (e.g., activities you engage in, places you go)?
 ___No Change ___Some ___Moderately ___Quite a Bit ___Extreme Change
11. Do you avoid certain situations due to fear of having a panic attack?
 ___No ___Yes
12. If you answered "yes" to question 11, please indicate situations you avoid.
-

13. Please indicate how severely you experienced each of the following symptoms during your most recent panic attack and during your most severe panic attack.
- 0=Does not Occur 1=Mild 2=Moderate 3=Severe 4=Very Severe

	<u>Most Recent</u>				<u>Most Severe</u>					
a. Shortness of breath/ smothering sensation.....	0	1	2	3	4	0	1	2	3	4
b. Dizziness, unsteady feelings or faintness.....	0	1	2	3	4	0	1	2	3	4
c. Racing or pounding heart.....	0	1	2	3	4	0	1	2	3	4
d. Trembling or shaking.....	0	1	2	3	4	0	1	2	3	4
e. Sweating.....	0	1	2	3	4	0	1	2	3	4
f. Choking.....	0	1	2	3	4	0	1	2	3	4
g. Nausea or abdominal distress.	0	1	2	3	4	0	1	2	3	4
h. Feeling that things are not real.....	0	1	2	3	4	0	1	2	3	4
i. Numbness/tingling sensations.	0	1	2	3	4	0	1	2	3	4
j. Hot flashes or chills.....	0	1	2	3	4	0	1	2	3	4
k. Chest pains or discomfort....	0	1	2	3	4	0	1	2	3	4
l. Fear of dying.....	0	1	2	3	4	0	1	2	3	4
m. Fear of going crazy or losing control.....	0	1	2	3	4	0	1	2	3	4
n. Visual difficulties (e.g., blurring, tunnel vision)...	0	1	2	3	4	0	1	2	3	4
o. Hearing difficulties (e.g., difficulty hearing, ringing in ears).....	0	1	2	3	4	0	1	2	3	4
p. Difficulty concentrating.....	0	1	2	3	4	0	1	2	3	4
q. Desire to escape from scene of attack.....	0	1	2	3	4	0	1	2	3	4

- r. Thoughts or images that
you cannot get rid of.....0 1 2 3 4.....0 1 2 3 4
- s. Difficulty speaking.....0 1 2 3 4.....0 1 2 3 4
- t. Feelings of embarrassment.....0 1 2 3 4.....0 1 2 3 4

14. When a panic attack occurs, generally what is the time period between the onset of the attack and when the panic is most intense?
- Just a few minutes (0-10 minutes)
 - 10 to 30 minutes
 - 30 minutes to one-hour
 - Several hours
 - More than one day

15. Have any of your attacks developed suddenly and increased to peak intensity within 10 minutes of noticing the first symptom?
- _____ No _____ Yes

16. How long on average, does a panic attack last (from start to finish)?
- Just a few minutes (0-10 minutes)
 - 10 to 30 minutes
 - 30 minutes to one-hour
 - Several hours
 - More than one day

17. How much does the thought of future panic attacks concern you?
- No concern at all
 - I get mildly anxious
 - I get moderately anxious
 - I get very anxious
 - I get extremely anxious

18. How serious (either psychologically or medically) do you think your panic attacks are?

Not at all serious _____ Extremely Serious _____

0__ 1__ 2__ 3__ 4__

19. To what extent have you considered seeking treatment for your panic attacks?
- I have never considered seeking treatment
 - I have thought about seeking treatment, but not seriously
 - I have seriously thought about seeking treatment, but doubt that I will actually do so
 - I have seriously thought about seeking treatment and intend to do so in the future

e. I have asked for treatment in the past (or I am currently receiving treatment) specifically for panic attacks

20. Have you ever been told there is a medical reason for your attacks?
 _____ No _____ Yes If yes, what? _____

21. During an attack, have you ever lost control or done anything uncontrolled which you later regretted?
 _____ No _____ Yes If yes, explain _____

22. Please describe where you were at and what you were doing when you experienced your last three panic attacks (if you've had three or more) and indicate if the panic attack was expected in each situation.

	Expected	Unexpected
a. _____	_____	_____
b. _____	_____	_____
c. _____	_____	_____

23. To the best of your knowledge, have any of the following members of your family experienced panic attacks?

	Age	Yes	No	Don't Know	Not Applicable
Mother	_____	_____	_____	_____	_____
Father	_____	_____	_____	_____	_____
Sister(s)	_____	_____	_____	_____	_____
Brother(s)	_____	_____	_____	_____	_____
Daughter(s)	_____	_____	_____	_____	_____
Son(s)	_____	_____	_____	_____	_____

Appendix D

Mobility Inventory for Agoraphobia

1. Please indicate, in the column marked "DISCOMFORT," the degree to which you experience anxiety or nervousness in the places and situations described below. Use the following scale.

- | | |
|------------------------|----------------------------|
| 1. No discomfort | 4. Considerable discomfort |
| 2. Mild discomfort | 5. Extreme discomfort |
| 3. Moderate discomfort | |

2. Please indicate, in the column marked "AVOIDANCE," how often you actually avoid the places and situations described below because of anxiety-related discomfort. Use the following scale.

- | | |
|------------------------------|---------------------------|
| 1. Never avoid | 4. Avoid most of the time |
| 2. Rarely avoid | 5. Always avoid |
| 3. Avoid about half the time | |

(You may use numbers half-way between those listed if you think it is appropriate. For example, 3 1/2 or 4 1/2.)

Skip those situations that do not apply to you.

