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AN INQUIRY INTO PERSONALITY DIFFERENCES AMONG NONRECOVERED AGORAPHOBICS, FULLY RECOVERED AGORAPHOBICS, AND NONPHOBICS

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ABSTRACT

AN INQUIRY INTO PERSONALITY DIFFERENCES AMONG NONRECOVERED AGORAPHOBICS, FULLY RECOVERED AGORAPHOBICS, AND NONPHOBICS

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The clinical presentation of Panic Disorder with agoraphobia is associated with additional psychopathology including chronic anxiety and global neuroticism. It has been demonstrated that posttreatment improvement in the core syndrome of panic attacks and phobic avoidance is correlated with improvement in associated symptomatology. However, there is controversy regarding the nature of the relationship between the core syndrome and associated psychopathology.

The present study was designed to investigate the relationship between Panic Disorder with agoraphobia and both chronic anxiety and global neuroticism by contrasting fully recovered agoraphobics, nonrecovered agoraphobics and nonphobics on measures of chronic anxiety and neuroticism. It was hypothesized that fully recovered agoraphobics would score lower on both measures than nonrecovered agoraphobics but higher than nonphobics. The presence of anxiety and neuroticism in fully recovered agoraphobics would support the theory that these characteristics were primary to and predisposed the development of Panic disorder with

agoraphobia.

There were 30 subjects in each sample group which were relatively homogenous with respect to age, income, and socioeconomic status. Posttreatment agoraphobics were divided into fully recovered and nonrecovered groups using a hierarchical criterion procedure, based upon DSM-III-R criteria, through administration of Recovery and Avoidance Inventories. Subsequently, fully recovered agoraphobics, nonrecovered agoraphobics and nonphobics were contrasted on indices of global neuroticism (Eysenck Personality Questionnaire-Neuroticism Scale) and chronic anxiety (State-Trait Anxiety Inventory, A-Trait Form). Nonrecovered agoraphobics were found to be significantly more anxious and neurotic than both fully recovered agoraphobics and nonphobics. No difference in anxiety or neuroticism was found between fully recovered agoraphobics and nonphobics.

The results of the present study were not consistent with the theory that chronic anxiety or global neuroticism predisposed the development of Panic Disorder with agoraphobia. Additionally, it was concluded that current agoraphobics were homogenous with respect to treatment experience in terms of anxiety and neuroticism.

This dissertation is dedicated to my spouse, Dr. Ann Washabaugh Sams, who provided continual love, encouragement, and feedback. I love you and I thank you.

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CHAPTER I

INTRODUCTION

In May of 1987, the American Psychiatric Association revised the Diagnostic and Statistical Manual of Mental Disorders - Third Edition (DSM-III), originally published in 1980. There were numerous diagnostic changes in the category of Anxiety Disorders, reflecting an explosion of literature in this area. In fact, a recent review of psychiatric research in the Journal of the American Medical Association was devoted almost exclusively to Anxiety Disorders, particularly Panic Disorder and Agoraphobia (Freedman, & Glass, 1984). The present study is offered as a contribution to the understanding of Panic Disorder and Agoraphobia.

Agoraphobia as a clinical syndrome had its roots in the nineteenth century. Originally coined by Westphal in 1871, Agoraphobia has also been referred to as locomotor anxiety (Abraham, 1913); street fear (Miller, 1913); phobic anxiety depersonalization syndrome (Roth, 1959); phobic anxious states (Klein, 1964); and nonspecific insecurity fears (Snaith, 1968). Typically, it involves the onset of panic attacks, and subsequently, increasing constriction and

avoidance of daily activities to prevent further attacks.

In a nationwide British survey of agoraphobics, Burns and Thorpe (1977) determined the hierarchy of situations most likely to generate anxiety to be as follows: joining a line in a store, a definite appointment, feeling trapped at the hairdresser, increasing distance from home, domestic arguments, thinking about problems, and particular places in the neighborhood. In a later article (1980), using data from the same study, they reported wide fluctuations on a daily basis for most agoraphobics.

The DSM-III described the agoraphobic syndrome in a manner based largely on Marks' observations in 1970. To this extent, Agoraphobia was considered a Phobic Disorder along with Simple and Social Phobia. Classification required specification of the presence of panic attacks. Diagnostic features included: a marked fear or avoidance of being alone or in public places from which escape might be difficult or help not available in case of sudden incapacitation, and increasing constriction of normal activities (APA, 1980).

Subsequent research suggested that there was a qualitative biological distinction between panic attacks and anxiety and that a predisposition to panic attacks was genetically inherited. This evidence led many researchers to conclude that panic attacks were primary in the development of Agoraphobia and distinct from Generalized

Anxiety Disorder. Further research appeared to support these conclusions and, in 1987, they were incorporated by the American Psychiatric Association into the revised edition of the DSM-III (DSM-III-R).

In the DSM-III-R, Panic Disorder is still classified as a subtype of Anxiety Disorder. However, Agoraphobia is no longer considered a unified clinical subtype of Phobic Disorder. Agoraphobic avoidance has been divided between Anxiety and Phobic Disorders. Agoraphobia without panic attacks is still considered a Phobic Disorder. However, Agoraphobia associated with panic attacks is considered an Anxiety Disorder, with Agoraphobia only a specification for Panic Disorder. Diagnostic criteria for Panic Disorder with agoraphobia require presence of Panic Disorder and subsequent development of agoraphobia.

The most accurate estimates of prevalence of Panic Disorder with agoraphobia vary from approximately 2-5% of the population (Myers et al., 1984; Robbins, Helzer, & Weissman, 1984). Onset usually occurs between the ages of 18 and 35 (Marks, 1970a). Though etiology is highly controversial, the models of Klein (1980, 1981) and Sheehan (1982b, 1983) appear to be the most widely accepted. They postulate a genetic predisposition toward Panic Disorder, with agoraphobic avoidance developing from spontaneous, endogenous panic attacks.

A product of this current theoretical orientation is that associated symptomatology is considered solely a consequence of Panic Disorder with agoraphobia. Researchers have consistently identified the following related symptoms: chronic anxiety, global neuroticism, depression, obsessive compulsiveness, hypochondriasis, dependency, sexual dysfunction, and assertion deficits. There is evidence that many of these symptoms decrease as phobic constriction, panic frequency and panic intensity are decreased. For example, Chambless (1985) demonstrated that as frequency of panic attacks and severity of avoidance behavior increased in a population, so did anxiety, depression, neuroticism, fear of fear, and panic intensity.

However, many researchers theorize that Panic Disorder with agoraphobia arises out of characterological and/or environmentally induced problems in coping, such as: threatened anxious attachment (Bowlby, 1974, 1980); dependency due to separation fears and/or lack of skills (Goldstein, & Chambless, 1978); chronic anxiety (Hallam, 1978); and interpersonal conflict (Doctor, 1982; Last, 1984). This line of research suggests that neurotic symptomatology associated with Panic Disorder with agoraphobia may contribute to the development of the core syndrome of panic attacks and phobic avoidance.

Need for the Study

Past research correlating agoraphobic recovery with neurotic improvement has typically measured scores on neurotic scales both before and after treatment (Bland, & Hallam, 1981; Hafner, 1981; Mavissakalian, 1985; Milton, & Hafner, 1975). These studies have demonstrated that as patients recover from Panic Disorder with agoraphobia, there is a decrease in the severity of associated neurotic symptomatology. Conclusions were drawn that the change in phobic and panic severity caused the change in associated symptomatology. Additionally, this line of research was used to support the theory that associated symptomatology was only a secondary consequence of Panic Disorder with agoraphobia.

If neurotic symptomatology was solely a consequence of the core syndrome of panic attacks and phobic avoidance, we would expect that fully recovered agoraphobics would demonstrate considerably less neurotic symptomatology than nonrecovered agoraphobics. However, researchers have not distinguished between posttreatment agoraphobics and fully recovered posttreatment agoraphobics. Previous research has employed posttreatment measures of panic attacks and phobic avoidance as an index of recovery without specifying that patients be fully recovered. Consequently, previous research has not contrasted fully recovered agoraphobics with nonrecovered agoraphobics on measures of anxiety and

neuroticism. If fully recovered agoraphobics scored as high as nonrecovered agoraphobics on measures of anxiety and neuroticism, it would suggest that associated symptomatology was not solely a consequence of the core syndrome.

Since available research has made no distinction between fully recovered and nonrecovered agoraphobics on post treatment measures, it is unclear how degree of recovery and associated symptomatology interact. If neurotic symptomatology were solely a consequence of panic attacks and phobic avoidance, we would expect that fully recovered agoraphobics would be no more anxious or neurotic than nonphobics. However, previous research has not contrasted fully recovered agoraphobics with nonphobics on measures of anxiety or neuroticism. If fully recovered agoraphobics scored higher than nonphobics on these measures, it would further suggest that associated symptomatology was not solely a consequence of the core syndrome.

While it has been demonstrated that agoraphobics have more neurotic symptomatology than nonphobics, previous research has not contrasted nonrecovered agoraphobics who have completed a treatment program with nonphobics. It is possible that posttreatment neurotic symptomatology could be reduced to nonphobic levels in the absence of full recovery. Researchers have assumed that agoraphobics were a homogenous population with no differentiation based on previously

unsuccessful treatment experience. If nonrecovered agoraphobics did not score higher than nonphobics on measures of anxiety and neuroticism, then agoraphobics should not be viewed as a homogenous population based on previous treatment experience; future research on associated symptomatology would require that agoraphobics be differentiated into groups with or without treatment experience.

The primary usefulness of differentiating between fully recovered and nonrecovered agoraphobics is in the possibility of determining the effect of level of recovery on degree of neurotic improvement. If fully recovered agoraphobics evidenced more neurotic symptomatology than nonphobics, but less neurotic symptomatology than nonrecovered agoraphobics, support would be given the theory that a high index of neuroticism predisposes to Panic Disorder with Agoraphobia. Such results would be inconsistent with the theory that Panic Disorder with agoraphobia is primary in the development of all associated neurotic symptomatology.

Purpose of the Study

This study was designed to evaluate nonrecovered agoraphobics, fully recovered agoraphobics, and nonphobics on measures of anxiety and neuroticism. For the purposes of this study, the nonphobic group was defined as a sample with

similar demographic characteristics as the phobic groups that was presumed to be normal with respect to panic attacks and phobic avoidance. The fully recovered group was defined as a previously identified agoraphobic sample that had not demonstrated panic or phobic symptomatology for at least six months. Contrasting fully recovered agoraphobics with both nonrecovered agoraphobics and nonphobics addressed the primacy of Panic Disorder with Agoraphobia in the development of associated symptomatology. Contrasting nonrecovered agoraphobics with nonphobics addressed the relative homogeneity of current agoraphobics. Evaluating the effect of level of recovery on degree of neurotic improvement addressed the possible primacy of anxiety and neuroticism in the development of Panic Disorder with agoraphobia.

Research Questions

The following research questions were addressed in this study:

1. Will nonrecovered agoraphobics evidence significantly higher chronic anxiety than nonphobics as measured by scores on the State-Trait Anxiety Inventory, A-Trait Form (STAI-T)?
2. Will nonrecovered agoraphobics evidence significantly higher anxiety than fully recovered agoraphobics as measured by scores on the STAI-T?

3. Will fully recovered agoraphobics evidence significantly higher chronic anxiety than nonphobics as measured by scores on the STAI-T?
4. Will nonrecovered agoraphobics evidence significantly higher global neuroticism than nonphobics as measured by scores on the Eysenck Personality Questionnaire-Neuroticism scale (EPQ-N)?
5. Will nonrecovered agoraphobics evidence significantly higher global neuroticism than fully recovered agoraphobics as measured by scores on the EPQ-N?
6. Will fully recovered agoraphobics evidence significantly higher global neuroticism than nonphobics as measured by scores on the EPQ-N?

Overview of Remaining Chapters

In Chapter II, the relevant literature is reviewed in the following areas: identification of the agoraphobic syndrome, current diagnostic criteria, onset and etiology, and associated psychopathology. The research design and procedures are presented in Chapter III, including reliability and validity information for each of the measures used. In Chapter IV, the results and statistical analysis are presented. In Chapter V, conclusions and summary are presented, along with recommendations for further research.

CHAPTER II

LITERATURE REVIEW

In this chapter, relevant research and theory are reviewed in four major areas. Initially, the establishment of Agoraphobia as a syndrome is described. Current diagnostic criteria are presented based on theoretical revisions of the syndrome. Onset and etiology are explored. Finally, associated psychopathology is reviewed.

Establishment of the Agoraphobic Syndrome

The term Agoraphobia was first coined by Westphal in 1871. In a detailed series of monographs, he described three individuals who became extremely anxious in certain specific situations, such as, crossing an open square, walking through an open street, being in a church or theater, or being in any place where many people were gathered. He described the symptoms of anxiety as dizziness, heart palpitations, perceptions of heat, blushing, and trembling. He reported increasing anticipatory anxiety and phobic avoidance of these situations; he noted that this anxiety was alleviated by the

presence of a companion and in one case by holding an umbrella.

Other clinicians of his era supported Westphal's observations. In 1870, Benedict described a similar case, although he viewed dizziness as the central feature, rather than anxiety. In 1871, Cordes described similar cases and suggested that anxiety was stimulated by thoughts and was not merely a reaction to the environment. In 1885, Legrand du Saule detailed a similar case and suggested that the disorder was more common in men.

