THE ROLE OF COGNITIONS IN PANIC:

A SELF-EFFICACY ANALYSIS

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(ABSTRACT)

Panic attacks are, by definition, frightening experiences. Recent research has attempted to develop efficacious treatments for individuals suffering from these sudden episodes of fear. The current investigation sought to examine the efficacy of a new cognitive-behavioral treatment, Guided Imaginal Coping. This treatment aims to improve the coping repertoires of panic sufferers. Pilot results with this treatment suggest that it is an effective treatment. The current study sought to extend these findings by evaluating the process of change over time. The second purpose of the current investigation was to examine whether the concept of self-efficacy could help explain the process of change over time. Nineteen subjects diagnosed with Panic Disorder or Agoraphobia with panic attacks were assigned to one of two treatment groups: Guided Imaginal Coping or Panic Education, a nondirective treatment. Subjects in each group received six individual and four group therapy sessions. In addition to treatment, each subject met with a trained evaluator before treatment began, the five weeks of treatment, posttreatment, and one and two month follow-up periods. During these evaluations, subjects participated in a taped anxiety induction procedure and completed questionnaires concerning their degree of self-efficacy, level of panic symptoms, catastrophic thoughts, avoidance, and frequency of coping. Results indicated that subjects in both treatment groups had significant reductions in symptoms, catastrophic thoughts, and number of attacks. Guided Imaginal Coping subjects demonstrated reduction in avoidance and showed continued improvement in symptom reduction

during the follow-up period. Both groups showed improved coping abilities. Self-efficacy and rated coping were significantly related at all evaluation periods. Cross-lagged panel analyses revealed that self-efficacy appeared to lead to changes in catastrophic thoughts whereas self-efficacy and symptom level appeared mutually causal. Self-efficacy and avoidance did not appear consistently related nor did self-efficacy and coping frequency. Rated coping effectiveness appeared to improve in both groups although coping frequency did not. Results are discussed related to treatment outcome and to panic not being as refractory a problem as presumed. Implications are discussed in terms of models of panic and the significance of self-efficacy and catastrophic cognitions to these models and to the amelioration of panic.

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INTRODUCTION

Overview

It is becoming increasingly apparent that panic attacks represent a common phenomena among patients with anxiety disorders as well as among individuals without diagnosable conditions. As such, research exploring the efficacious treatment of panic is becoming important. The current investigation examined a new cognitive-behavioral treatment, Guided Imaginal Coping. This treatment seeks to systematically alter the coping repertoire of panic sufferers. This treatment has been evaluated in pilot studies and the current investigation was designed to explore the efficacy of the treatment over time. A second question of the present investigation was whether the concept of self-efficacy can account for the process of change over time in treatment. The characteristics of panic will first be discussed. This will be followed by a brief discussion of various etiological components of panic with particular attention to the role of cognitions in the etiology and possible treatment of panic. The state of the art of cognitive-behavioral treatment of panic will then be examined followed by the rationale of applying the concept of self-efficacy in examining the process of change in the treatment of panic.

Background

Panic attacks are sudden episodes of intense fear or apprehension. The Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition-Revised (DSM-III-R) (American Psychiatric Association, 1987) lists the following common symptoms of an attack: "shortness

of breath, dizziness, palpitations or tachycardia, trembling or shaking, sweating, choking, nausea, depersonalization, paresthesias, hot or cold flashes, chest pain, and fear of dying, going crazy, or doing something uncontrolled" (p. 238). An individual must experience at least four of these symptoms during an attack. At some point in the development of the disorder, these symptoms must occur suddenly and intensify within ten minutes. In terms of quantity of attacks necessary for diagnosing the disorder, four attacks must occur within a four week period or one attack has occurred followed by one month's fear of having another attack.