	DISCOMFORT	AVOIDANCE
Theaters	_____	_____
Supermarkets	_____	_____
Classrooms	_____	_____
Department Stores	_____	_____
Restaurants	_____	_____
Museums	_____	_____
Elevators	_____	_____
Auditoriums or stadiums	_____	_____
Parking garages	_____	_____
High places	_____	_____
Enclosed spaces (e.g. tunnels)	_____	_____
Open spaces	_____	_____
a) Outside (e.g. fields, wide streets, courtyards)	_____	_____
Staying at home alone	_____	_____
b) Inside (e.g. large rooms, lobbies)	_____	_____
Other (specify)	_____	_____
<u>Riding in</u>	_____	_____
Buses	_____	_____

Trains	_____	_____
Subways	_____	_____
Airplanes	_____	_____
Boats	_____	_____
<u>Driving/riding in a car</u>		
a) at any time	_____	_____
b) on expressways	_____	_____
<u>Situations</u>		
Crossing bridges	_____	_____
Parties/social gatherings	_____	_____
Walking on the street	_____	_____
Staying at home alone	_____	_____
Being far away from home	_____	_____

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**129-130,
132-136**

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Appendix F

State-Trait Anxiety Inventory (Form Y)

SELF EVALUATION QUESTIONNAIRE
STAT Form Y-2

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate 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.

- 1 = ALMOST NEVER**
2 = SOMETIMES
3 = OFTEN
4 = ALMOST ALWAYS

	1	2	3	4
1. I feel pleasant.....	0	0	0	0
2. I feel nervous and restless.....	0	0	0	0
3. I feel satisfied with myself.....	0	0	0	0
4. I wish I could be as happy as others seem to be.....	0	0	0	0
5. I feel like a failure.....	0	0	0	0
6. I feel rested.....	0	0	0	0
7. I am "calm, cool, and collected".....	0	0	0	0
8. I feel that difficulties are piling up so that I cannot overcome them.....	0	0	0	0
9. I worry too much over something that really doesn't matter.....	0	0	0	0
10. I am happy.....	0	0	0	0
11. I have disturbing thoughts.....	0	0	0	0
12. I lack self-confidence.....	0	0	0	0
13. I feel secure.....	0	0	0	0
14. I make decisions easily.....	0	0	0	0
15. I feel inadequate.....	0	0	0	0
16. I am content.....	0	0	0	0
17. Some unimportant thought runs through my mind and bothers me.....	0	0	0	0
18. I take disappointments so keenly that I can't put them out of my mind.....	0	0	0	0
19. I am a steady person.....	0	0	0	0
20. I get in a state of tension or turmoil as I think over my recent concerns and interests...	0	0	0	0

Appendix J

Nonsense Syllable Stimuli

<u>Syllable</u>	<u>Glaze value</u>	<u>Syllable</u>	<u>Glaze</u>
BEH.....	20	NAH.....	13
BYV.....	13	NUB.....	13
CEF.....	0	PAF.....	13
CYJ.....	20	PYB.....	7
DAQ.....	7	QAP.....	0
DAX.....	0	QEM.....	7
FEH.....	0	RIX.....	13
FOJ.....	20	RYX.....	13
GAX.....	0	SEB.....	13
GEF.....	13	SIJ.....	0
HEG.....	20	TAH.....	20
HUC.....	7	TUV.....	13
JAT.....	20	VAF.....	0
JEC.....	13	VIB.....	13
KAJ.....	13	WAJ.....	20
KEB.....	7	WEZ.....	7
LAJ.....	0	YAB.....	13
LIW.....	20	YAZ.....	7
MEC.....	7	ZAJ.....	7
MIB.....	7	ZAS.....	13

Appendix K

Phase 3 Nonsense Syllable Series

Position	Syllable	Position	Syllable
1	KAJ	2	WEZ
3	GAX+	4	TAH
5	FEH	6	NUB
7	FOJ	8	GAX +
9	KEB	10	YAB
11	TAH	12	LIW
13	GAX -	14	HUC
15	GAX	16	JEC
17	FEH	18	GAX +
19	SEB	20	FEH
21	GAX +	22	HEG
23	GAX -	24	GEF
25	MIB	26	PAF
27	LAJ	28	GAX -
29	CYJ	30	TAH
31	GAX +	32	BEH
33	FEH	34	KAJ
35	GAX -	36	PAF
37	GEF	38	HEG
39	GAX +	40	LAJ
41	FEH	42	GAX -
43	MIB	44	GAX-
45	KAJ	46	GAX +
47	JEC	48	FEH
49	GAX -	50	YAB
51	HUC	52	GAX -
53	TAH	54	GAX +
55	FEH	56	BEH
57	FOJ	58	GAX +
59	LIW	60	WEZ
61	SEB	62	GAX -
63	TAH	64	FEH
65	KEB	66	GAX +
67	TAH	68	KAJ
69	CYJ	70	NUB
71	GAX +	72	FEH
73	SEB	74	TAH
75	KAJ	76	GAX -
77	WEZ	78	LIW
79	GAX -	80	KEB
81	FEH	82	NUB
83	FOJ	84	LAJ
85	MIB	86	GAX +
87	YAB	88	GAX -

89	TAH	90	BEH
91	GAX -	92	JEC
93	GAX +	94	GEF
95	PAF	96	GAX +
97	FEH	98	GAX -
99	CYJ	100	TAH
101	HEG	102	HUC
103	GAX +	104	FEH
105	KAJ	106	LAJ
107	JEC	108	GAX +
109	BEH	110	GAX -
111	FEH	112	GAX -
113	KEB	114	HUC
115	GAX -	116	FEH
117	GAX +	118	TAH
119	GAX -	120	YAB
121	KAJ	122	FOJ
123	MIB	124	GAX +
125	WEZ	126	PAF
127	GAX +	128	NUB
129	FEH	130	TAH
131	GAX -	132	KAJ
133	HEG	134	TAH
135	GAX +	136	CYJ
137	LIW	138	GEF
139	JEC	140	SEB

+ = conditioning trials
- = test trials

Appendix L

Written presentation

You have been asked to participate in this study based on your responses to a questionnaire you filled out during mass screening. If you choose to participate in this study, you will receive one credit towards your Psychology 221 research requirement. The procedure should take between 30 and 40 minutes. The study involves two tasks. The first one involves listening to several series of tones of various frequencies and volumes over a set of headphones while we measure the sweat activity of your hand. This is done by attaching two sensors to two fingers of one of your hands with tape. The sensors are completely safe and there is no discomfort involved. You will be asked to rate the loudness of the various tones.