The term Agoraphobia is derived from the Greek words agora and phobos. Agora means flight, panic, assembly, or place of assembly. Phobos means flight, panic, fear, or terror. Hence the term Agoraphobia literally means flight from or fear of crowds, or places for crowds. It should be noted that others have used different terms in describing the same condition, including: locomotor anxiety (Abraham, 1913); street fear (Miller, 1913); phobic anxiety depersonalization syndrome (Roth, 1959); phobic anxious states (Klein, 1964); nonspecific insecurity fears (Snaith, 1968); and endogenous phobic anxiety (Sheehan, 1982b). Several authors have incorrectly identified Agoraphobia simply as an intense fear of open spaces (Eysenck, 1972; Rycroft, 1968; Wolman, 1973). Unfortunately, much of the lay public has accepted and propagated this definition.

There have been numerous anecdotal and clinical accounts of the agoraphobic syndrome. Additionally, several factor analytic studies have explored the syndrome. In 1965, Roth, Garside, and Gurney performed a factor analytic study of 275 neurotic patients using data gathered in a clinical setting. They found neurotic symptoms clustering around an agoraphobic factor which included, in descending order of strength: situational phobias (Agoraphobia), panic attacks, depersonalization and derealization, temporal lobe features, marked precipitant, sudden onset, and dizzy attacks. The authors concluded that there was an agoraphobic factor distinct from general anxiety neurosis.

In a 1967 study, Marks factor analyzed questionnaire replies from 72 phobic patients and found that seven major symptoms loaded on an agoraphobic factor. In descending order of strength the factors included: fears of fainting in public, a train journey, being left alone, being in a crowd or open space, watching a surgical operation, and crossing a bridge or street.

Marks' use of a small sample and general phobic population prompted Marks and Herst to perform a larger study in 1970. They sent questionnaires to members of a phobic support group called The Open Door. The results were based on 900 respondents. They found the specific fears included: trains, buses, theaters, elevators, crowds, shops, tunnels, parties, travelling in a car, streets or open

spaces, public speaking, eating in public, and bridges. Associated symptomatology included: exhaustion, dizziness, fears of fainting, headaches, shaking, palpitations, tension, depersonalization, obsessions, and panic attacks.

In 1978, Hallam and Hafner performed a factor analysis of items describing commonly feared situations. They replicated earlier factor analytic studies that had been performed on the Fear Survey Schedule using normal populations (Geer, 1965; Lang, & Lazovic, 1973; Wolpe, & Lang, 1964). These studies had previously identified four frequently occurring clusters in normal populations that included: fears of interpersonal events, small animals, aggression and death, and physical pain or surgery. Hallam and Hafner had 171 phobic outpatients complete the schedule and found an agoraphobic factor that involved fears of travel, open spaces, and crowded or confined places.

The following year, Arrindell (1979) replicated the factor analysis of Hallam and Hafner with a larger sample of 703 phobic outpatients. He found the same five factors as Hallam and Hafner and agreed that the agoraphobic factor involved fears of travel, open spaces, and crowded or confined places. He concluded that this factor was clearly independent of the others and could not be reduced to a subset of different fears or a trait of general fearfulness.

Other studies indirectly validated the existence of an agoraphobic syndrome. In a 1959 study, Roth concluded that

phobic anxiety depersonalization syndrome (Agoraphobia) was not a graded affective phenomenon that merged into other disorders. He maintained that the syndrome was not present in a mild form and appeared to have all-or-none characteristics . In 1973, Hersen supported this conclusion with a review of factor analytic studies of the Fear Survey Schedule on normal populations. He did not find an agoraphobic factor, further suggesting the lack of occurrence in a mild form. Additionally, in the Arrindell study (1979), higher order factor analysis revealed that the agoraphobic factor was independent of the neurotic factor. Finally, in an analysis of follow-up studies, Munby and Johnson (1980) found that agoraphobics were not subsequently diagnosed with anxiety neurosis or depression in later years.

In 1970, Marks described the agoraphobic syndrome in a seminal article. He chose the term Agoraphobia because the most common and debilitating elements of the syndrome were fears of entering various public places. He described the syndrome as a cluster of phobias centered around entering public places. Subsequent researchers generally conceptualized the syndrome in this manner.

This direction in research and theory championed by Marks prompted a reclassification of Anxiety Disorders when the Diagnostic and Statistical Manual of Mental Disorders - Third Edition (DSM-III) was published by the American

Psychiatric Association in 1980. The Anxiety Disorders were categorized as Anxiety States and Phobic Disorders, with the former encompassing Panic, Obsessive Compulsive, Generalized Anxiety, and Posttraumatic Stress Disorders and the latter encompassing Simple, Social, and Agora-Phobias.

Within the classification of Agoraphobia in the DSM-III, specification was required regarding the presence of panic attacks. Diagnostic criteria for Agoraphobia included a fear of panic attacks with consequent travel restrictions and dependency on a companion away from home, or intense anxiety in agoraphobic situations (APA, 1980). Agoraphobia was defined as a cluster of phobias with onset typically associated with recurrent panic attacks. Further, it was suggested that anticipatory fear of panic attacks produced phobic avoidance of situations associated with these attacks.

Thus, the DSM-III was suggesting that Agoraphobia with panic attacks was a phenomenon learned primarily through classical conditioning and the process of generalization. The DSM-III stated that an individual developed panic attacks and became phobic of situations in which they occurred. Through generalization to increasingly dissimilar stimuli, it was suggested that the individual became increasingly constricted until confinement to the home was possible. This conceptualization reflected the prevailing line of research in 1980 that classified Agoraphobia as a

distinct clinical entity of phobic type.

Current Diagnostic Criteria

In 1959, Klein began to notice that inpatient agoraphobics responded to imipramine, whereas treatment with psychotherapy, social therapy, and phenothiazines had been unsuccessful. In 1962, Klein and Fink concluded that imipramine appeared to reduce panic frequency. Subsequently, Klein (1964) reported on 32 agoraphobic patients who received imipramine, monoamine oxidase (MAO), or no medication. He found panic attacks were reduced in all patients receiving either antidepressant.

This research, in concert with that of King (1962), who demonstrated the effectiveness of phenelzine in treating panic attacks, prompted Klein to conclude that some antidepressants differentially affected panic and chronic anxiety. Thus, he postulated in 1964 that panic and anxiety were qualitatively different phenomena. He supported this theory in a double blind study (1967) that demonstrated the effectiveness of imipramine versus placebo or chlorpromazine in the treatment of Agoraphobia.

More recent studies have also concluded that imipramine promoted recovery from panic attacks and Agoraphobia (Mavissakalian, 1985; Mendel, & Klein, 1969; Sheehan, 1980; Zitrin, Klein, & Woerner, 1978, 1980). Other researchers supported King in concluding that phenelzine promoted agoraphobic recovery (Kelly, Guiguis, & Frommer, 1970;

Mountjoy, Roth, Garside, & Leitch, 1977; Sheehan, 1980; Tyrer, 1975; Tyrer, Candy, & Kelly, 1973a, 1973b;).

This line of research began to cast doubt on the classification of Agoraphobia as a distinct clinical syndrome. Research suggesting that antidepressants were effective in treating panic attacks, but not chronic anxiety, led many researchers to conclude that panic attacks were qualitatively different than anxiety. Additional support for this conclusion was provided by the assertion that benzodiazepines were effective in the treatment of chronic anxiety, but not panic attacks and Agoraphobia. A consensus emerged among some researchers that panic attacks were primary to phobic avoidance in the development of Agoraphobia rather than associated symptomatology, such as chronic anxiety.

It should be noted that no studies have been performed using antidepressants in patients with a primary complaint of generalized anxiety. Consequently, it has not been established that antidepressants are ineffective in the treatment of generalized anxiety. Further, it has been argued that in studies of patients with other primary diagnoses, antidepressants have significantly reduced anxiety (Matthews, 1981). In 1983, Marks concluded that tricyclics have a broad range of treatment effects not limited to depression or panic attacks.

There are numerous methodological considerations in drawing conclusions from studies of drug specificity. In 1983, Mavissakalian reported high drop out rates and relapse rates of approximately 25% following treatment. Zitrin et al. (1980) reported that 20% of agoraphobics responded to antidepressant drug treatment with insomnia, nervousness, irritability, and exacerbation of panic attacks. Matthews, Gelder, and Johnston (1981) found that systematic instructions to practice entering phobic situations were an important factor in therapist assisted treatments. Unfortunately, most pharmacologic studies of Agoraphobia employed such instructions, confounding the effects of medication (Sheehan, 1980; Zitrin et al., 1978, 1980).

There is also controversy regarding the antipanic and antiphobic qualities of antidepressants independent of their antidepressive effects. Researchers have evaluated the possible effect of pretreatment depression on treatment outcome, which would expected if the medication ameliorated panic and avoidance primarily through antidepressant qualities. Two studies found no interaction between depression and treatment outcome of phobic states with MAO inhibitors (Kelly et al., 1970; Sheehan, 1980). Others found the same lack of interaction between depression and treatment outcome with imipramine (Sheehan, 1980; Zitrin et al., 1978, 1980). Zitrin et al. (1980) noted that the presence of high scores on pretreatment depression was

associated with a poor outcome on several measures of agoraphobic severity.

However, other researchers have argued for an interaction between depression and agoraphobic treatment effects. In a review article, Marks (1983) indicated that eleven of fifteen studies which analyzed outcome of both depression and target problem found concordance of improvement or no change. He described two of the four nonconcordant studies as providing further evidence of the antidepressant nature of the drugs (McNair, & Kahn, 1981; Telch, 1983).

In the Telch study, three groups were contrasted: imipramine only, imipramine plus exposure in vivo, and placebo plus exposure in vivo. The imipramine only subjects had instructions to rest and refrain from entering phobic situations. This group improved only on depression. The placebo plus exposure group improved in phobia and panic but little in depression. The imipramine plus exposure group improved on measures of panic, phobia, and depression. Telch concluded that imipramine functioned primarily to alleviate depression and that entering phobic situations was critical to recovery.

In Marks' article (1983), he concluded that, "One predictor of drug effect seems to be the starting level of dysphoria, drug effects generally being absent where this is low" (p. 341). He asserted that drug effects were typically

found in studies where initial depression was higher, whereas effects were not found where depression was lower. Marks concluded that imipramine was not effective in treating nondepressive Agoraphobia and functioned primarily through alleviation of depression and subsequent promotion of exposure.

Overall, while it is clear that some antidepressants can be effective in the treatment of Agoraphobia, the mechanism of action remains unclear. These medications are certainly not a cure given the high relapse rate and general lack of treatment effect in the absence of practice exposure.

The assertion of a qualitative distinction between anxiety and panic was further supported by evidence of the ineffectiveness of benzodiazepines and beta blocking agents in the treatment of panic attacks and Agoraphobia. In a 1976 study, Hafner and Marks concluded that there was no significant difference between diazepam and placebo in the treatment of Agoraphobia, although diazepam patients experienced less discomfort during treatment. Hersec and DuFrance (1979) concluded that propranolol was effective in treating panic attacks after onset, but did not facilitate improvement in panic associated with Agoraphobia. Shader, Goodman, and Gever (1982) and Sheehan (1982a, 1982b) concluded that these drugs functioned to alleviate generalized anxiety, but did not prevent panic attacks.

The results of these studies are contradicted by those of Johnson and Goth (1973) which found diazepam to be effective in facilitating treatment of Agoraphobia. It has been suggested that, since their sample involved only severe agoraphobics, with higher levels of generalized anxiety, treatment with diazepam resulted in greater exposure and practice. Hafner and Miller (1977) compared the treatment effectiveness of propranolol plus exposure with exposure alone, and found the non-propranolol group less mobile and more symptomatic at three-month follow-up. Accordingly, in the only study to administer high doses of diazepam at regular intervals, Noyes, Anderson, and Clancy (1984) found that diazepam and propranolol each reduced panic frequency in a double blind placebo controlled study.

Some researchers have concluded that a newly developed triazolobenzodiazepine, alprazolam, holds promise for the treatment of Agoraphobia and panic attacks (Sheehan, Coleman, & Greenblatt, 1984; Vittone, & Uhde, 1985). In a double blind placebo controlled experiment, Chouinard (1981) found alprazolam effective in the treatment of generalized anxiety and panic disorders. Although this evidence contradicted the drug specificity theory, some researchers maintained that alprazolam treatment effects are due to its special chemical structure and consequent antidepressant properties (Chouinard, 1982; Feighner et al., 1983; Sheehan et al., 1984). Vittone and Uhde (1985) also reported that

after initial dramatic improvement symptoms often relapse.

Overall, it seems premature to conclude that anxiolytic medications are ineffective in the treatment of panic attacks and Agoraphobia. Similarly, whereas some antidepressants have been effective in the treatment of panic attacks and Agoraphobia, it is not clear that treatment effects are independent of the antidepressant qualities. Studies of drug specificity do not appear to provide convincing evidence that panic and anxiety are qualitatively different phenomena.

Further evidence for the distinction between panic and anxiety, and for the primacy of panic attacks in the development of Agoraphobia, came from studies suggesting that ingestion of certain chemicals had differential effects on panic or anxiety patients and controls. In 1967, Pitts and McClure concluded that infusion of sodium lactate reliably produced panic attacks in significantly more anxiety neurotic patients than normals. They also indicated that subsequent infusions of calcium mitigated the effects of the lactate. They speculated that lactate-induced panic attacks involved the chelation of calcium by lactate, which interfered with normal nerve transmission. Other researchers have replicated the results of this study (Bonn, 1973; Fink, Taylor, & Volovka, 1969; Kelly, Mitchell-Heggs, & Sherman, 1971). It was suggested that these studies provided some evidence of a biological marker for panic

attacks, that is, that panic patients have a specific biological vulnerability (Carr, & Sheehan; Klein, 1981; Shader et al., 1982).