These criteria represent a change from previous diagnostic requirements (DSM-III; American Psychiatric Association, 1980). Previously, to receive a diagnosis of Panic Disorder, individuals must have experienced three attacks in three weeks. Anticipatory anxiety was not included in diagnostic decisions. Even with prior criteria, research suggested that panic attacks were quite prevalent. The NIMH Epidemiological Survey found a six month incidence of Panic Disorder in approximately 1% of the population (Meyers, Weissman, Tischler, Holzer, Leaf, Orvaschel, Anthony, Boyd, Burk, Kramer, & Stoltsman, 1984) and a lifetime incidence of 1.5% (Robins, Helzer, Weissman, Orvaschel, Gruenberg, Burke, & Regier, 1984). Additionally, approximately one-third of the adult population experiences one or more panic attacks a year (Norton, Harrison, Hauch, & Rhodes, 1985). These individuals, who likely failed to previously meet diagnostic criteria, may now be diagnosable as Panic Disorder under the revised criteria. Further, 80% of patients diagnosed with any anxiety disorder experience panic attacks (Barlow, Vermilyea, Blanchard, Vermilyea, Di Nardo, & Cerney, 1985). Even with this prevalence, the actual number of Panic Disorder sufferers may still be underestimated given that the symptoms of panic mimic many physical ailments such as hypoglycemia, hyperthyroidism, caffeinism, and

drug intoxication and withdrawal (Grant, Katon, & Beitman, 1983).

Etiological Model for Panic Attacks

Several hypotheses have been advanced to explain the etiology of panic. These can be grouped as genetic (e.g. Harris, Noyes, Crowe, & Chaudry, 1983; Noyes, Crowe, Harris, Hamra, McChesney, & Chaudhry, 1986), biological (e.g. Carr & Sheehan, 1984), and psychological (e.g. Mandler, 1975). Each of these models has received some empirical support. Yet, in the examination of effective treatments and the process of change during treatment, a more complete understanding of the problem of panic and its amelioration requires examination of aspects of each of these models.

Although the biological and psychological hypotheses seem theoretically and conceptually distinct, a rapproachment is necessary to account for existing data on panic. An example of a conceptual rapproachment (Clum & Pickett, 1984) is presented in Appendix A. Block A represents predisposing factors. Several biological and genetic factors have been linked to panic attacks. For example, Noyes et al. (1986) report that the morbidity risk for panic disorder among relatives of patients with panic disorder is 17.3%. This is significantly greater than the 2.3% risk reported for relatives of control probands (Crowe, Noyes, & Pauls, 1983). Harris and colleagues (1983) reported a morbidity risk for Agoraphobia in relatives of probands as 8.6%, Panic Disorder as 7.7%, and all Anxiety Disorders as 31.7%. This compares with 4.2%, 4.2%, and 14.8% respectively for normal controls. In the only twin study aimed at Panic Disorder, Torgersen (1983) examined proband wise concordance rates for Agoraphobia, Panic Disorder, Obsessive Compulsive Disorder, and Generalized Anxiety Disorder. His results show higher concordance for MZ twins than for DZ twins in all categories except Generalized Anxiety

where DZ twins had higher concordance rates. Overall MZ concordance was 34% while DZ concordance was 17%. Importantly, however, no MZ co-twin had the same anxiety disorder as the proband.

Several problems are apparent in the genetic literature. First, the level of disconcordance is quite high. If a strong genetic component were present, the concordance rates would presumably be higher. Further, Torgersen's (1983) results are not diagnosis specific. That is co-twins were at higher risk for having some anxiety disorder, but not the same disorder as the index case. Together, these findings suggest that a specific transmission of a particular disorder is unlikely. More likely, individuals may inherit a predisposition to develop some type of anxiety disorder. Unfortunately, there are not yet adoption studies to address the relative contribution of environmental experiences and genetic predisposition in the etiology of Panic Disorder.