After a break of about ten minutes we will begin the second task. The second task involves your reading out loud to yourself a series of "nonsense syllables" which will appear on a television monitor while you are seated in a comfortable chair. We will again measure the sweat activity of your hand while you read these syllables. We need to take our measurements while you are completely relaxed. Reading and saying the syllables will help keep you from thinking about anything that might disturb you. You will again wear headphones and you may hear some tones as you read the words. You may be asked some questions about the syllables you saw and the sounds you heard at the conclusion of your participation.

You are free to withdraw from the experiment at any time. If you choose to do so no negative consequences will accompany this decision and you will still receive credit for participating in the study. You may ask questions about the study. These will be answered before you participate as long as doing so will not bias the data collected in the study. If a particular question cannot be answered prior to your participation in the study it will be answered afterward.

Confidentiality will be maintained with regard to any

information obtained in this study. Subjects will be assigned a number and assistants who will come in contact with this data will use only these numbers for identification. Data will remain with the principal researcher until such time as it is not needed. At this time it will be appropriately disposed of.

Appendix M

THE UNIVERSITY OF NORTH CAROLINA AT GREENSBORO
Consent to Act as a Human Subject
 (Short Form)

Subject's Name _____
 Date of Consent _____

I hereby consent to participate in the research project entitled Response to Nonsense Syllables and Tones During Relaxation. An explanation of the procedures and/or investigations to be followed and their purpose, including any experimental procedures, was provided to me by Harvey Richman or associate. I was also informed about any benefits, risks, or discomforts that I might expect. I was given the opportunity to ask questions regarding the research and was assured that I am free to withdraw my consent to participate in the project at any time without penalty or prejudice. I understand that I will not be identified by name as a participant in this project.

I have been assured that the explanation I have received regarding this project and this consent form have been approved by the University Institutional Review Board which ensures that research projects involving human subjects follow federal regulations. If I have any questions about this, I have been told to call the Office of Research Services at (919)334-5878.

I understand that any new information that develops during the project will be provided to me if that information might affect my willingness to continue participation in the project. In addition, I have been informed of the compensation/treatment or the absence of compensation/treatment should I be injured in this project.

 Subject's Signature

 Witness to Oral Presentation
 and Signature of Subject

If subject is a minor or for some other reason unable to sign, complete the following:

Subject is _____ years old or unable to sign because _____

 Parent(s)/Guardian Signature

SHORT FORM 1/90

Appendix N

Instructions

Instructions Preceding Phases 1 and 2

You are going to listen to several series of tones as you sit in the chair and I measure the sweat activity of your hand. We will need to wait a few minutes for the sensor readings to stabilize. You will then hear three series of tones. When the tones begin, just relax and listen. I would like you to indicate when the volume of the tones has reached a level that is clearly unpleasant but at which you would be willing to hear these tones, or similar tones, a number of times during the remainder of the experiment. Indicate this by pressing the button by your right (or left) hand. Pressing the button will signal me in the adjoining room and I will stop that series of tones. Do the same for the next two series of tones. A prompt will appear on the television monitor (e.g., SERIES 1) to help you keep track of which series you are hearing. After the third series of tones ends, the television monitor will shut off. This is to remind you that during the next group of sounds you will hear, you should at no time press the button. Just close your eyes and listen to the sounds. When you are done listening to these sounds, we will take a short break before proceeding.

Instructions Preceding Phase 3

I am going to re-attach the sensors and give you the headphones as before. We will again need to allow a few minutes for the sensor readings to stabilize. You are going to see a series of nonsense syllables appear on the television monitor. Say each syllable out loud one time as it appears.

This is so that you will not think about anything other than the syllables. I will be in the adjoining room monitoring the sensor readings. You may hear some tones over the headphones as you say the syllables. Just continue reading the syllables out loud. You may be asked some questions regarding the syllables you have seen and the tones you heard at the end of the experiment. You will see a message on the television monitor letting you know when the experiment is over. I will be in shortly after that.

Appendix O

Post Experimental Questionnaire

1. Were you aware of a relationship between the tone and a particular nonsense syllable or syllables?

____ Yes

____ No

2. If so, what was the syllable(s)?

Write your answer(s) here. _____

Appendix P

Debriefing

Thank you for participating in this study. It was not possible to disclose, in detail, the exact goals of this study prior to your participation as this might have affected your responses. The first goal of this study was to collect data on the types of personality traits that are common to individuals who do and do not experience panic attacks. Clinical psychologists are often interested in gathering this type of information. This data was obtained through the questionnaires that you filled out during mass screening. The second goal of this study was to assess how different personality types may affect (a) physiological reactions to stimuli such as the tones and static sounds you heard and (b) how readily individuals learn associations between stimuli (the tones and a particular syllable). These issues will be examined by comparing subjects with differing personality types on (a) the volume level of tones tolerated, (b) skin response to those tones and static sounds, (c) how quickly participants got used to hearing the static sounds, and (d) how quickly participants learned to associate one of the syllables with a tone.

The primary independent variable in this experiment was group or personality type. Group assignment was based on responses to the Panic Attack Questionnaire and the Mobility Inventory for Agoraphobia which you completed as part of this study. Several dependent variables were used in this experiment: (a) the volume level of tones tolerated, (b) skin response to those tones and static sounds, (c) how quickly participants got used to hearing the static sounds, and (d) how quickly participants learned to associate one of the words with the tones.

We very much appreciate your participation as the information obtained through this study may add to our understanding of the role that physiological responses and learning processes may play in certain types of human behavior. We ask that you do not discuss this study with your fellow students as some of them might also be participants in this study.