Other researchers have seriously questioned the lactate infusion studies. Grosz and Farmer (1969) reported serious methodological shortcomings in the Pitts and McClure study. Ackerman and Sachar (1974) reviewed the lactate studies and suggested that lactate infusions were nonspecific stressors in patients conditioned to overreact to altered bodily sensations. In a review of all published studies of lactate infusion and panic attacks, Margraf, Ehlers, and Roth (1986) raised other concerns. They maintained that conclusions were tenuous, since demand characteristics, expectancy bias, and other variables were not adequately controlled.

In a recent study, Ehlers, Margraf, and Roth (1986) found that panic patients had higher overall levels of anxiety. However, when baseline levels were considered, they found that lactate induced increases in subjective anxiety and physiological arousal were approximately equal in panic patients and controls. They concluded that lactate effects were moderate overall.

Other chemicals have been used for infusion studies to differentiate between panic and anxiety. Pitts and Allen (1969) concluded that infusions of yohimbine, an alpha-adrenergic agonist, produced panic symptoms similar to sodium lactate. In 1969, Frolich and Tarazi reported that

panic could also be induced in patients with hyperdynamic beta-adrenergic states, but not in normals, by infusion of the beta-adrenergic agonist isoproterenol. Further, they found the effects were reversed by an infusion of propranolol, a beta blocker. The researchers concluded that there was an increased beta-adrenergic sensitivity in some patients.

Finally, other studies have suggested that panic patients respond differently to caffeine than controls (Boulanger, & Uhde, 1982; Boulanger, Uhde, Wolff, & Post, 1984). These studies provided some evidence that caffeine heightened anxiety and that panic patients experienced greater increases in physiological arousal and subjective anxiety. However, these studies also created nonspecific stressors in individuals conditioned to overreact to altered bodily sensations.

The preponderance of research in the area of panic attacks and chemical infusion at least suggests, if not confirms, that panic patients react with greater arousal than normals. This is consistent with the research of Lader and Wing (1966) and Lader et al. (1967) which demonstrated that agoraphobics display more spontaneous fluctuations in skin resistance and habituate more slowly than normals and simple phobics. However, conclusions drawn from infusion research which suggest qualitative differences between panic and anxiety, or a biological predisposition to panic, remain

highly speculative.

Researchers postulating a qualitative distinction between panic and anxiety consider Agoraphobia to be a secondary manifestation of Panic Disorder. They emphasize the similarity of patients with Panic Disorder and those with Agoraphobia. They suggest that if Agoraphobia were a distinct clinical entity, there would be at least mild differences between panic and agoraphobic patients. In general, research does not support such a difference.

In a study by Garvey and Tuason (1984), no differences were found between and panic and agoraphobic patients in psychosocial stressors, symptomatology, or family history. In 1985, Thyer, Nesse, Cameron, and Curtis compared twenty panic disordered patients with twenty agoraphobics on a variety of demographic and personality variables. No significant differences were found on demographic data. Differences in personality variables were found only for paranoid ideation and interpersonal sensitivity. They concluded that agoraphobics and panic disordered patients were virtually indistinguishable.

Further support for the primacy of panic attacks in the development of agoraphobia lies in the fact that Agoraphobia typically follows panic onset and that Agoraphobia without panic is rare. Garvey and Tuason (1984) found that Agoraphobia never preceded panic attacks. Thyer and Himle (1985) reported that panic attacks preceded the development

of agoraphobia by an average of nine years. Garvey and Tuason also found that 92% of their sample of agoraphobics experienced panic attacks, while 83% of the agoraphobics in the study of Thyer et al. (1985) had panic attacks. This latter study also reported that "histograms plotting the ages of onset for the conditions of Agoraphobia with panic attacks and Panic Disorder appear virtually identical as unimodal curves with an average age of 26" (p. 209). This was in contrast to histograms plotting the ages of Simple and Social Phobias which were quite negatively skewed, with most cases occurring before age 30.

The preponderance of research on drug specificity, panic induction, hypersensitivity of panic patients, and similarities among panic and agoraphobic patients produced a revised agoraphobic theory, championed by Klein (1980, 1981) and Sheehan (1982b, 1983). This theory integrated the available research and concluded: that panic attacks are qualitatively different than anxiety; that people inherit a biological predisposition to panic; and that Agoraphobia is a secondary manifestation of spontaneous panic attacks. This model prompted a general revision of the diagnostic criteria for Agoraphobia.

In 1987, the task force responsible for reviewing the DSM-III made numerous revisions in publishing the Diagnostic and Statistical Manual of Mental Disorders, Third Edition - Revised (DSM-III-R) (American Psychiatric Association).

Among these changes was the classification of Anxiety Disorders. Principally, Agoraphobia with panic attacks was shifted from the category of Phobic Disorder to Anxiety State. Panic Disorder became the primary diagnosis and required specification of the presence of agoraphobia. The diagnostic features of Panic Disorder with agoraphobia required presence of full blown panic attacks (Panic Disorder) and subsequent fear of places or situations from which escape might be difficult or embarrassing, or assistance not available if needed. The DSM-III-R indicated that a product of this fear was restriction of travel, requirement of a companion, or intense anxiety in agoraphobic situations. Diagnosis required specification of current severity of agoraphobic avoidance and panic attacks.

Agoraphobia without history of panic attacks is still categorized as a Phobic Disorder in the DSM-III-R. Diagnostic features include: absence of panic attacks; and presence of the agoraphobic syndrome based on a fear of suddenly developing a symptom that could be incapacitating or embarrassing, such as, dizziness or falling, depersonalization or derealization, loss of bladder or bowel control, vomiting, or cardiac distress. Additionally, specification is required regarding the existence of limited symptom attacks, which are identified by the presence of fewer than four of the thirteen criteria for panic attacks.

Prevalence and Etiology

Studies of agoraphobic prevalence were conducted when it was considered a singular entity of phobic type. Hollingshead and Redlich estimated community prevalence at five per thousand in 1958. A later study (Agras, Sylvester, & Oliveau, 1969), using a small Vermont community, reported prevalence of 6.3 per thousand. A 1974 study by the Phobic Society of England reported community prevalence of six per thousand.

The most accurate current estimate of agoraphobic prevalence is based on two studies sponsored by the National Institute of Mental Health (NIMH) as part of the Epidemiologic Catchment Area (ECA) Study (Myers et al., 1984; Robbins et al., 1984). Both studies involved careful random selection and multisite sampling. Myers et al. found six month prevalence ranges from 2.7% to 5.8% in females and 0.9% to 3.4% in males, depending on the site. Robbins et al. reported lifetime prevalence ranges from 5.3% to 12.5% in females and 1.5% to 5.2% in males, depending on the site.

As suggested by the ECA figures, prevalence of Agoraphobia differs between the sexes. These two studies suggested that at least 67% of agoraphobics are female. Numerous earlier studies reported that 64% to 89% of agoraphobic samples were female (Errera, & Coleman, 1963; Friedman, 1950; Klein, 1964; Marks, 1970b; Marks, & Gelder, 1965; Roth, 1959; Sim, & Houghton, 1966; Snaith, 1968;

Terhune, 1949; Tucker, 1956; Warburton, 1963). More recent studies suggested that approximately 85% of agoraphobics are female (Bowen, & Kohout, 1979; McGinnis, Nolan, & Hartman, 1977; Thorpe, & Burns, 1980). It is likely that the actual community prevalence of Agoraphobia is slightly less than 85% female, as most studies involve individuals who have sought some type of treatment, and it is widely recognized that females are more likely to pursue treatment than males.

In psychiatric practice, it is generally agreed that the prevalence of phobias as the main complaint is approximately two to three percent in America and England (Errera, & Coleman, 1963; Hare, 1961; Marks, 1970a; Roth, 1959; Terhune, 1949).

Research findings for onset age of Agoraphobia have been rather consistent, usually beginning in young adults between the ages of 18 and 35 with a mean age in the middle to late 20s (Buglass, Clarke, Henderson, Kreitman, & Presley, 1977; Marks, & Herst, 1970; Shafar, 1976; Solyom, Beck, Solyom, & Hugel, 1974; Sheehan, Sheehan, & Minichiello, 1981; Tearnan, Telch, & Keefe, 1984; Thyer et al., 1985). Marks (1970a) reported that the disorder was rare in children. Although some studies reported a bimodal distribution for age of onset with peaks at approximately twenty and thirty years (Marks, & Gelder, 1966; Mendel, & Klein, 1969), more recent studies by Sheehan et al. (1981) and Thyer et al. (1985), using large samples and careful

screening, strongly suggested a unimodal distribution. Thyer et al. reported a mean onset age of 26.3 years for Agoraphobia with panic attacks, while Sheehan et al. found a mean onset age of 24.1 years for endogenous anxiety disorders. Similarities in onset age and distribution among panic and agoraphobic patients have been previously indicated as support for combining the two conditions.

The etiology of Agoraphobia is highly controversial, as is typical of most psychological disorders. Theoretical orientation impacts on conceptualization of etiology and often results in producing different interpretations of the same data.

Basic demographic information regarding Agoraphobia appears rather consistent. Marks (1970a) reported that his Open Door sample was average with respect to education, occupational status, income and religious affiliation. However, Rock and Goldberger (1978) concluded that agoraphobics had lower educational attainment than simple phobics. In 1983, Weissman reviewed epidemiological research and concluded that Agoraphobia was twice as likely to occur in those with lower educational levels. Chambless (1985) combined social occupation with education to compute social class based on the Hollingshead (1957) Two-Factor Index of Social Position. She concluded that Agoraphobia was significantly more likely to occur among individuals with lower social position. It does appear that Agoraphobia

is more common in individuals with lower socioeconomic status.

Research into the clinical course of Panic Disorder with agoraphobia has included genetic inheritance, separation anxiety and interpersonal context. Typically, evidence of genetic inheritance has been based upon family, twin, and adoption studies. In reviews of relevant literature, Burns and Thorpe (1977) and Burns (1980) found a higher incidence of Anxiety Neurosis in the families of Anxiety Neurosis patients. In 1981, Carey and Gottesman reviewed four studies that used the family history method (Brown, 1942; Cohen, 1951; McInnes, 1937; Noyes et al., 1978). They concluded that research has "consistently reported a prevalence of about 15% in the first degree relatives of anxiety neurotics, despite wide variations in the time, methods and diagnostic habits of the investigators" (p. 315). In the most recent of the studies, Noyes et al. (1978) conducted a family history on 112 patients diagnosed with Anxiety Neurosis. The risk for anxiety among first degree relatives of anxiety neurotics was 18%, in contrast to 3% for controls.

In 1983, Crowe, Noyes, Pauls, and Slymen conducted a family study of 19 patients, using DSM-III criteria for Panic Disorder. This study was more powerful methodologically than previous research as the family members were interviewed. The age corrected risk for

Anxiety Disorder in relatives of the patients was 41%, in contrast to an 8% risk for families of controls. The risk to siblings also increased with the number of affected parents. Crowe and his associates also conducted a follow-up study (1983b) by comparing 278 first degree relatives of 41 patients with Panic Disorder to a similar number of relatives of controls. The rate of definite or probable Panic Disorder was 24.7% in panic relatives, as opposed to 2.2% in control relatives. Harris, Noyes, Crowe, and Chaudhry (1983) compared the risk of Anxiety Disorder in the first degree relatives of agoraphobics, panic disordered patients and controls. The morbidity risk in relatives was 32% for agoraphobics, 33% for panic disordered patients and 15% for controls. In both the Crowe et al. and Harris et al. studies, female relatives were at higher risk than males.

While Panic Disorder and Agoraphobia seem clearly familially transmitted, twin and adoption studies are required to separate genetic and environmental effects. In a 1969 study, Slater and Shields compared 150 pairs of same sex twins, where one twin had been previously diagnosed with Anxiety Neurosis. They reported that 41% of the monozygotic (MZ) co-twins had a history of Anxiety Neurosis whereas only 9% of the dizygotic co-twins were afflicted. In 1983, Torgensen conducted a similar study that differentiated Panic and Generalized Anxiety Disorders. He concluded that

MZ co-twins tended toward Panic Disorder more frequently than DZ co-twins. He further concluded that the same was not true of co-twins with generalized anxiety. This study provided the strongest evidence that Panic Disorder was genetically inherited.

In contrast, Raskin, Peeke, Dickman, and Pinsker (1982) were unable to find differential incidence of panic or generalized anxiety in the families of patients with either disorder. The data from Leckman, Weissman, Merinkangas, Pauls, and Prusoff (1983) on depressed and anxious patients supported the study by Raskin et al. In studies more specific to Agoraphobia, Solyom et al. (1974) and Buglass et al. (1977) found no significant differences in phobic tendencies in the families of agoraphobics and controls.

There are problems with drawing conclusions for genetic transmission of Panic Disorder with agoraphobia. In the twin studies, lower rates were reported for the DZ twins (Slater, & Shields, 1969; Torgensen, 1983) than has been consistently reported for siblings in general (Carey, & Gottesman, 1981). Furthermore, the rates for the MZ twins were within sampling error of the rates for other first degree relatives (Carey, & Gottesman, 1981; Slater, & Shields, 1969; Torgensen, 1983). Margraf et al. (1986) concluded that there was familial risk for Anxiety and Panic Disorder, but that determination of the genetic component required adoption studies which were lacking. Additionally,

they concluded that "the larger percentage of nonconcordant monozygotic twins (59-69%) and nonaffected first degree relatives (averaging about 85%) show the strongest influence of nongenetic factors" (p. 558). They suggested that panic risk had not been demonstrated to be independent of risk for generalized anxiety.