Biological links to panic include research on adrenaline (Breggin, 1964), sodium lactate infusions (Sheehan, Carr, Fishman, Walsh, & Peltier-Saxe, 1985), and drug specificity in the treatment of panic (Shehi & Patterson, 1984). Breggin (1964) reported that previously anxious individuals, when injected with adrenaline responded with physiological changes associated with anxiety attacks. Normal subjects had smaller physiological responses which were not termed attacks. Pitts and McClure (1967) proposed an infusion model of panic based upon research that patients with anxiety attacks had excessive sensitivity to lactate. The anxiety subjects responded to lactate infusions with attacks while normals did not. Sheehan et al. (1985) report that approximately 80% of panic attack patients have an attack when infused with sodium lactate. Less than 20% of normal subjects have an attack. Further, the attacks

which normal subjects experience are less severe and more transient than the panic attack population's.

An additional biological link concerns drug specificity in the treatment of anxiety. Based on this specificity, Klein (1981) proposed two anxiety systems, anticipatory anxiety and panic. For example, tricyclic antidepressants and monoamine oxidase inhibitors relieve panic symptoms but not anticipatory anxiety (Shehi & Patterson, 1984). However, a number of drug treatment studies do not support the specificity hypothesis. For example, a new benzodiazepine, alprozolam, has been demonstrated to effectively treat both panic and anticipatory anxiety (Chouinard, Annable, Fontaine, & Solyom, 1982).

Although some biological links can be specified, they cannot completely account for the etiology of panic. Even at the level of predisposing factors, psychological factors are important. For example, the course of panic disorder waxes and wanes in terms of frequency and occurrence. Additionally, idiosyncratic stressors can often be identified for panic attack sufferers. Hibbert (1984a) reported that a large majority of his sample of panic patients (88%) had experienced significant idiosyncratic stressors within 12 months preceding the onset of their panic attacks. Ottaviani and Beck (1987) examined 30 Panic Disorder patients and determined in each case that the initial panic episode was preceded by some type of stress - either psychological or physical. Further, panic attack patients, as compared to matched normal controls, experienced significantly more stressful life events such as illness or death in their family in the 2 months preceding their first attack (Faravelli, 1985).

Block B refers to triggering events or cognitions. These develop generally out of the individual's awareness and trigger the first acute panic attack (Block C). The individual does not

have prior experiences to "make sense out of" the first attack. These events and cognition are proposed as a way to accomplish this. For example, a woman from a strongly critical family felt pressure from her husband to find a job. She found a very high pressured job which made her feel "under stress." However, she felt she could not leave the position because of her husband's insistance that she work. The situation left her feeling trapped between undesirable alternatives: a highly stressful job versus pressure from her husband to maintain it. After a particularly stressful week in which she was generally unable to sleep, she suffered her first panic attack Sunday evening before she was scheduled to return to work. The attack was explained as the culminating effect of being trapped in a highly stressful situation.

Because the triggering and predisposing factors are generally out of the individual's awareness, the first attack is not interpreted as being related to anxiety (Clum & Pickett, 1984). Rather, it is often viewed by the individual as evidence that they are going crazy, having a heart attack, or dying. Medical specialists are often sought at this level. When subsequent minor anxiety symptoms appear (Block D), the conceptualization proposes that they are sufficient to elicit the precipitating cognitions (Block E), further exacerbating the symptoms. This theoretically results in a vicious cycle where most panic attack sufferers become hypervigilant in looking for the symptoms, likely out of fear of another attack. This is what Goldstein and Chambless (1978) have termed "fear of fear." Individuals become sensitized to the physiological arousal which occurs during attacks. They then become preoccupied with their focus on these sensations, seeing them as a warning signal for another attack (Beck & Emery, 1985). This "fear of fear" can also be seen in the somatization behavior of panic patients. King, Margraf, Ehlers, and Maddock (1986) compared panic subjects and normal controls. Both

groups were without physical health difficulties. Subjects completed inventories concerning their anxiety and degree of somatization. They also provided relevant background information such as length of anxiety problems. The results indicated that panic sufferers present a pattern of significantly greater somatization including greater focus on "nonspecific neurotic symptoms," greater gastrointestinal awareness, and greater cardiovascular awareness. Importantly, this somatization was not related to level of state anxiety nor to length of panic problems. Thus, it cannot be viewed as merely a function of conditioning after lengthly battles with panic. Rather, it appears likely to coexist suggesting a cognitive element of Panic Disorder.