Appendix Q

Tables

Table 1

Participant Demographics by Group

Age				
	LAN	HAN	LAP	HAP
Mean	19.21	20.10	20.30	20.95
Minimum	18.00	18.00	18.00	18.00
Maximum	22.00	38.00	35.00	37.00

Race				
	LAN	HAN	LAP	HAP
White	19	19	18	18
Black	1	0	2	1
Other	-	1	-	1
Total n	20	20	20	20

Sex				
	LAN	HAN	LAP	HAP
Female	16	13	14	16
Males	04	07	06	04
Total n	20	20	20	20

Table 2

Descriptive Statistics on Personality
and Dependent Variable Data by Group

Low Avoidant Nonpanickers ^a

Variable	Mean	Std. Dev.	Minimum	Maximum
Neuroticism	7.70	3.40	3.00	15.00
Extroversion	14.30	4.01	7.00	23.00
Anxiety Index	-1.05	.91	-2.21	1.27
Impulsivity Index	-.39	1.40	-2.52	3.73
MIA Avoidance	.89	.31	0.00	1.11
Stimulus Sens.	86.42	7.85	71.70	100.00
SR Baseline	470.95	189.41	227.00	800.00
SR Response Mag.	-2.69	4.94	-20.95	1.40
SR Response Slope	-1.13	1.53	-6.23	.24
SR Hab. (slope)	-571.70	803.50	-2,315.00	464.00
SR Hab. (count)	2.10	1.94	0.00	6.00
Cond. (t-stat.)	3.10	1.42	.29	5.34

^a n = 20

High Avoidant Nonpanickers ^a

Variable	Mean	Std. Dev.	Minimum	Maximum
Neuroticism	12.20	4.66	1.00	19.00
Extroversion	11.60	4.44	3.00	18.00
Anxiety Index	-.34	.93	-1.70	1.94
Impulsivity Index	.04	.94	-1.92	1.52
MIA Avoidance	2.10	.24	1.81	2.89
Stimulus Sens.	87.75	7.45	78.30	100.00
SR Baseline	402.15	163.40	218.00	771.00
SR Response Mag.	-3.75	3.24	-10.60	1.19
SR Response Slope	-1.61	1.28	-4.43	.03
SR Hab. (slope)	-689.50	1,712.13	-4,071.00	4,167.00
SR Hab. (count)	2.75	2.67	0.00	8.00
Cond. (t-stat.)	3.38	1.93	-0.90	6.43

^a n = 20

Table 2 (Cont.)

Low Avoidant Panickers ^a

Variable	Mean	Std. Dev.	Minimum	Maximum
Neuroticism	11.80	5.15	5.00	24.00
Extroversion	12.25	5.04	5.00	22.00
Anxiety Index	-.04	1.25	-1.77	2.13
Impulsivity Index	.03	1.39	-1.68	3.29
MIA Avoidance	1.06	.07	.93	1.11
Stimulus Sens.	87.63	7.80	73.00	100.00
SR Baseline	341.30	126.98	206.00	795.00
SR Response Mag.	-5.17	4.38	-17.92	0.05
SR Response Slope	-2.25	1.75	-5.74	-0.01
SR Hab. (slope)	-861.00	2,459.36	-5,143.00	7,851.00
SR Hab. (count)	3.10	2.75	0.00	8.00
Cond. (t-stat.)	2.54	1.02	0.89	4.66

^a n = 20High Avoidant Panickers ^a

Variable	Mean	Std. Dev.	Minimum	Maximum
Neuroticism	16.45	3.49	9.00	22.00
Extroversion	9.20	6.13	1.00	20.00
Anxiety Index	1.29	1.01	-0.71	2.69
Impulsivity Index	.07	1.38	-2.22	2.29
MIA Avoidance	2.03	.36	1.67	2.97
Stimulus Sens.	86.35	7.18	76.70	100.00
SR Baseline	435.55	172.49	222.00	803.00
SR Response Mag.	-7.05	6.34	-21.80	0.15
SR Response Slope	-2.96	2.62	-7.62	0.04
SR Hab. (slope)	-1,603.60	1,974.18	-5,452.00	631.00
SR Hab. (count)	4.40	2.46	0.00	8.00
Cond. (t-stat.)	3.44	1.80	-1.03	5.82

^a n = 20

Table 3

Pearson Correlations Among the Dependent Variables *

	Stim. Sens.	Response Mag.	Response Slope	Hab. (Slope)	Hab. (Count)	Cond.
Base- line	-.03 (.7714)	.05 (.6581)	.11 (.3419)	.05 (.6899)	-.15 (.1960)	-.13 (.2542)
Stim. Sens.		.14 (.2342)	.12 (.2746)	-.10 (.4043)	.006 (.9590)	.04 (.7455)
Response Mag.			.93 (.0001)	.59 (.0001)	-.35 (.0014)	.01 (.9235)
Response Slope				.56 (.0001)	-.38 (.0004)	-.01 (.9033)
Hab. (Slope)					-.29 (.0095)	-.001 (.9924)
Hab. (Count)						.07 (.5452)
Cond.						