In contrast to genetic inheritance, many researchers and theoreticians have related the etiology of Agoraphobia to early childhood separation anxiety. In 1959, Greenson maintained that the agoraphobic individual had a history that produced loss of internal object when separated form the real object (mother or significant other). He theorized that this created loss of ego functioning, which in turn yielded panic. In 1969, Rhead concluded that Agoraphobia was the result of failure to achieve separation and individuation from the original bond with the mother.

Bowlby based his theory of Agoraphobia on attachment and object relations (Bowlby, 1980, 1984). He suggested that individuals are born with a genetic tendency to maintain a steady state relationship with the familiar or safe environment. He maintained that attachment behavior was formed by the mother protecting the child from external danger and meeting the child's needs. Bowlby speculated that agoraphobics have been sensitized into anxious attachment by actual separation or threats of abandonment by

caretakers.

Rachman developed a similar safety signal theory (1984), in which he suggested that avoidance behavior was an attempt to maintain safety, rather than reduce anxiety. This idea was based on previous work by Mowrer (1974) and Gray (1981). Rachman asserted that agoraphobic symptoms were produced in part by a balance between perceived threat and perceived safety. He speculated that any event which altered sense of safety could initiate agoraphobic symptomatology, including separation from significant other.

Klein (1980, 1981) based his theory of agoraphobic development on Bowlby's attachment theory. Klein postulated the presence of an innate alarm mechanism that was activated by separation from attachment figures. He theorized that this mechanism contained a protest and a despair component, similar to Bowlby's (1969, 1973) first two stages of separation response. Klein viewed panic as active, help seeking behavior within the protest component of the alarm mechanism. Klein further suggested that the activation threshold of this alarm mechanism was chronically lowered in panic patients.

There is some empirical support for the hypothesis that separation and attachment anxiety are precursors to panic and phobic avoidance. In 1964, using uncontrolled, nonblind interview techniques, Klein found a history of separation

anxiety in 50% of panic patients. In 1984, Gittelman-Klein and Klein replicated this study with more rigorous methodology. Using data from a previous study (Klein, Zitrin, Woerner, & Ross, 1983) they reported that 50% of female agoraphobic patients had a history of separation anxiety as opposed to 27% of patients with specific phobias. Gittelman-Klein and Klein (1973, 1980) lent further support for the hypothesis that separation anxiety was a precursor to panic through studies that concluded imipramine had a positive effect in treating school phobia. Gittelman-Klein theorized that imipramine was effective in treating both school phobia and panic because each involved the same core mechanism. Other researchers have suggested that operationally defining separation anxiety as school phobia is problematic because school phobia has diverse causes.

Most research relating panic and agoraphobic avoidance to separation and attachment has not supported the preceding theories. Controlled studies have suggested that panic is not related to separation anxiety when operationalized by death of a parent, long term separation before age fifteen, short term separation, or school phobia (Berg, Butler, & Pritchard, 1974; Buglass et al., 1977; Solyom et al., 1974). In a recent study, Thyer et al. (1985) found no difference in separation anxiety between simple phobics and agoraphobics with panic attacks, using their own questionnaire as a measure of childhood separation anxiety.

In a study contrasting panic with chronic anxiety patients, Raskin et al. (1982) reported no differences in childhood anxiety, long term separation before age ten, or number of separations experienced before onset of disorder. Similarly, Coryell, Noyes, and Clancy (1983) found no difference between patients with Panic Disorder and Primary Unipolar Depression on measures of early loss or parental divorce. In 1981, Tennant, Smith, Bebbington, and Hurry reported that separations before age five were unrelated to adult depression or anxiety states, whereas separation experiences after age five were more associated with depression than anxiety. These results were replicated the following year (Tenant, Hurry, & Bebbington, 1982). Additionally, in Torgensen's 1983 article, he concluded that his research provided evidence for a genetic basis of all phobic-related anxiety except separation anxiety. In sum, the weight of evidence does not support a link between childhood separation/attachment anxiety and Panic Disorder or Agoraphobia.

Another postulated etiology for onset of Agoraphobia considers current life events as causative or precipitating factors, often in the context of current interpersonal relationships. Buglass et al. (1977) found that 60% of their sample reported experiencing some type of background stress prior to onset of Agoraphobia. In the Roth study (1959), 96% of 135 agoraphobics reported background stress

preceding the development of the disorder, with 83% reporting onset associated with calamitous circumstances. Solyom et al. (1974) elaborated on this earlier research by identifying the most common precipitants: domestic crises, 35%; death of a relative or friend, 33%; and serious illness in the family, 23%. Recent studies by Doctor (1982) and Last (1984) further implicated stress in the development of Agoraphobia and suggested the major stressor was related to interpersonal conflict.

Goldstein and Chambless (1978) hypothesized the following four necessary and sufficient conditions for the development of panic attacks: "fear of fear as the most central phobic element; low levels of self sufficiency due to anxiety and/or lack of skills; a tendency to misperceive the causal antecedents of uncomfortable feelings; and onset in a climate of notable interpersonal conflict" (p. 51). These researchers speculated that a conflict between normal desires for individuation/autonomy and familiarity/security contributed to the development of Agoraphobia. Goldstein and Chambless maintained that agoraphobics are trapped in situations they cannot resolve due to separation fears and/or lack of coping skills. They suggested that panic results from the exacerbation of conflict due to external stressors such as illness or death. They suggested that the panic experience is misinterpreted and when paired with a low level of self- sufficiency, reinforced the agoraphobic's

sense of dependency. Goldstein and Chambless postulated a self-defeating feedback loop between panic attacks and dependency which promoted increasing phobic constriction.

Most relevant research has supported the theory that agoraphobic onset is associated with interpersonal conflict. Some researchers have concluded that agoraphobics have less satisfactory marriages than normals (Goldstein, & Chambless, 1978; Milton, & Hafner, 1979), whereas others found no difference (Buglass et al., 1977). Wolpe described two clinical cases in which treatment was strongly affected by marital issues (1970, 1973). Holmes (1982) postulated a phobic-counterphobic marital system, in which the spouse or significant other played a major role in the maintenance of symptoms. Numerous studies (Hafner, 1977, 1979, and 1984; Hand, & Lamontague, 1976; Milton, & Hafner, 1979) have found that improvement in agoraphobic symptoms resulted in deterioration of the marital relationship, in the development of symptomatology in the spouse, or in the emergence of new symptoms in the agoraphobic. Further evidence for the interpersonal context of Agoraphobia consisted of studies that found increased treatment efficacy when the spouse was included in the treatment program (Bland, & Hallam, 1981; Emmelkamp, 1980). In a series of studies, Hafner demonstrated that problematic marriages adversely affected response to therapist-assisted, in-vivo exposure (Hafner, 1976, 1977, 1979). Thus, it appeared that

current interpersonal conflict provided the background for and maintenance of the agoraphobic syndrome in some individuals.

Evidence for the interpersonal context of Agoraphobia does not necessarily contradict other theories relating separation anxiety and Agoraphobia. Indeed, Bowlby has suggested that Agoraphobia is a currently adaptive response to threatened anxious attachment. Early experience of threatened attachment can be seen as forming a predisposition to react to current attachment issues in an agoraphobic manner.

In summary, the etiology of Agoraphobia remains controversial. While research supports familial risk for Panic Disorder with agoraphobia, evidence of genetic transmission and biological vulnerability is questionable. The preponderance of evidence does not support early separation anxiety as a precursor of panic attacks or phobic avoidance. Research does support the theory that Agoraphobia is at least maintained as an adaptive response to threatened attachment in current interpersonal relationships, and is not merely a secondary consequence of Panic Disorder.

Associated Symptomatology

It has been consistently demonstrated that agoraphobics suffer from a wide range of psychological problems in

addition to panic attacks and numerous phobias. For example, it has been repeatedly found that Agoraphobia is associated with depression (Bowen, & Kohout, 1979; Buglass et al., 1977; Chambless, 1985; Clancy et al., 1978; Jarret, & Schnurr, 1978; Jasin, 1981; Marks, 1967, 1970a; Noyes et al., 1980; Schapira, 1972; Snaith, 1968). This is consistent with Seligman's model of learned helplessness (1975), in that panic attacks and phobic constriction create feelings of dependency and helplessness. Emmelkamp and Cohen-Kettenis (1975) found that depression among agoraphobics was highly correlated with phobic severity as measured by the Fear Survey Schedule.

Elevated levels of hypochondriasis have also been found in agoraphobics (Buglass et al., 1977; Jasin, 1981; Sheehan, Ballenger, & Jacobson, 1980). Sheehan and Sheehan (1983) described increasing hypochondriasis as part of the natural development of Panic Disorder with agoraphobia. They suggested that hypochondriasis represented the initial search for organic cause of panic attacks. In the later stages, they concluded that hypochondriasis was the cognitive and affective product of an increased awareness of bodily function.

In addition to increased incidence of depression and hypochondriasis, other studies have demonstrated that agoraphobics are deficient in assertion (Chambless, 1982, 1985; Chambless, & Mason, 1986; Goldstein, & Chambless,

1978). Goldstein and Chambless (1978) suggested that lack of assertion was part of the self-destructive feedback loop that promoted panic attacks and dependency. They implied that assertion deficits were both effects of and contributors to Panic Disorder with agoraphobia. Furthermore, lack of assertion is highly consistent with the association among Agoraphobia, depression, and learned helplessness.

Sexual dysfunction is another symptom of agoraphobics that has been commonly reported by researchers. In 1953, Webster found that 92% of his agoraphobic sample was sexually maladjusted. Subsequent research by Roberts (1964) and Marks and Gelder (1965) reported rates of 53% and 55%, respectively. In the study by Buglass et al. (1977), there were no reported differences in sexual adjustment between agoraphobics and controls prior to onset. However, a significant proportion of his agoraphobic sample reported diminished libido after onset.

Among agoraphobics, the primary associated symptomatology appears to combine global neuroticism and generalized anxiety. It was suggested in the earliest monographs of Westphal (1871) and Freud (1895) that agoraphobics were pervasively fearful and neurotic individuals. More recent studies have concluded that agoraphobics were more globally neurotic than normals (Goldstein, & Chambless, 1978; Marks, 1967; Solyom et al.,

1974). Goldstein and Chambless defined neuroticism by generalized anxiety, emotional immaturity and lack of self-sufficiency.

In their 1977 study, Buglass et al. found that agoraphobics had significantly higher levels of generalized, chronic anxiety than controls. This finding was strongly supported by other researchers (Chambless, 1985; Marks, 1967, 1970a; Snaith, 1968). DeMoor (1985) has characterized this chronic anxiety as a feeling of doom produced by the chronic expectation of being overwhelmed by panic at any moment.

The concept of panic-induced, chronic anxiety is consistent with Razran's theory of interoceptive conditioning (1961). He maintained that internal bodily sensations can become conditioned stimuli for fear and anxiety. Goldstein and Chambless (1978) further delineated this mechanism and suggested that:

> In the case of anxiety, a client's own physiological signs of arousal become the conditioned stimuli for the powerful conditioned response of a panic attack. Since the client is carrying the phobic stimuli with him or her always, this fear of fear is a less situation-bound, more portable phobia. Consequently, the phobia tends to generalize widely, and such clients often have high levels of so-called free floating anxiety (p. 55).

Thus, there is considerable evidence that chronic

anxiety is induced by expectation of panic. This evidence is consistent with the generally accepted line of research suggesting that spontaneous panic attacks are primary in the development of Agoraphobia and associated symptomatology.

Although it is recognized that expectation of panic can facilitate generalized anxiety, researchers such as Goldstein and Chambless (1978) would suggest that high levels of anxiety were present before the onset of panic. Although Klein postulated a biological model (1981), he also suggested that anxiety based upon fear of separation was a precursor of Panic Disorder. In 1978, Hallam suggested that Agoraphobia had not been adequately differentiated from Anxiety Neurosis. He maintained that Agoraphobia was not a unified, clinical syndrome of phobic type. Hallam speculated that Agoraphobia and associated symptomatology, including panic attacks, were a secondary manifestation of a core condition of neurotic or generalized anxiety.

Subsequent research and theory championed by Klein (1981) and Sheehan (1980) supported Hallam's assertion that Agoraphobia was not a unified, clinical syndrome, but suggested that symptomatology associated with Agoraphobia, including generalized anxiety, was secondary to a core syndrome of panic attacks. Although this theoretical orientation formed the classification strategy for the DSM-III-R, it appears premature to conclude that the etiology and pathogenesis of Panic Disorder with agoraphobia do not

involve a history of generalized or chronic anxiety. A complete investigation would require prospective evaluation of generalized anxiety in individuals who eventually develop Panic Disorder with agoraphobia, or retrospective evaluation of generalized anxiety in fully recovered agoraphobics. Unfortunately, neither type of study has been conducted.