The Relationship between Cognitions and Panic

Clearly, cognitions are critical to the etiological model presented above. A variety of theories have been proposed concerning the relationship between cognitions and physiological arousal. For example, the James-Lange theory (1922) stated that bodily changes followed perceptions. This bodily change, per se, is emotion. An alternate perspective maintains that emotions arise only as a function of cognitive appraisal (Arnold, 1960; Leventhal, 1980). Schachter and Singer (1962) provided a moderated position. This theory posits that emotion is the result of arousal and subsequent cognitive interpretation. Importantly, the cognitive interpretation determined the final experiential nature of the previously ambiguous physiological arousal. Each of these theories has received mixed empirical support. Cognitions, in some capacity, are critical to each theory. For example, Lang (1979) provided experimental evidence that cognitions can elicit emotion, specifically anxiety.

Lang's (1979) theory of emotional imagery is based upon psychophysiology, information processing, and behavior therapy. He maintains that symbolic stimuli or images can evoke

affective reactions. In a series of experiments, Lang, Kozak, Miller, Levin, and McLean (1980) demonstrated that subjects trained to focus on physiological cues in imaginal scenes had significantly greater physiological responding as compared to both a group trained to focus upon stimulus cues and a no instruction control group. The imaged scenes were a neutral, an action, and a feared scene. This research was extended to examine differences among subjects with different fears, social phobias and snake phobias (Lang, Levin, Miller, & Kozak, 1983). Again, physiological-response trained subjects had greater physiological arousal than stimulus-cue or control groups.

Two issues emerge from Lang's seminal work. First, cognitions and physiological arousal are intricately related. Furthermore, at least in artificial situations, specific cognitions can result in varied physiological responding. This does not provide information as to whether this would occur in a more naturalistic setting where individuals were not prompted to monitor certain cues. The second issue suggested by Lang's work concerns the relationship between a focus on somatic cues and anxiety. Subjects trained to focus upon these cues demonstrated more evidence of anxiety. Again, while subjects were trained in this focus, a somatic focus may be a relevant issue in anxiety subjects. This possibility will be explored with regard to panic in more detail below.

More specific to anxiety, the significance of cognitions in anxiety disorders has been discussed (e.g. Beck & Emery, 1985; Williams & Rappoport, 1983). Beck, Laude, and Bohnert (1974) found that anxiety patients had automatic thoughts related to themes of danger such as loss of bodily control and dying. May (1977) reported that self-regulated phobic thoughts were associated with increased physiological responding and fear. Related to panic, Hibbert (1984a)

identified characteristic ideations for a group of panic patients. These tended to center on personal harm and danger such as having a heart attack or dying. Contrary to the temporal sequencing in Lang's (1979) theory, Hibbert found subjects reported their arousal first. They then made a cognitive attribution concerning reasons for the arousal. Hibbert's data explains panic attacks as somatic symptoms followed by some kind of cognitive reaction. Hibbert suggests that panic subjects systematically misconstrue somatic or physiological symptoms as more dangerous than they really are. The subjects tend to , in the term of Beck et al. (1974) "catastrophize" and subsequently panic. Additionally, Last, O'Brien, and Barlow (1985), based on a group of agoraphobic subjects with panic attacks, reported that negative thoughts were correlated with anxiety. There was some evidence (although not consistently significant) to support an inverse relationship between positive thoughts and anxiety. The negative thoughts were generally catastrophic while the positive ones were related to coping. In a series of analogue studies, Wade, Malloy, and Proctor (1977) demonstrated that the content of subjects' imagery is related to their avoidance and fear. In a fearful situation, high fearful subjects reported more aversive imagery than low fearful subjects. Further, aversive imagery was positively associated with level of fear and avoidance. Imagery content was associated with fear and avoidance when subjects imagined themselves in the situations as well as when they were actually confronted with the fearful stimuli (snakes were used in this series of experiments). Finally, Ottaviani and Beck (1987) reported that panic was associated with thoughts and images related to physical and mental catastrophe. Further, the authors questioned all patients regarding their thoughts during attacks. All subjects showed misattribution of somatic or psychological experiences which triggered their attacks. Like other similar research, this