* N=80

Table 4

Analysis of Variance Assessing Relationship Between
Panicker/MIA Avoidance Status and Neuroticism

Source	Type III SS	df	F	p
Panicker Status	348.6125	1	19.37	<.0001
Avoidance Status	418.6125	1	23.26	<.0001
Panicker*Avoidance	.1125	1	.01	.9372
Error	1,367.5500	76		
Total	2,134.8875	79		

A Posteriori Group Comparisons on Neuroticism ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	16.45 (3.49)	A
LAP	11.80 (5.15)	B
HAN	12.20 (4.66)	B
LAN	7.70 (3.40)	C

Least significant difference = 2.67

^a df for all tests = 76

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 5

Analysis of Variance Assessing Relationship Between
Panicker/MIA Avoidance Status and Extroversion

Source	Type III SS	df	F	p
Panicker Status	99.0125	1	4.01	.0488
Avoidance Status	165.3125	1	6.70	.0116
Panicker*Avoidance	.6125	1	.02	.8752
Error	1,875.9500	76		
Total	2,140.8875	79		

A Posteriori Group Comparisons on Extroversion ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	9.20 (6.13)	B
LAP	12.25 (5.04)	A B
HAN	11.60 (4.44)	A B
LAN	14.30 (4.02)	A

Least significant difference = 3.13

^a df for all tests = 76

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 6

Analysis of Variance Assessing Relationship Between
Panicker/MIA Avoidance Status and Anxiety

Source	Type III SS	df	F	p
Panicker Status	34.5961	1	32.50	<.0001
Avoidance Status	20.8968	1	19.63	<.0001
Panicker*Avoidance	1.9066	1	1.79	.1847
Error	80.8906	76		
Total	138.2901	79		

A Posteriori Group Comparisons on Anxiety ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	1.29 (1.01)	A
LAP	-0.04 (1.25)	B
HAN	-0.34 (0.93)	B
LAN	-1.05 (0.91)	C

Least significant difference = .65

^a df for all tests = 76

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 7

Analysis of Variance Assessing Relationship Between
Panicker/MIA Avoidance Status and Impulsivity

Source	Type III SS	df	F	p
Panicker Status	1.0067	1	.60	.4400
Avoidance Status	1.1215	1	.67	.4151
Panicker*Avoidance	.7583	1	.45	.5025
Error	126.9571	76		
Total	129.8437	79		

A Posteriori Group Comparisons on Impulsivity ^a ^b

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	.07 (1.38)	A
LAP	.03 (1.39)	A
HAN	.04 (.94)	A
LAN	-.39 (1.40)	A

Least significant difference = .81

^a df for all tests = 76

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 8

Analysis of Variance for Main and Interactive Effects
of Panicker Status and MIA Avoidance Status
on Stimulus Sensitivity

Source	Type III SS	df	F	p
Panicker Status	14.7623	1	.26	.6135
Avoidance Status	17.6890	1	.31	.5804
Panicker*Avoidance	34.0605	1	.59	.4435
Error	4,362.0670	76		
Total	4,396.3020	79		

A Posteriori Group Comparisons on Stimulus Sensitivity ^a ^b

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	86.35 (7.12)	A
LAP	87.63 (7.80)	A
HAN	87.75 (7.45)	A
LAN	86.42 (7.85)	A

Least significant difference = 4.80

^a df for all tests = 76

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 9

Analysis of Covariance for Main and Interactive Effects
of Panicker Status and MIA Avoidance Status
on Response Magnitude

Source	Type III SS	df	F	p
Baseline	1.3046	1	.05	.8158
Panicker Status	158.3631	1	6.64	.0120
Avoidance Status	43.7566	1	1.83	.1798
Panicker*Avoidance	4.2871	1	.18	.6729
Error	1789.6605	75		
Total	2003.9121	79		

A Posteriori Group Comparisons on Response Magnitude ^a ^b

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-7.05 (6.33)	B
LAP	-5.17 (4.38)	A B
HAN	-3.75 (3.24)	A
LAN	-2.69 (4.94)	A

Least Significant Difference = 3.08

^a df for all tests = 75

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 10

Analysis of Covariance for Main and Interactive Effects
of Panicker Status and MIA Avoidance
Status on Response Slope

Source	Type III SS	df	F	p
Baseline	1.8439	1	.53	.4702
Panicker Status	27.6170	1	7.89	.0063
Avoidance Status	7.3783	1	2.11	.1507
Panicker*Avoidance	.6729	1	.19	.6623
Error	262.5061	75		
Total	302.1447	79		

A Posteriori Group Comparisons on Response Slope ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-2.96 (2.62)	A
LAP	-2.25 (1.75)	B
HAN	-1.61 (1.28)	C
LAN	-1.13 (1.53)	D

Least Significant Difference = .4553

^a df for all tests = 75

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 11

Revised Analysis of Covariance for Main and Interactive
Effects of Panicker Status and MIA Avoidance
Status on Response Slope

Source	Type III SS	df	F	p
Baseline	.9093	1	1.74	.1910
Response Magnitude	223.8614	1	428.67	<.0001
Panicker Status	.5945	1	1.14	.2894
Avoidance Status	.1386	1	.27	.6080
Panicker*Avoidance	.0077	1	.01	.9035
Error	38.6448	74		
Total	302.1447	79		

A Posteriori Group Comparisons on Response Slope ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-2.96 (2.62)	A
LAP	-2.25 (1.75)	B
HAN	-1.61 (1.28)	C
LAN	-1.13 (1.53)	D

Least Significant Difference = .4553

^a df for all tests = 74

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 12

Analysis of Covariance for Main and Interactive Effects
of Panicker Status and MIA Avoidance Status
on Habituation Slope

Source	Type III SS	df	F	p
Baseline	646,562.6816	1	.19	.6648
Panicker Status	6,461,318.0792	1	1.89	.1732
Avoidance Status	3,819,115.0385	1	1.12	.2938
Panicker*Avoidance	2,408,849.5278	1	.70	.4038
Error	256,287,413.3180	75		
Total	269,828,149.8000	79		

A Posteriori Group Comparisons on Habituation Slope ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-1603 (1974)	B
LAP	-861 (246)	A B
HAN	-689 (1712)	A B
LAN	-571 (893)	A

Least Significant Difference = 968.64

^a df for all tests = 75

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 13

Revised Analysis of Covariance for Main and Interactive
Effects of Panicker Status and MIA Avoidance
Status on Habituation Slope

Source	Type III SS	df	F	p
Baseline	313,920.544	1	.13	.7166
Response Magnitude	81,405,968.740	1	34.45	<.0001
Panicker Status	18,527.273	1	.01	.9297
Avoidance Status	288,298.970	1	.12	.7279
Panicker*Avoidance	1,230,152.796	1	.52	.4729
Error	174,881,444.578	74		
Total	269,828,149.800	79		