Summary

In summary, the classification strategy for Anxiety Disorders in the DSM-III-R (APA, 1987) represented a major reconceptualization of Panic Disorder and Agoraphobia. Whereas Agoraphobia had been considered a clinical entity of phobic type, it was subsequently viewed as a secondary consequence of spontaneous, endogenous panic attacks which were considered qualitatively different than anxiety. Evidence for the distinction between panic and anxiety was found in studies of drug specificity, chemical infusion, and similarity of panic and agoraphobic patients. Theories of etiology addressing the primacy of panic attacks in the development of Agoraphobia was discussed in three areas: genetic inheritance, separation anxiety, and current interpersonal situation. Symptomatology associated with phobic avoidance and panic attacks was generally viewed as a consequence of the core syndrome, although a body of literature suggested that neurotic characteristics and

chronic anxiety predisposed individuals to Panic Disorder and Agoraphobia.

CHAPTER III

METHODOLOGY

A plan for the design and implementation of research procedures to investigate the hypotheses generated in Chapter I is presented. The population of interest is defined and sampling techniques are described, along with reliability and validity information. The design of the study and research hypotheses are described. Procedures for analytic treatment of the data are presented.

Population

The agoraphobic sample under study in the present investigation was drawn from a population of male and female outpatients who had been diagnosed with Agoraphobia with panic attacks, according to DSM-III criteria (APA, 1980), and who completed a structured, sixteen week treatment program, within the period of 1981 to 1986. Treatment strategies included: didactic instruction, systematic desensitization in vivo and in vitro, participant modeling, attendance by significant other, and individual and pharmacological therapy as needed. The specific content of each group was nearly identical. All groups met at an

outpatient clinic in Birmingham, Michigan and were conducted by the same psychologist.

As indicated in Table 1, demographic data for the agoraphobic sample was collected and revealed that 78% of the patients were female with a mean age of 32.4. The mean years of education was 13.2 with mean family income of 42,800.

Table 1: Demographic Characteristics of Agoraphobic and Nonphobic Sample Groups

	Agoraphobic	Nonphobic
Percent Female	78.3	73.3
Mean Age	32.4	34.4
Mean Years Education	13.2	14.1
Mean Family Income	42,800	45,600

The nonphobic sample was drawn from a population of individuals who had completed a four week adult education seminar in 1988. The seminar was a sampling of practical, psychological topics that included addictive behaviors, relationships, assertiveness, and stress management. The seminar was offered to the community as a public service and conducted in Birmingham, Michigan.

Demographic data was collected for the nonphobic sample and revealed that 73% of the students were female with a mean age of 34.4. The mean years of education was 14.1 with an average family income of 45,600.

There were possible limitations in generalizing from the nonphobic sample to a nonphobic population. The demographic data indicated that nonphobic participants were wealthier and better educated than the nonphobic population. Given these factors, it is likely that the sample was also more intelligent than the nonphobic population. Additionally, the seminar's emphasis on coping behaviors may have attracted a more psychopathological sample than the nonphobic population.

There were several possible limitations in generalizing findings from the agoraphobic sample to the agoraphobic population at large. Considering the evidence that agoraphobics are usually below average with respect to socioeconomic status, the demographic data indicated that phobic participants were wealthier and better educated than the phobic population. Consequently, it is likely that the phobic sample was more intelligent than the population.

Another possible limitation to generalization is that patients were out of treatment for varying lengths of time. There was no control over events during this period that may have impacted on recovery, such as, medication, individual psychotherapy, or membership in support groups.

Finally, voluntary respondents to requests for data may be systematically different from nonrespondents on the measured characteristics. For instance, it is conceivable that the most debilitated or neurotically anxious agoraphobics would not wish to complete an inventory detailing their symptoms. These considerations limit the extent to which findings from this study can be generalized to other agoraphobics who have completed a group treatment program, or other nonphobics.

Sample-Selection Criteria

The study sample consisted of three groups: posttreatment, nonrecovered agoraphobics (n=30, Group A) from the TERRAP program in Birmingham, Michigan, selected for a response pattern on Recovery and Avoidance Inventories attesting to presence of panic attacks and/or phobic avoidance in the past six months; posttreatment, fully recovered agoraphobics (n=30, Group B) from the TERRAP program in Birmingham, Michigan, selected for a response pattern on the Recovery and Avoidance Inventories attesting to absence of panic and phobic avoidance in the past six months; and a nonphobic Group (n=30, Group C) from an adult education course in Birmingham, Michigan.

Procedures

Phobic subjects in the study (Groups A and B) were recruited from all patients who completed the sixteen week treatment program within the period of 1981 to 1986. Prior to treatment, each patient had been diagnosed with Agoraphobia with panic attacks. The diagnosis was based on an extensive structured written history and a personality questionnaire. Individuals evidencing psychotic symptomatology were excluded, as were those evidencing Obsessive Compulsive or Major Depressive Disorders. The intent was to create a relatively homogenous group of agoraphobics.

For purposes of this study, all ex-patients were mailed an introductory letter and a consent form by their therapist, based upon ethical research principles of the American Psychological Association. The consent form provided a brief description of the study and procedures for confidentiality, along with a request for their consent to participate in the study. Individuals who participated returned the signed consent form to the researcher in an enclosed, addressed, stamped envelope.

Participants were then mailed a packet of materials which included the State-Trait Anxiety Inventory (Spielberger, 1970), and the Eysenck Personality Questionnaire (Eysenck, & Eysenck, 1975). Additionally, subjects were sent a 20 item Avoidance Inventory (Hardy,

1981) designed to determine the presence and severity of agoraphobic avoidance. Finally, subjects received an eight item Recovery Inventory designed for this study to identify fully recovered agoraphobics, according to criteria established in the DSM-III-R (APA, 1987). Subjects were requested to complete the inventories and return them in the enclosed, stamped envelope.

Nonphobic subjects in this study (Group C) were recruited from adults attending an adult education seminar in psychology. At the conclusion of the course, individuals were given a consent form similar to that used with the phobic groups. Those willing to be subjects and sign the consent form were given packets containing the State-Trait Anxiety Inventory, the Eysenck Personality Questionnaire, and the Avoidance Inventory. Subjects were requested to complete the inventories and return them in an enclosed, stamped envelope.

Instrumentation

Empirical Evaluation of the STAI

The State-Trait Anxiety Inventory (STAI) was developed based on a theory of acute and chronic anxiety that was first suggested by Cattell, in 1966, and modified by Spielberger (1966, 1972, 1976, 1983). They hypothesized that anxiety could be conceptualized both as an immediate, unpleasant emotional state and as a stable individual

difference or personality trait. The inventory was designed to measure anxiety levels involving both physiological arousal and subjective sensation. The complete inventory attempts to discriminate between situational and cross-situational anxiety. Separate scales for state and trait anxiety were developed, each comprising 20 questions that ask individuals about their feelings. Spielberger (1983) suggested that the A-State Form (STAI-S) measured anxiety in the present situation. This type of anxiety is presumed to fluctuate over time. The A-Trait Form (STAI-T) is presumed to measure long-term, cross situational anxiety. Subjects who score higher on the STAI-T are expected to demonstrate wider fluctuations in the STAI-S over time.

The reliability of the STAI has been demonstrated with test-retest data and measures of internal consistency. The STAI-T has consistently shown moderately high test-retest correlations over varying periods of time. The test-retest study was conducted using college students as the normative sample. Correlation coefficients in the range of 0.73 to 0.86 were calculated.

As indicated in Table 2, test-retest correlation coefficients were calculated for time periods of one hour, twenty days and 104 days. Even with the latter time period coefficients were 0.73, and 0.77 for males and females, respectively. Correlation coefficients of test-retest data

for the STAI-S were quite low, ranging from 0.16 to 0.54. Since the STAI-S was designed to measure current, situational anxiety, low test-retest correlations would be expected. Since the reliability coefficients were substantially higher for the STAI-T, it does appear to be a measure of cross situational anxiety.

Evidence of reliability and internal consistency was obtained using the Crombach (1951) modification of the Kuder-Richardson formula. Internal reliability coefficients were calculated ranging from 0.83 to 0.92. Further evidence of internal consistency was indicated by calculation of item remainder coefficients. Median item remainder coefficients for the STAI-S were 0.55 for high school students, 0.45 for college freshman, and 0.55 for college undergraduates. On the STAI-T, using the same groups, coefficients were calculated at 0.54, 0.46 and 0.53, respectively.

Validity data has also been obtained for the STAI-T. Concurrent validity was demonstrated by correlating the STAI-T with other measures of anxiety. As indicated in Table 3, the STAI-T was correlated with the Individual Perception of Anxious Trait (IPAT), the Taylor Manifest Anxiety Scale (TMAS), and the Zuckerman Affect Adjective checklist (AACL). Among college students and nonpsychiatric patients, correlations ranged from 0.53 to 0.85. These correlations were rather high and approached the test-retest

Table 2: Test-Retest Reliability Coefficients of College Undergraduates on the STAI.

	1 Hour		20 Days		104 Days	
College Students	n	r	n	r	n	r
T-Anxiety M	88	.84	38	.86	25	.73
T-Anxiety F	109	.76	75	.76	22	.77
S-Anxiety M	88	.33	38	.54	25	.33
S-Anxiety F	109	.60	75	.57	22	.31

Table 3: Concurrent Validity Coefficients for the STAI-T.

	College F			College M			NP Patients	
	n = 126			n = 80			n = 66	
Scale	STAI	IPAT	TMAS	STAI	IPAT	TMAS	STAI	IPAT
IPAT	.75			.76			.77*	
TMAS	.80	.85		.79	.73		.83	.84
AACL	.54	.57	.53	.58	.51	.41		

Note. Tables 2 and 3 from Manual for the State-Trait Anxiety Inventory STAI (Form Y) by Spielberger, C.D, 1983, Palo Alto, CA: Consulting Psychologists Press.

* n = 112 for correlation between STAI and IPAT

reliability coefficients, further suggesting the STAI-T is a measure of trait anxiety.

Empirical Evaluation of the EPQ

The Eysenck Personality Questionnaire, or EPQ (Eysenck, & Eysenck, 1975), is an updated version of the Eysenck Personality Inventory (Eysenck, 1970), to which a Psychoticism scale has been added. It is based on Eysenck's theory of personality, which emphasizes two distinct dimensions encompassing Extroversion-Introversion and Neuroticism or stability-instability. His theory presumed that psychiatric abnormalities were continuous with normality, and that neuroticism and psychoticism were discrete and independent. The EPQ is an 80-item inventory designed to measure neuroticism, psychoticism and extroversion. There is also a Lie scale, designed to explore test-taking attitude. The Neuroticism scale has been used extensively in numerous research articles.

The reliability of the EPQ has been demonstrated with test-retest data and measures of internal consistency. The Neuroticism scale has shown high test-retest correlations over varying periods of time. The primary test-retest study was conducted for a one-week period, employing 257 subjects in four different occupational groups. As indicated in Table 4, the EPQ yielded correlation coefficients principally in the range of 0.80 to 0.84, respectively.

Table 4: Test-Retest Reliability Coefficients of Different Occupations on the EPQ.

	Male				Female			
Occupation	n	P	E	N	L	n	P	E	N	L
Dental Students	80	.83	.89	.87	.90	31	.80	.88	.80	.87
Polytech Students	23	.80	.89	.92	.79	8	.78	.96	.89	.87
Social Workers	16	.79	.92	.91	.76	44	.86	.93	.86	.84
Univ. Students	17	.76	.89	.90	.90	38	.51	.80	.74	.61
Total	136	.90	.89	.86	.89	121	.71	.87	.80	.86
Grand Total	256	.78	.89	.86	.84					

Note. From Manual for the Eysenck Personality Questionnaire (p. 8) by Eysenck, H. J. & Eysenck, S. B. G., 1975, San Diego, CA: EdITS/Educational and Industrial Testing Service.

Table 5: Reliability Coefficients of Internal Consistency on the EPQ

Scale	Random Group	Criminals	Random Group	Criminals
P	.74	.71	.68	.77
E	.85	.84	.84	.86
N	.84	.84	.85	.88
L	.81	.82	.79	.86
n in sample	500	934	500	71

Note. From Manual for the Eysenck Personality Questionnaire (p. 8) by Eysenck, H. J., & Eysenck, S. B. G., 1975, San Diego, CA: EdITS/Educational and Industrial Testing Service.

Evidence of internal consistency was obtained using the Crombach (1951) modification of the Kuder-Richardson formula. As indicated in Table 5, the items within each scale were correlated by sex for normal and criminal groups. Correlation coefficients ranged principally from 0.80 to 0.90. The Neurotic scale was especially consistent, with coefficients ranging from 0.84 to 0.88.

Validity data has also been obtained for the EPQ. Concurrent validity has been demonstrated for the Neurotic scale with moderately high correlations between the EPQ and other measures of neuroticism. Additionally, in a criterion group study, scores on the EPQ discriminated between normals and diagnosed neurotics. It appears that the Neurotic scale of the EPQ is an excellent index of neuroticism.

Empirical Evaluation of the Avoidance Inventory

The Avoidance Inventory is comprised of 20 questions designed to measure the existence and severity of current agoraphobic avoidance. It was developed by Arthur Hardy (1981) as part of the Comprehensive Phobic Evaluation questionnaire administered to all prospective patients prior to treatment in a sixteen-week group program. The Avoidance Inventory encompasses virtually all situations related to agoraphobic avoidance and evaluates mobility, both alone and accompanied.

Reliability data for the Avoidance Inventory had not been previously obtained. For the purposes of this study,

evidence of reliability and internal consistency was obtained using the Crombach (1951) modification of the Kuder-Richardson formula. Internal reliability coefficients of 0.85, 0.89 and 0.91 were calculated for the nonrecovered, nonphobic and fully recovered groups, respectively, suggesting a high level of internal consistency.

Validity data for the Avoidance Inventory had not been previously obtained. However, it has extremely high face validity as there is a well-defined consensus among researchers regarding common agoraphobic situations (Chambless, 1985; Marks, 1967; Marks, & Herst, 1970; Thorpe, 1977), and the Avoidance Inventory is fully representative of this consensus. Additionally, the treating psychologist indicated that all patients who responded to the inventory in an agoraphobic manner were clearly phobic during treatment.

Empirical Evaluation of the Recovery Inventory

The Recovery Inventory is comprised of eight questions designed to discriminate fully recovered from nonrecovered agoraphobics, according to the criteria in the DSM-III-R. It was designed for this study as a companion to the Avoidance Inventory. It measures the length of time a patient has been free of phobic avoidance and Panic Disorder. The DSM-III-R requires a six-month absence of panic attacks and phobic symptomatology for consideration of full recovery. The first two items determine the presence

of panic attacks in the past six months, along with a description of the most severe attack, according to DSM-III-R criteria. The next two items determine the presence of phobic avoidance within the past six months. Items five, six and seven determine change in phobic symptomatology in the past six months. These items were predicated on the fact that agoraphobic symptomatology should not change in the previous six months in a fully recovered agoraphobic. The final item allows for independent confirmation of recovery from the subject's significant other.

Reliability data for the Recovery Inventory had not been previously obtained. Calculation of correlation coefficients for reliability and internal consistency was not appropriate given the hierarchical criterion nature of the inventory. Since the inventory required a discrete criterion response across all items for consideration of full recovery, there could be no variation of scores among fully recovered individuals. The responses of individuals who fulfilled the criterion would necessarily yield a correlation coefficient of 1.

Visual inspection of the initial data revealed that an unusually high number of individuals (12 of 30) who responded in a nonphobic manner to the first seven items could not report independent confirmation of recovery on item eight. Anecdotal evidence from these and other participants strongly suggested that independent

confirmation was not a reliable index of recovery. Consequently, response to item eight was eliminated from the hierarchical criterion procedure.

Validity data for the Recovery Inventory had not been previously obtained but it has extremely high face validity. The questions were directly predicated upon the criteria listed in the DSM-III-R with the exception of the eighth item, which was subsequently eliminated.

Design

The design for this study was descriptive and retrospective in nature. All subjects were administered the STAI-T, the EPQ-N and the Avoidance Inventory. Additionally, phobic subjects were administered the Recovery Inventory. Phobic groups were differentiated through a hierarchical criterion procedure. The first criterion for inclusion in the fully recovered group was an appropriate set of responses to the Recovery Inventory, as follows: an indication on the first item that the previous six months had been panic-free; absence of symptom identification on the second item; a negative response on the third and fourth items, indicating a six-month absence of phobic avoidance; and an affirmative response on the fifth, sixth and seventh items, indicating a six-month absence of change in phobic severity. Participants who fulfilled the criterion for full recovery of the Recovery Inventory potentially constituted

the fully recovered group, reportedly free from panic and phobic avoidance for six months.

The second criterion for inclusion in the fully recovered group was an appropriate set of responses to the Avoidance Inventory. With this measure, individuals were required to rank, on a likert scale of 0-5, degree of discomfort in 20 common agoraphobic situations. The total score of each potentially fully recovered agoraphobic was contrasted with the mean of the scores for the nonphobic group, using a one-tailed z test. The alpha level selected for significance was $p<.05$. Individuals whose scores were within the normal range at the .05 level of significance constituted the fully recovered group (Group B).

The criteria for membership in the nonrecovered group was also based on responses to the Recovery and Avoidance Inventories. Individuals who did not fulfill the criterion of all seven items on the Recovery Inventory, or whose subsequent scores on the Avoidance Inventory were not within the normal range, constituted the nonrecovered group (Group A). This design provided rigorous criteria for group membership, particularly in the fully recovered group. Thus, the possibility of Type I error appeared to be minimized.

For purposes of data analysis, equal cell sizes were ensured ($n = 30$) for all three groups by randomly discarding individual information until the number in each group

totalled 30. This process entailed elimination of data from 11 nonrecovered agoraphobics and 4 nonphobics.

Testable Hypotheses

1. Nonrecovered agoraphobics will score higher on the STAI-T than nonphobics.
2. Nonrecovered agoraphobics will score higher on the STAI-T than fully recovered agoraphobics.
3. Fully recovered agoraphobics will score higher on the STAI-T than nonphobics.
4. Nonrecovered agoraphobics will score higher on the EPQ-N than nonphobics.
5. Nonrecovered agoraphobics will score higher on the EPQ-N than fully recovered agoraphobics.
6. Fully recovered agoraphobics will score higher on the EPQ-N than nonphobics.

Analysis of the Data

In the manner specified above, the three groups (Group A, Group B, and Group C) were contrasted on the STAI-T and the EPQ-N using one way analysis of variance procedures for planned comparisons. This procedure was selected because of the independent nature of the groups. Demographic homogeneity was enhanced through the selection of adult subjects from the same city area.

The significance level for acceptance/rejection of all null hypotheses was set at alpha = $p<.05$.

The Statistical Package for the Social Sciences (SPSS) was the computer program used for data analysis.

CHAPTER IV

ANALYSIS OF RESULTS

In this chapter, the results of the investigation are reported. The research hypotheses are presented along with the relevant statistical analysis. Subsequently, acceptance or rejection of each hypothesis is indicated.

Participants in this study were nonrecovered agoraphobics, fully recovered agoraphobics, and a nonphobic group. The agoraphobic sample groups each comprised 30 individuals drawn from 414 outpatients who had completed the TERRAP treatment program in Birmingham, Michigan. The sample of 30 participants who comprised the nonphobic group was drawn from 57 individuals who had completed an adult education course in Birmingham, Michigan.

All participants in the agoraphobic sample was administered the Recovery Inventory and the Avoidance Inventory to discriminate between fully recovered and nonrecovered agoraphobics. Additionally, all participants completed the State-Trait Anxiety Inventory-Trait Form (STAI-T) and the Eysenck Personality Questionnaire-Neuroticism scale (EPQ-N).

Results of Hypothesis Tests

The analysis of variance (ANOVA) statistic was used to test the hypotheses concerning personality differences among nonrecovered agoraphobics, fully recovered agoraphobics and nonphobics. The first three hypotheses were designed to analyze differences in trait anxiety among groups, while the last three hypotheses were designed to analyze differences in global neuroticism among groups. The six hypotheses are presented in two sets (1 with 2 and 3, 4 with 5 and 6), with each set reflecting the specific dependent measure used. Following each set of hypotheses is the empirical data related to acceptance or rejection of each of the three hypotheses. In all analyses, an alpha level of $p<.05$ was required for consideration of statistical significance.

Hypotheses Investigating Differences Between Groups on STAI-T Scores

Alternate Hypothesis 1: Nonrecovered agoraphobics will score higher than nonphobics on the STAI-T.

Alternate Hypothesis 2: Nonrecovered agoraphobics will score higher than fully recovered agoraphobics on the STAI-T.

Alternate Hypothesis 3: Fully recovered agoraphobics will score higher than nonphobics on the STAI-T.

The means and standard deviations of the STAI-T scores along with the cell sizes used to test Hypotheses 1, 2, and 3 are listed in Table 6. The results of the one-way

Table 6: STAI-T Scores of Agoraphobic and Nonphobic Groups

Group	n	X	SD
Nonrecovered Agoraphobics	30	50.53	8.92
Fully Recovered Agoraphobics	30	42.3	9.7
Nonphobics	30	42.17	11.34

Table 7: One Way Analysis of Variance for Agoraphobic and Nonphobic Groups on STAI-T Scores

Source	SS	df	MS	F	p
Between Groups	1378.07	2	689.03	6.84	.0017
Within Groups	8763.93	87	100.73		
Total	10,142.00	89			

analysis of variance for Hypotheses 1, 2, and 3 are listed in Table 7.

The results of the one-way ANOVA indicated that there was a significant difference ($p<.01$) among the groups on mean STAI-T scores. Post-hoc analysis was performed using the Scheffe procedure to determine which groups differed. The results indicated that the mean score of nonrecovered agoraphobics on the STAI-T was significantly higher than the mean score of the nonphobics. Thus, for Hypothesis 1, the null hypothesis was rejected in favor of the alternate hypothesis. Additionally, the mean score of nonrecovered agoraphobics on the STAIT-T was significantly higher than the mean score of fully recovered agoraphobics. Thus, for Hypothesis 2, the null hypothesis is rejected in favor of the alternate hypothesis. However, there was no significant difference in mean score between fully recovered agoraphobics and nonphobics. Thus, for Hypothesis 3, the null hypothesis was not rejected.

Hypotheses Investigating Differences Between Groups on EPQ-N Scores

Alternate Hypothesis 4: Nonrecovered agoraphobics will score higher than nonphobics on the EPQ-N.

Alternate Hypothesis 5: Nonrecovered agoraphobics will score higher than fully recovered agoraphobics on the EPQ-N.

Alternate Hypothesis 6: Fully recovered agoraphobics will score higher than nonphobics on the EPQ-N.

The means and standard deviations of the Neuroticism scale of the EPQ, along with the cell sizes used to test Hypotheses 4, 5, and 6 are listed in Table 8. The results of the analysis of variance for Hypotheses 4, 5, and 6 are listed in Table 9.

The results of the one-way ANOVA indicated that there was a significant difference ($p<.01$) among the groups on EPQ-N scores. Post-hoc analysis was performed using the Scheffe procedure to determine which groups differed. The results indicated that the mean score of nonrecovered agoraphobics on the EPQ-N was significantly higher than the mean score of nonphobics. Thus, for Hypothesis 4, the null hypothesis is rejected in favor of the alternate hypothesis. Additionally, the mean score of nonrecovered agoraphobics on the EPQ-N was significantly higher than the mean score of fully recovered agoraphobics. Thus, for Hypothesis 5, the null hypothesis is rejected in favor of the alternate hypothesis. However, there was no significant difference between fully recovered agoraphobics and nonphobics on EPQ-N scores. Thus, for Hypothesis 6, the null hypothesis was not rejected.

Table 8: EPQ-N Scores of Agoraphobic and Nonphobic Groups

Group	n	X	SD
Nonrecovered Agoraphobics	30	16.83	5.06
Fully Recovered Agoraphobics	30	12.77	5.22
Nonphobics	30	13.23	5.19

Table 9: One-Way Analysis of Variance for Agoraphobics and Nonphobics on EPQ-N Scores

Source	SS	df	MS	F	p
Between Groups	297.16	2	148.58	5.59	.005
Within Groups	2312.9	87	26.6		
Total	2610.06	89			

Summary of the Hypothesis Tests

In this chapter, the results of the investigation were reported. Hypothesized personality differences among nonrecovered agoraphobics, fully recovered agoraphobics and nonphobics were examined. The results of the hypothesis testing were as follows:

Hypotheses	Results
Alternate Hypothesis 1: Nonrecovered agoraphobics will score higher than nonphobics on the STAI-T.	Accepted
Alternate Hypothesis 2: Nonrecovered agoraphobics will score higher than fully recovered agoraphobics on the STAI-T.	Accepted
Alternate Hypothesis 3: Fully recovered agoraphobics will score higher than nonphobics on the STAI-T.	Not Accepted
Alternate Hypothesis 4: Nonrecovered agoraphobics will score higher than nonphobics on the EPQ-N.	Accepted
Alternate Hypothesis 5: Nonrecovered agoraphobics will score higher than fully recovered agoraphobics on the EPQ-N.	Accepted
Alternate Hypothesis 6: Fully recovered agoraphobics will score higher than nonphobics on the EPQ-N.	Not Accepted

CHAPTER V

SUMMARY, CONCLUSIONS, AND IMPLICATIONS

Summary

Review of the Problem

The purpose of this investigation was to evaluate nonrecovered agoraphobics, fully recovered agoraphobics and nonphobics on measures of anxiety and neuroticism. The study design allowed for conclusions regarding the primacy of panic attacks, phobic avoidance, anxiety, and neuroticism in the development of Panic Disorder with agoraphobia. If neurotic symptomatology was only a consequence of the core syndrome (panic attacks and phobic avoidance), as suggested by Klein (1980, 1981) and Sheehan (1982a, 1983) then the DSM-III-R (APA, 1987) would provide an accurate classification scheme. Cost effective treatment would necessarily emphasize amelioration of panic attacks and phobic avoidance. However, if panic attacks and phobic avoidance were a partial consequence of underlying chronic anxiety, then cost effective treatment might place slightly more emphasis on amelioration of anxiety and neuroticism. Additionally, treatment termination might be premature upon mere cessation of panic attacks and phobic avoidance. These

diagnostic and clinical implications provided the impetus for this study.

Review of the Literature

A review of the literature was conducted in four related areas. First, the identification of agoraphobia as a clinical syndrome was discussed. Originally coined by Westphal in 1871, the term Agoraphobia had been referred to as locomotor anxiety (Abraham, 1913); street fear (Miller, 1913); phobic anxiety depersonalization syndrome (Roth, 1959); phobic anxious states (Klein, 1964); and nonspecific insecurity fears (Snaith, 1968). In 1980, the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) (APA) emphasized the phobic component and classified Agoraphobia as a Phobic Disorder with diagnostic features that included a marked fear or avoidance of being alone or in public places from which escape might be difficult or help not available in case of sudden incapacitation, and increasing constriction of normal activities (APA, 1980).