A Posteriori Group Comparisons on Habituation Slope ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-1603 (1974)	B
LAP	-861 (246)	A B
HAN	-689 (1712)	A B
LAN	-571 (893)	A

Least Significant Difference = 968.64

^a df for all tests = 74

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 14

Analysis of Covariance for Main and Interactive Effects of
Panicker Status and MIA Avoidance Status on Habituation
Utilizing a Proportion of Initial Response

Source	Type III SS	df	F	p
Baseline	1,000.4048	1	.00	.9826
Response Magnitude	1,131,160.2189	1	.54	.4650
Panicker Status	940,999.3953	1	.45	.5050
Avoidance Status	870,229.3589	1	.42	.5214
Panicker*Avoidance	102,203.3644	1	.05	.8259
Error	155,151,294.9881	74		
Total	158,134,014.1875	79		

A Posteriori Group Comparisons on Habituation Utilizing
a Proportion of Initial Response ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	-230.30 (237.88)	A
LAP	-560.65 (797.88)	A
HAN	-157.40 (413.69)	A
LAN	-322.95 (411.95)	A

Least Significant Difference = 912.37

^a df for all tests = 74

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 15

Analysis of Covariance for Main and Interactive Effects
of Panicker Status and MIA Avoidance Status
on Habituation Count

Source	Type III SS	df	F	p
Baseline	9.8414	1	1.62	.2074
Panicker Status	29.1052	1	4.78	.0318
Avoidance Status	20.0810	1	3.30	.0733
Panicker*Avoidance	4.7567	1	.78	.3794
Error	456.3086	75		
Total	522.3875	79		

A Posteriori Group Comparisons on Habituation Count ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	4.40 (2.46)	A
LAP	3.10 (2.75)	A B
HAN	2.75 (2.67)	B
LAN	2.10 (1.94)	B

^a df for all tests = 75

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 16

Analysis of Covariance for Main and Interactive Effects
 Effects of Panicker Status and MIA Avoidance
 Status on Conditionability

Source	Type III SS	df	F	p
Baseline	6.4260	1	2.62	.1095
Panicker Status	2.2312	1	.91	.3430
Avoidance Status	7.5090	1	3.07	.0841
Panicker*Avoidance	3.9276	1	1.60	.2093
Error	183.7211	75		
Total	200.3670	79		

A Posteriori Group Comparisons on Conditionability ^{a b}

Group	Mean (std. dev.)	Fisher's LSD Test
HAP	3.44 (1.80)	A
LAP	2.53 (1.02)	A
HAN	3.38 (1.93)	A
LAN	3.10 (1.42)	A

Least Significant Difference = .986

^a df for all tests = 75

^b Group means with the same letter did not differ at the .05 level of statistical significance.

Table 17

Kolmogorov-Smirnov Statistics and Associated
Probability Values for Conditioning
Versus Random Sample Comparisons

LAN Group			HAN Group		
Participant	K-S	p	Participant	K-S	p
1	.56	.0006	1	.73	<.0001
2	.64	<.0001	2	.75	<.0001
3	.28	.2657	3	.65	<.0001
4	.63	<.0001	4	.35	.1057
5	.48	.0049	5	.48	.0049
6	.55	.0006	6	.38	.0396
7	.38	.0470	7	.30	.1813
8	.38	.0470	8	.30	.1813
9	.63	<.0001	9	.25	.3752
10	.35	.0763	10	.38	.0470
11	.30	.1813	11	.30	.1813
12	.42	.0181	12	.45	.0090
13	.60	<.0001	13	.53	.0013
14	.30	.1813	14	.38	.0470
15	.50	.0025	15	.60	<.0001
16	.23	.5095	16	.63	<.0001
17	.45	.0090	17	.45	.0090
18	.28	.2656	18	.40	.0281
19	.23	.5095	19	.50	.0025
20	.72	.0001	20	.58	.0003

Table 17 (Cont.)

LAP Group			HAP Group		
Participant	K-S	p	Participant	K-S	p
1	.48	.0049	1	.60	<.0001
2	.43	.0162	2	.60	<.0001
3	.50	.0025	3	.55	.0006
4	.30	.1813	4	.53	.0013
5	.30	.1813	5	.25	.3752
6	.35	.0763	6	.13	.9853
7	.33	.1196	7	.48	.0033
8	.23	.5095	8	.23	.5095
9	.40	.0281	9	.50	.0025
10	.60	<.0001	10	.60	.0001
11	.28	.2656	11	.38	.0470
12	.33	.1196	12	.63	<.0001
13	.38	.0470	13	.65	<.0001
14	.35	.0893	14	.35	.0763
15	.55	.0006	15	.20	<.0001
16	.48	.0049	16	.35	.6604
17	.43	.0162	17	.25	.0763
18	.33	.1196	18	.20	.6604
19	.63	<.0001	19	.43	.0162
20	.38	.0470	20	.55	.0006

Table 18

**Canonical Correlation Between Personality Variables
and Dependent Variables**

F Tests for Canonical Variable Pairs

Variable Pair	Num. df	Den. df	F	p
1	20	236.4302	.84	.6682
2	12	190.7856	.37	.9720
3	6	146.0000	.21	.9741
4	2	74.0000	.23	.7925

**Multivariate F Test for Overall Personality-Dependent
Variable Relationships**

Test Statistic	Num. df	Den. df	F	p
Roy's Greatest Root	5	74	2.66	.0286

Personality Variable - Canonical Variable Correlations

	Per1	Per2	Per3	Per4
Neuroticism	.60	.76	.27	-.02
Extroversion	-.89	.05	.18	-.41
Anxiety	.79	.22	.53	-.23
Impulsivity	-.22	.17	.90	.32

Table 18 (Cont.)