The second area of review involved a description of the research that prompted a change in the predominant theory of agoraphobia which was incorporated, subsequently, into the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R) (APA, 1987). Based primarily on the theories of Klein (1980, 1981) and Sheehan (1982b, 1983), the DSM-III-R emphasized the panic component of

agoraphobia by classifying Panic Disorder with agoraphobia as an Anxiety Disorder. The diagnostic features of Panic Disorder with agoraphobia require presence of full blown panic attacks (Panic Disorder) and subsequent fear of places or situations from which escape might be difficult or embarrassing, or assistance not available if needed.

The third area investigated in the literature review was prevalence and etiology of Agoraphobia. The most accurate lifetime estimates of prevalence for Agoraphobia with panic attacks ranged from approximately 2-5% of the population (Myers et al., 1984; Robbins et al., 1984). Marks (1970a) indicated that onset usually occurred between the ages of 18 and 35 (Marks, 1970a). Klein (1980, 1981) and Sheehan (1982b, 1983) championed the most widely accepted view of etiology. They postulated a genetic predisposition toward Panic Disorder, with agoraphobia an emergent consequent of spontaneous, endogenous panic attacks.

The fourth area of review was the relationship between Panic Disorder with Agoraphobia and associated symptomatology. Researchers had consistently identified the following symptoms: chronic anxiety, global neuroticism, depression, obsessive compulsiveness, hypochondriasis, dependency, sexual dysfunction and assertion deficits. The models of Klein and Sheehan theorized that these symptoms were solely the product of the core syndrome of Panic

Disorder with agoraphobia. However, other researchers concluded that chronic anxiety and global neuroticism contributed to the development of both Panic Disorder and Agoraphobia (Bowlby, 1980, 1984; Goldstein, & Chambless, 1978).

Review of the Procedures

The current study was designed to contrast nonrecovered agoraphobics, fully recovered agoraphobics, and nonphobics on measures of anxiety and neuroticism. First, the nonrecovered and fully recovered agoraphobics were differentiated through the use of the Avoidance Inventory (Hardy, 1981) and a Recovery Inventory developed for this study. A hierarchical criterion procedure was employed which initially required an appropriate set of responses to the Recovery Inventory. Subsequently, individual scores on the Avoidance Inventory were contrasted with the mean of scores for the nonphobic group using a one-tailed *z*-Test. Individuals whose scores were within the normal range at the .05 significance level comprised the fully recovered group. Individuals who were excluded by the hierarchical procedure comprised the nonrecovered group.

The sample of agoraphobics (Groups A and B) consisted of 60 outpatients who had been diagnosed with Agoraphobia with panic attacks and who completed a structured, sixteen-week treatment program in Birmingham, Michigan. The sample of nonphobics (Group C) consisted of 30 individuals who had

of nonphobics (Group C) consisted of 30 individuals who had completed an adult education course in Birmingham, Michigan.

The dependent measures in this study were scores on the State-Trait Anxiety Inventory-Trait Form (STAI-T) and the Eysenck Personality Questionnaire-Neuroticism scale (EPQ-N). The three groups were contrasted on each dependent measure using a one-way analysis of variance procedure.

Review of the Results

Hypotheses 1 and 4 were designed to determine differences between nonrecovered agoraphobics and nonphobics in chronic anxiety and global neuroticism, respectively. For both hypotheses, the null hypothesis was rejected ($p<.05$) and the alternate hypothesis accepted. Nonrecovered agoraphobics evidenced more anxiety and neuroticism than a demographically similar nonphobic group.

Hypotheses 2 and 5 were designed to determine differences between nonrecovered agoraphobics and fully recovered agoraphobics in chronic anxiety and global neuroticism, respectively. For both hypotheses, the null hypothesis was rejected ($p<.05$) and the alternate hypothesis accepted. Nonrecovered agoraphobics evidenced more anxiety and neuroticism than fully recovered agoraphobics.

Hypotheses 3 and 6 were designed to determine differences between fully recovered agoraphobics and nonphobics in chronic anxiety and global neuroticism, respectively. For both hypotheses, the null hypothesis was

evidence more anxiety and neuroticism than a demographically similar, nonphobic group.

Conclusions and Discussion

Three major conclusions can be drawn on the basis of the results of the hypothesis tests. First, nonrecovered agoraphobics are significantly more anxious and neurotic than nonphobics. Previous treatment experience does not need to be considered when examining the relationship between Panic Disorder with agoraphobia and associated symptomatology of anxiety and neuroticism. This conclusion validates the findings and methodology of Buglass (1977), Chambless (1982, 1985), Marks (1967), Jasin (1981), Sheehan and his associates (1983), and other researchers who evaluated associated symptomatology in agoraphobics without determining the presence of previous unsuccessful treatment.

Another conclusion that can be drawn from the results of the hypothesis tests is that nonrecovered agoraphobics are significantly more anxious and neurotic than fully recovered agoraphobics. This conclusion helps validate the research of Chambless (1985) who concluded that as frequency of panic attacks and avoidance behavior increased in a population so did anxiety and neuroticism.

Finally, it can be concluded from the results that fully recovered agoraphobics are not significantly more anxious or neurotic than nonphobics. Given the restrictions

of the data, agoraphobics appear to recover completely with respect to panic attacks, phobic avoidance, anxiety, and neuroticism. It is also suggested that treatment ameliorates panic attacks and phobic avoidance without maintaining the presence of anxiety and neuroticism.

Several inferences can be drawn from the major conclusions. If a high index of anxiety and neuroticism predisposes an individual to the development of Panic disorder with agoraphobia, we would anticipate the possibility of complete recovery from panic attacks and phobic avoidance in the presence of significant, if moderate, levels of anxiety and neuroticism. If Panic Disorder with agoraphobia were a potential secondary consequence of high levels of anxiety and neuroticism, it is reasonable to anticipate that treatment might ameliorate panic attacks and phobic avoidance without completely ameliorating anxiety and neuroticism. Thus, the results of this study are inconsistent with Hallam's theory that agoraphobia and panic attacks are a secondary manifestation of a core condition of chronic or neurotic anxiety. Additionally, these results do not lend credence to Goldstein and Chambless' theory that a low level of self sufficiency due to anxiety and/or lack of skills is a precursor to the development of agoraphobia.

However, the results of the present study are consistent with the models of agoraphobia championed by

Klein (1980, 1981) and Sheehan (1982b, 1983) that postulate that associated psychopathology is an emergent consequent of panic attacks and phobic avoidance. These researchers have maintained that anxiety and neuroticism are produced by the fearful expectation of panic attacks and their unpredictable, spontaneous nature. These researchers have postulated a linear, biological model of etiology which maintains: that panic attacks are qualitatively different than anxiety and biologically determined; that agoraphobia is a secondary manifestation of the clinical syndrome of Panic Disorder; and that associated psychopathology is solely an emergent consequent of Panic Disorder with agoraphobia.

Finally, the results of this investigation are consistent with the current classification scheme for agoraphobia in the DSM-III-R (APA, 1987), which is based primarily on the models of Klein (1980, 1981) and Sheehan (1983). The classification scheme presumes that associated anxiety and neuroticism are a product of the core condition of panic attacks and phobic avoidance. In the DSM-III-R, agoraphobia is considered a secondary consequence of Panic Disorder when it is associated with panic attacks. The DSM-III-R classifies Panic Disorder with agoraphobia as a subtype of Anxiety Disorder, but also as a clinical entity distinct from chronic anxiety. Accurate diagnosis requires recognition and assessment of the core syndrome of panic

recognition and assessment of the core syndrome of panic attacks and phobic avoidance.

Limitations

The primary limitation of this study is it's reliance upon the self-report method for collecting data about the participants. Evaluation of degree of recovery and associated psychopathology was performed on the basis of paper and pencil measures without an interview, behavioral observations, or physiological measures. The generally apparent nature of the measures provided an opportunity for manipulation of the results, which required that interpretations be made with caution.

Another limitation of this study was it's reliance on a previous diagnosis of outpatients for participation in the TERRAP program. The screening therapist made clinical diagnoses on the basis of self-report measures without an interview, behavioral observations, or physiological measures. The potential existed for manipulation of the measures to facilitate admission into the program. Additionally, the criteria employed for diagnosis was based on the original DSM-III (APA, 1980) which categorized Agoraphobia as a distinct clinical entity of phobic type.

A related limitation of this study is common to most research conducted with humans. Voluntary respondents to requests for data may be systematically different than

is possible that fully recovered agoraphobics who might still be chronically anxious would not wish to confront this apparent discrepancy.

Another limitation of this study is that it was not determined if participants were taking medication which might have mood altering or psychotropic effects. An individual taking imipramine or alprazolam may fulfill the criteria for full recovery in the DSM-III-R, but not actually be considered completely recovered. It is possible that an individual taking medication might not experience panic attacks or phobic avoidance but would again if withdrawn from the medication.

Implications for Future Research and Practice

Implications for Future Research

Initially, this study should be replicated using methodology that would address it's limitations. The reliability and validity of participant diagnosis could be enhanced by including an interview and behavioral observations. Diagnostic criteria for initial screening and subsequent recovery should reflect changes incorporated in the DSM-III-R. The criteria for full recovery should include the absence of mood altering or psychotropic medication. Future research should employ additional dependent measures in examining previously demonstrated symptomatology associated with Panic Disorder with

agoraphobia. Instruments should be employed which measure depression, hypochondriasis, assertion deficits and sexual dysfunction.

This researcher's clinical experience suggests that many apparently fully recovered agoraphobics subsequently retrogress to panic attacks and/or phobic avoidance. A prospective study which evaluated fully recovered agoraphobics on measures of associated symptomatology and later contrasted retrogressed with recovered individuals on the measures of the original evaluation would be useful in evaluating the nature of recovery.

However, in analyzing the relationship between anxiety and Panic Disorder with Agoraphobia, the most powerful future research would consist of a longitudinal study of individuals with a neurotic diagnosis such as Generalized Anxiety Disorder. These individuals could be contrasted with a nonanxious group to determine the relative incidence of subsequent development of Panic Disorder with Agoraphobia.

Implications for Future Practice

The empirical investigation of Panic Disorder with agoraphobia and associated symptomatology found a significant difference in chronic anxiety and global neuroticism between nonrecovered agoraphobics and both fully recovered agoraphobics and nonphobics. No difference in chronic anxiety or global neuroticism was found between

fully recovered agoraphobics and nonphobics. These results were interpreted as consistent with the theory that associated symptomatology was an emergent consequent of Panic Disorder with Agoraphobia. Further, it was suggested that complete recovery from Panic Disorder with Agoraphobia yielded elimination of associated symptomatology.

Thus, it appears that treatment of patients with a diagnosis of Panic Disorder with agoraphobia should emphasize amelioration of panic frequency and intensity along with phobic avoidance. Cost effective treatment should consist of directed strategies to facilitate recovery from the core syndrome. These strategies include the following: education, stimulus control, relaxation training, systematic desensitization, and exposure with repeated practice. Additionally, a lifestyle shift should be encouraged which is likely to decrease the probability of panic attacks; such a shift would involve cognitions, assertion, chemical consumption, diet, and exercise.

Cost effective treatment of Panic Disorder with agoraphobia would appear to emphasize amelioration of panic attacks and phobic avoidance. Treatment discharge appears appropriate upon cessation of panic attacks and phobic avoidance, with the expectation that associated symptomatology would also be ameliorated.

The theory that Panic Disorder with agoraphobia is primary in the development of associated symptomatology has

major implications for clinical practice. Effective practice requires that the clinician accurately diagnose and treat the patient when presented with a complex constellation of symptoms. While the severity and diversity of symptomatology can be overwhelming, the clinician should remember that agoraphobics appear to recover across the constellation when the core syndrome is ameliorated.

APPENDICES

APPENDIX A

STATE-TRAIT ANXIETY INVENTORY (FORM Y)

(SELF EVALUATION QUESTIONNAIRE)

SELF-EVALUATION QUESTIONNAIRE

Developed by Charles D. Spielberger
in collaboration with
R. L. Gorsuch, R. Lushene, P. R. Vagg, and G. A. Jacobs

STAI Form Y-1

Name _____ Date _____ S ____

Age _____ Sex: M ____ F ____ T ____

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 feel *right* now, that is, *at this moment*. 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 your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	①	②	③	④
2. I feel secure	①	②	③	④
3. I am tense	①	②	③	④
4. I feel strained	①	②	③	④
5. I feel at ease	①	②	③	④
6. I feel upset	①	②	③	④
7. I am presently worrying over possible misfortunes	①	②	③	④
8. I feel satisfied	①	②	③	④
9. I feel frightened	①	②	③	④
10. I feel comfortable	①	②	③	④
11. I feel self-confident	①	②	③	④
12. I feel nervous	①	②	③	④
13. I am jittery	①	②	③	④
14. I feel indecisive	①	②	③	④
15. I am relaxed	①	②	③	④
16. I feel content	①	②	③	④
17. I am worried	①	②	③	④
18. I feel confused	①	②	③	④
19. I feel steady	①	②	③	④
20. I feel pleasant	①	②	③	④

APPENDIX B

EYSENCK PERSONALITY QUESTIONNAIRE

EPQ

(Adult)

Name _____ Age _____ Sex _____

Occupation _____ Date _____

Firm _____ Marital Status _____

Health Status _____

Weight _____ Height _____ Code _____

INSTRUCTIONS

Please answer each question by marking an [X] beside the "YES" or the "NO" following the question. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the question.