Dependent Variable - Canonical Variable Correlations

	Dep1	Dep2	Dep3	Dep4
Baseline	.42	-.08	-.54	.48
Stimulus Sens.	-.02	.50	-.67	-.52
Response Mag.	-.45	-.27	-.56	.39
Hab. Count	.79	.17	.29	-.08
Conditionability	-.21	.81	.23	.47

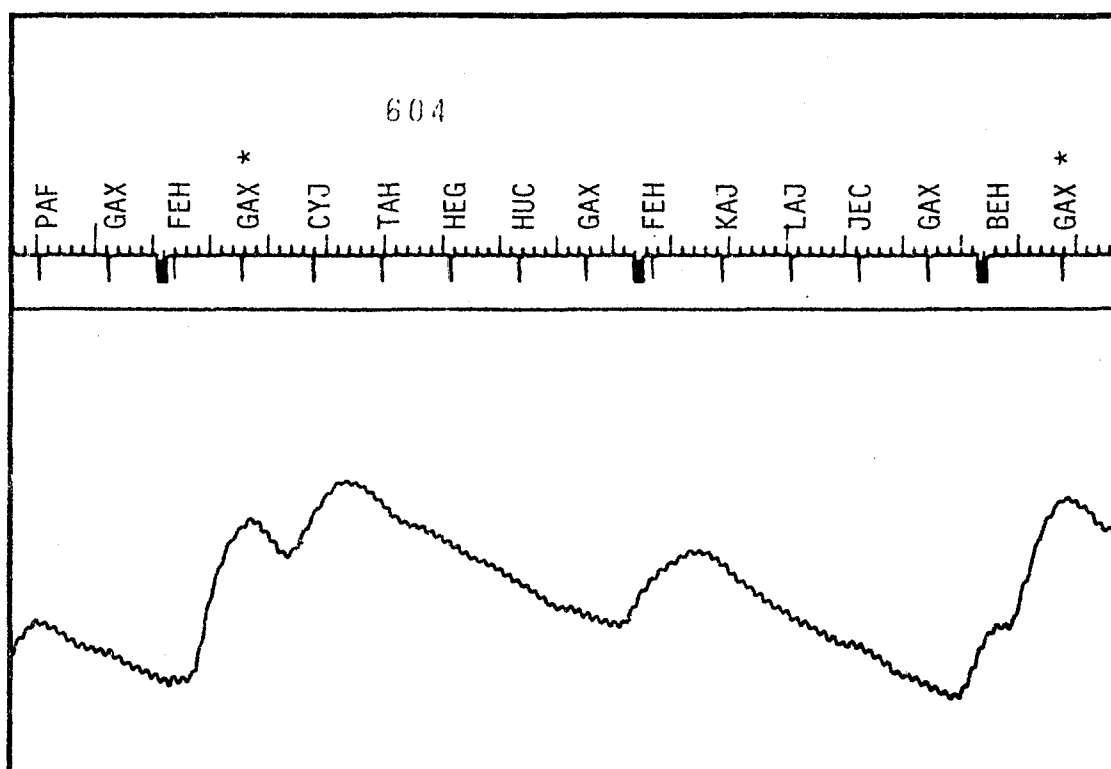
Correlations Between the Dependent Variables and the Canonical Variables of the Personality Variables

	Per1	Per2	Per3	Per4
Baseline	.16	-.02	-.06	.04
Stimulus Sens.	-.01	.10	-.07	-.04
Response Mag.	-.18	-.06	-.06	.03
Hab. Count	.31	.04	.03	-.01
Conditionability	-.08	.17	.02	.04