PLEASE REMEMBER TO ANSWER EACH QUESTION

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BOX 7234, SAN DIEGO, CALIFORNIA 92107

IN EVERY QUESTION, MARK JUST ONE BOX.

1. Do you have many different hobbies? YES ☐ NO ☐
2. Do you stop to think things over before doing anything? YES ☐ NO ☐
3. Does your mood often go up and down? YES ☐ NO ☐
4. Have you ever taken the praise for something you knew someone else had really done? YES ☐ NO ☐
5. Are you a talkative person? YES ☐ NO ☐
6. Would being in debt worry you? YES ☐ NO ☐
7. Do you ever feel "just miserable" for no reason? YES ☐ NO ☐
8. Were you ever greedy by helping yourself to more than your share of anything? YES ☐ NO ☐
9. Do you lock up your house carefully at night? YES ☐ NO ☐
10. Are you rather lively? YES ☐ NO ☐
11. Would it upset you a lot to see a child or an animal suffer? YES ☐ NO ☐
12. Do you often worry about things you should not have done or said? YES ☐ NO ☐
13. If you say you will do something, do you always keep your promise no matter how inconvenient it might be? YES ☐ NO ☐
14. Can you usually let yourself go and enjoy yourself at a lively party? YES ☐ NO ☐
15. Are you an irritable person? YES ☐ NO ☐
16. Have you ever blamed someone for doing something you knew was really your fault? YES ☐ NO ☐
17. Do you enjoy meeting new people? YES ☐ NO ☐
18. Do you believe insurance plans are a good idea? YES ☐ NO ☐
19. Are your feelings easily hurt? YES ☐ NO ☐
20. Are *all* your habits good and desirable ones? YES ☐ NO ☐
21. Do you tend to keep in the background on social occasions? YES ☐ NO ☐
22. Would you take drugs which may have strange or dangerous effects? YES ☐ NO ☐
23. Do you often feel "fed-up"? YES ☐ NO ☐
24. Have you ever taken anything (even a pin or button) that belonged to someone else? YES ☐ NO ☐
25. Do you like going out a lot? YES ☐ NO ☐
26. Do you enjoy hurting people you love? YES ☐ NO ☐
27. Are you often troubled about feelings of guilt? YES ☐ NO ☐
28. Do you sometimes talk about things you know nothing about? YES ☐ NO ☐
29. Do you prefer reading to meeting people? YES ☐ NO ☐
30. Do you have enemies who want to harm you? YES ☐ NO ☐
31. Would you call yourself a nervous person? YES ☐ NO ☐
32. Do you have many friends? YES ☐ NO ☐
33. Do you enjoy practical jokes that can sometimes really hurt people? YES ☐ NO ☐
34. Are you a worrier? YES ☐ NO ☐
35. As a child did you do as you were told immediately and without grumbling? YES ☐ NO ☐
36. Would you call yourself happy-go-lucky? YES ☐ NO ☐
37. Do good manners and cleanliness matter much to you? YES ☐ NO ☐
38. Do you worry about awful things that might happen? YES ☐ NO ☐
39. Have you ever broken or lost something belonging to someone else? YES ☐ NO ☐
40. Do you usually take the initiative in making new friends? YES ☐ NO ☐
41. Would you call yourself tense or "highly-strung"? YES ☐ NO ☐
42. Are you mostly quiet when you are with other people? YES ☐ NO ☐
43. Do you think marriage is old-fashioned and should be done away with? YES ☐ NO ☐
44. Do you sometimes boast a little? YES ☐ NO ☐
45. Can you easily get some life into a rather dull party? YES ☐ NO ☐

GO RIGHT ON TO THE NEXT PAGE.

46. Do people who drive carefully annoy you? YES ☐ NO ☐
47. Do you worry about your health? YES ☐ NO ☐
48. Have you ever said anything bad or nasty about anyone? YES ☐ NO ☐
49. Do you like telling jokes and funny stories to your friends? YES ☐ NO ☐
50. Do most things taste the same to you? YES ☐ NO ☐
51. As a child did you ever talk back to your parents? YES ☐ NO ☐
52. Do you like mixing with people? YES ☐ NO ☐
53. Does it worry you if you know there are mistakes in your work? YES ☐ NO ☐
54. Do you suffer from sleeplessness? YES ☐ NO ☐
55. Do you always wash before a meal? YES ☐ NO ☐
56. Do you nearly always have a "ready answer" when people talk to you? YES ☐ NO ☐
57. Do you like to arrive at appointments in plenty of time? YES ☐ NO ☐
58. Have you often felt listless and tired for no reason? YES ☐ NO ☐
59. Have you ever cheated at a game? YES ☐ NO ☐
60. Do you like doing things in which you have to act quickly? YES ☐ NO ☐
61. Is (or was) your mother a good woman? YES ☐ NO ☐
62. Do you often feel life is very dull? YES ☐ NO ☐
63. Have you ever taken advantage of someone? YES ☐ NO ☐
64. Do you often take on more activities than you have time for? YES ☐ NO ☐
65. Are there several people who keep trying to avoid you? YES ☐ NO ☐
66. Do you worry a lot about your looks? YES ☐ NO ☐
67. Do you think people spend too much time safeguarding their future with savings and insurances? YES ☐ NO ☐
68. Have you ever wished that you were dead? YES ☐ NO ☐
69. Would you dodge paying taxes if you were sure you could never be found out? YES ☐ NO ☐
70. Can you get a party going? YES ☐ NO ☐
71. Do you try not to be rude to people? YES ☐ NO ☐
72. Do you worry too long after an embarrassing experience? YES ☐ NO ☐
73. Have you ever insisted on having your own way? YES ☐ NO ☐
74. When you catch a train do you often arrive at the last minute? YES ☐ NO ☐
75. Do you suffer from "nerves"? YES ☐ NO ☐
76. Do your friendships break up easily without it being your fault? YES ☐ NO ☐
77. Do you often feel lonely? YES ☐ NO ☐
78. Do you always practice what you preach? YES ☐ NO ☐
79. Do you sometimes like teasing animals? YES ☐ NO ☐
80. Are you easily hurt when people find fault with you or the work you do? YES ☐ NO ☐
81. Have you ever been late for an appointment or work? YES ☐ NO ☐
82. Do you like plenty of bustle and excitement around you? YES ☐ NO ☐
83. Would you like other people to be afraid of you? YES ☐ NO ☐
84. Are you sometimes bubbling over with energy and sometimes very sluggish? YES ☐ NO ☐
85. Do you sometimes put off until tomorrow what you ought to do today? YES ☐ NO ☐
86. Do other people think of you as being very lively? YES ☐ NO ☐
87. Do people tell you a lot of lies? YES ☐ NO ☐
88. Are you touchy about some things? YES ☐ NO ☐
89. Are you always willing to admit it when you have made a mistake? YES ☐ NO ☐
90. Would you feel very sorry for an animal caught in a trap? YES ☐ NO ☐

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED ALL THE QUESTIONS

APPENDIX C

AVOIDANCE INVENTORY

AVOIDANCE INVENTORY

Some people have difficulty coping with certain situations. Please indicate on the scale 0-5 below the degree to which you are affected by the following situations.

0) No problem
1) Prefer not to
2) Can with someone and feel comfortable; alone feel extremely anxious
3) Can with someone but feel uncomfortable
4) Can with someone but feel very anxious
5) cannot without feeling panicky, producing panic

	0	1	2	3	4	5
1. Can you sit in the middle of a row of people such as in a movie or church?						
2. Can you go into unfamiliar places?						
3. Can you use elevators?						
4. Can you allow yourself to get in situations where you feel trapped?						
5. Do you fear having a reaction in public?						
6. Can you eat in restaurants?						
7. Can you tolerate crowds?						
8. Can you tolerate closed-in spaces?						
9> Can you tolerate heights?						
10. Can you cross bridges?						
11. Can you fly in airplanes?						
12. Can you go to parties?						
13. Can you go through the line in supermarkets?						
14. Can you sign your name in front of someone?						
15. Can you drive the freeways?						
16. Can you shop in department stores?						
17. Can you tolerate being alone?						
18. Can you tolerate wide open spaces?						
19. Can you go to work?						
20. Can you use escalators?						

APPENDIX D

RECOVERY INVENTORY

RECOVERY INVENTORY

1. Has it been at least six months since your last panic attack?

2. If you had 1 or more panic attacks in the past six months, please check the symptoms associated with the worst one:

   a. Shortness of breath
   b. Dizziness, feeling unsteady or feeling faint
   c. Heart palpitations or fast heart rate
   d. Trembling or shaking
   e. Sweating
   f. Choking
   f. Nausea or upset stomach
   h. Feelings or unreality (derealization)
   i. Feelings of watching yourself (depersonalization)
   j. Numbness or tingling sensations
   k. Flushes, hot flashes or chills
   l. Chest pain or discomfort
   m. Fear of dying
   n. Fear of going crazy or doing something uncontrolled

3. Have you engaged in agoraphobic avoidance in the past six months?

4. Has your level agoraphobic avoidance changed much in the past six months?

5. Were your responses to the Avoidance Inventory about the same as they would have been six months ago?

6. Have you recovered to at least your pre-agoraphobic level of avoidance (recognizing that these is a continuum from phobic to non-phobic)?

7. Have you recovered to this extent for six months?

8. Does your significant other believe that you have recovered to at least your pre-agoraphobic level of avoidance.

APPENDIX E

RESEARCH CONSENT FORM - NONPHOBIC

RESEARCH CONSENT FORM - NONPHOBIC

Name:_____ Date:_____

Dear Participant:

I am conducting a study about anxiety and panic attacks, in connection with my work as a therapist and my doctoral degree requirements at Michigan State University. Individuals who have completed the Birmingham Adult Education Lifestyle Course, under the direction of Dr. Doris Ball, are participants in a study to better understand anxiety and panic attacks. I am asking your cooperation in this study by completing the enclosed personality inventories, which are used regularly in counseling, and returning them in the enclosed, self-addressed and stamped envelope. The inventories are usually completed in 20-30 minutes. The data will be treated as completely confidential material, in accordance with standards set by the American Psychological Association. Following completion of the study, the data will remain completely confidential.

I understand that I am free not to participate at all or to discontinue my participation in this study at any time without penalty. I understand that, at my request, I can receive additional explanation of the study after my participation is completed, and that I may obtain results of the study upon my request. If I have any questions or wish to discuss my feelings after participation, I may contact the project director at the address and phone number below. I hereby agree to participate in the research project being conducted by Timothy L. Sams, M.S. (phone: 213-438-8646; address: 85 Ximeno, Long Beach, CA, 90803), under the direction of William C. Hinds, Ed.D., Michigan State University.

Signed: _____ Date: _____

APPENDIX F

RESEARCH CONSENT FORM - PHOBIC

RESEARCH CONSENT FORM - PHOBIC

Name:_____ Date:_____

Dear Participant:

I am conducting a study about agoraphobia and panic attacks, in connection with my work as a therapist and my doctoral degree requirements at Michigan State University. Individuals who have completed the 16 week TERRAP program from 1983 to the present, under the direction of Dr. Doris Ball, are participants in a study to better understand agoraphobia following treatment. I am asking your cooperation in this study by completing several brief personality inventories, which are used regularly in counseling. Ater signing and returning the consent form below, you will be mailed the inventories and requested to return them, upon completion, in an enclosed, self-addressed and stamped envelope. The inventories are usually completed in 20-30 minutes. The data will be treated as completely confidential material, in accordance with standards set by the American Psychological Association. Following completion of the study, the data will remain completely confidential.

I understand that I am free not to participate at all or to discontinue my participation in this study at any time without penalty. I understand that, at my request, I can receive additional explanation of the study after my participation is completed, and that I may obtain results of the study upon my request. If I have any questions or wish to discuss my feelings after participation, I may contact the project director at the address and phone number below. I hereby agree to participate in the research project being conducted by Timothy L. Sams, M.S. (phone: 213-438-8646; address: 85 Ximeno, Long Beach, CA, 48813), under the direction of William C. Hinds, Ed.D., Michigan State University.

Signed:_____ Date:_____

APPENDIX G

THERAPIST LETTER

THERAPIST LETTER

Doris V. Ball, Ph.D.
South Woodward Clinic
Birmingham, MI 49075
January 5, 1989

Dear TERRAP Member,

I am writing to all former TERRAP clients on behalf of Timothy Sams, M.S., Limited Licensed Psychologist. He specializes in the field of agoraphobia and is currently completing his doctoral dissertation on agoraphobia and panic disorder following treatment. We are requesting your brief participation in this original study.

Please be assured that your status as a TERRAP participant remains confidential. Only if you sign and return the enclosed research consent form will Mr. Sams be aware of your name and address. Your participation in this study is completely voluntary. You are free not to participate at all in this project. The conditions for participation in this study are outlined on the consent form.

Sincerely,

Doris V. Ball, Ph.D.
Fully Licensed
Psychologist

APPENDIX H

ACKNOWLEDGEMENT LETTER

ACKNOWLEDGEMENT LETTER

Dear Participants,

I appreciate your willingness to participate in this study of agoraphobia and panic attacks following treatment. Enclosed please find five personality inventories, which you are requested to complete and return to me in the enclosed, self-addressed and stamped envelope. Please answer these inventories in the order in which they are presented. They typically require a total of about 20 minutes to complete. Please note that the data will be treated as completely confidential and will remain confidential after the study.

Sincerely,

Timothy L. Sams, M.S.

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