Appendix R

Figures

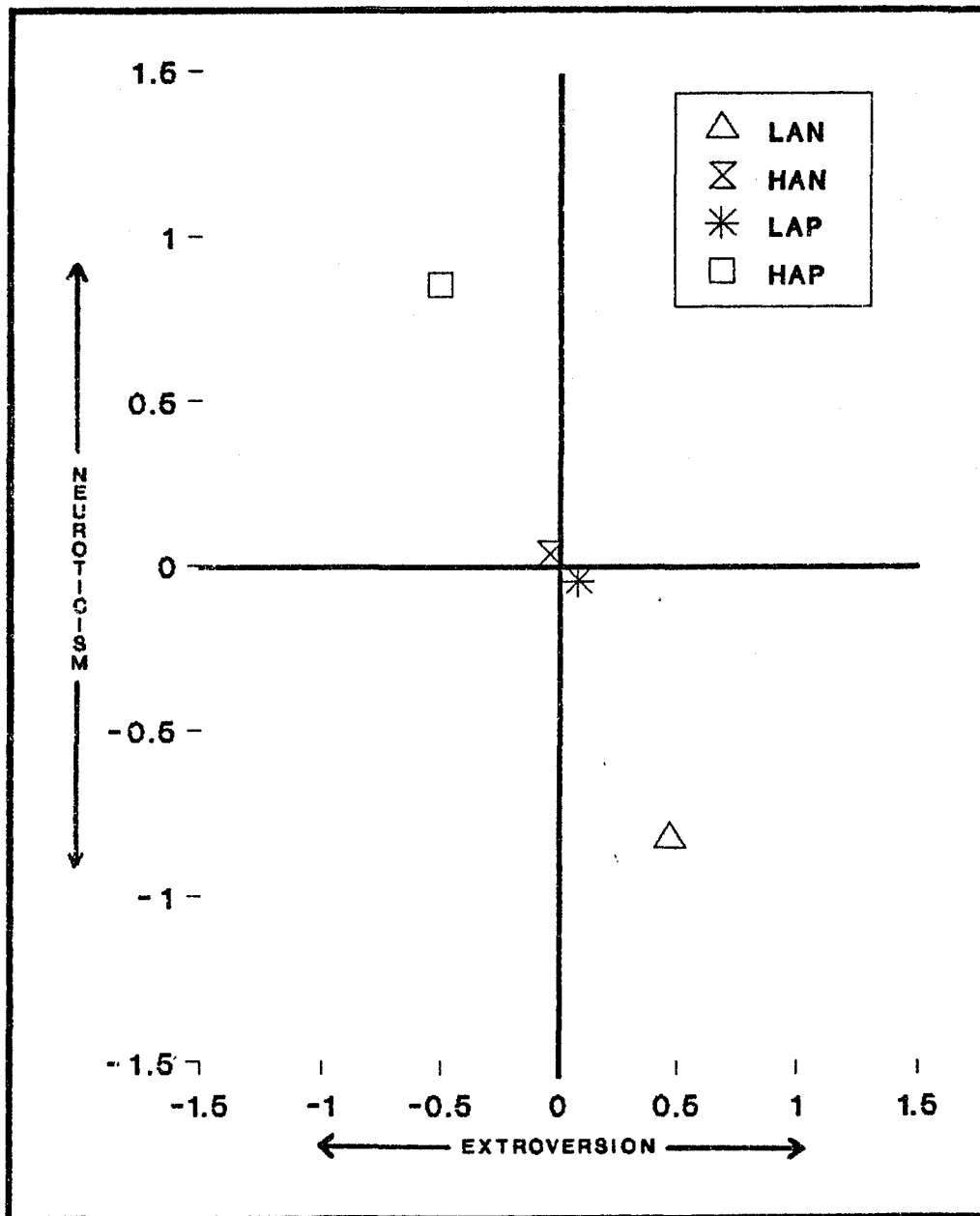
Figure 1

Sample SR Conditioning Record ^{a b c}

- ^a Each downward pen deflection at the top of the record indicates the appearance of a new syllable.
- ^b Each darker, wider downward pen deflection indicates the UCS tone.
- ^c Test trials are marked with an *

Figure 2

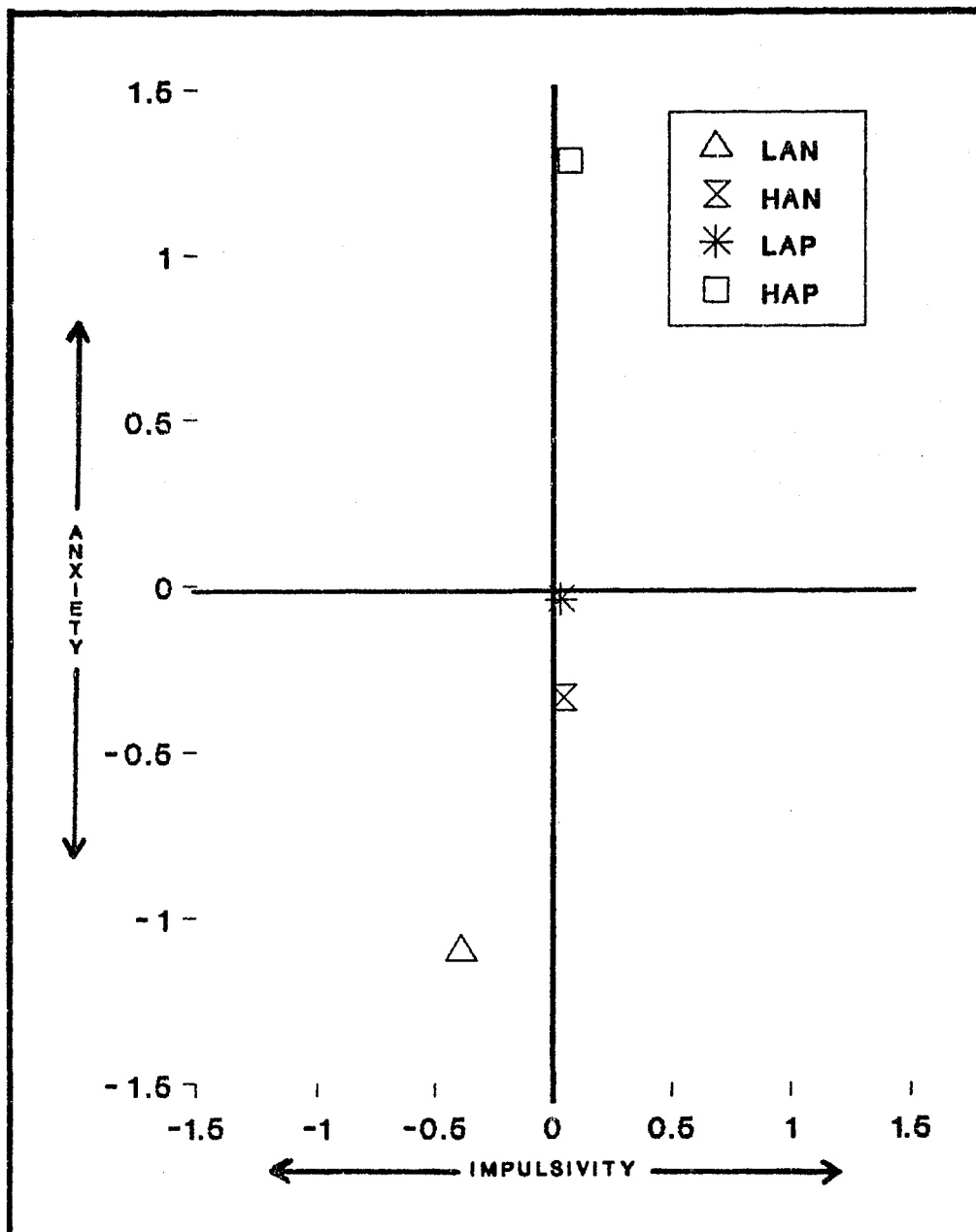
Experimental Groups Plotted as a Function of Their
Standardized Means on the Eysenck Dimensions ^a



^a Mean = 0, Std. Dev. = 1

Figure 3

Experimental Groups Plotted as a Function of Their
Standardized Means on the Gray Dimensions *



* Mean = 0, Std. Dev. = 1