study is retrospective and thus subject to bias or distortion. However, these studies do suggest that cognitions have a meaningful role of some sort in panic attacks.

Although the cumulative results of these studies support the notion that "negative" cognitions and anxiety reactions are related, the direction of the relationship remains confused. Last et al. (1985) correlated percentages of negative thoughts, positive thoughts, and self-reported anxiety during in vivo exposure. The methodology did not allow a test of causality. Hibbert (1984a) and Ottaviani and Beck (1987) relied completely on self-report of past events. Given the traumatic experience of panic, the subjects' recall of temporal sequencing of thoughts and arousal may be inaccurate. They may attempt to "make sense" of the attack by imposing a logical structure upon it after the fact.

A recent research strategy designed to address these methodological problems involves the induction of panic attacks. This strategy allows more direct access to an individuals' symptoms and thoughts before, during, and after an attack. Thus, it helps minimize the difficulties of relying on accurate recollections by subjects.

The first method described in the panic induction literature was sodium lactate infusion. Early research by Jones and Mellersh (1946) identified the connection between elevated lactic acid and anxiety. During physical exercise, normal subjects had the expected rise in lactate. However, Jones and Mellersh reported that anxiety subjects had excessive rises and also increased sensitivity to the elevated lactate level. Sodium lactate infusions are currently used for three main purposes: diagnosis, treatment, and research (Sheehan et al., 1985). Diagnostically, they are used to confirm the diagnosis of panic disorder. The infusions have also been used as an exposure based treatment for panic attacks (e.g. Bonn, Harrison, & Fees, 1973).

The rationale behind such a treatment approach involves the notion that individuals with panic attacks develop an anticipatory anxiety of having another attack, what Goldstein and Chambless (1978) term "fear of fear." In a sense then, this anticipatory anxiety is viewed as a phobic response to the panic itself. Thus, individuals are exposed to that which they fear.

Several issues remain unclear from the infusion data. The primary question of why people experience panic symptoms when infused has not been answered. Current research has ruled out hypotheses such as calcium depletion (Liebowitz, Gorman, Fyer, Dillon, Levitt, & Klein, 1986). Additionally, these authors report that infusions of sodium lactate produce somatically similar experiences to panic attacks. However, these are not accompanied by the cognitive components. Subjects do not report thoughts related to death, unreality, difficulty concentrating and so on. A further complication in interpreting the data comes from a psychological treatment study (Guttmacher & Nelles, 1984). A subject was treated with in vivo desensitization for agoraphobia with panic attacks. At posttreatment, the subject did not respond to lactate infusions even though she had at pretreatment. The authors provide no explanation for this finding. Several hypotheses could be generated for the lack of posttreatment panic upon infusion. First, lactate infusions may indeed be a form of exposure therapy. The subject, while exposed in vivo, was also exposed to her somatic symptoms. This hypothesis is weakened somewhat by relying on one trial of exposure to the feared somatic stimuli to result in a "cure." Another hypothesis is that the subject was not fearful of the second induction. Although similar to exposure, the rationale of this hypothesis is that she knew what to expect and was also aware of why she was feeling the way the infusion made her feel. In other words, she was able to attribute the somatic responses to the procedure rather than to herself. Unfortunately, the

authors did not address the subject's cognitions to examine her attributions or expectations of the infusions.

In a comprehensive review, Margraf, Ehlers, and Roth (1986) offer four major problems with lactate and other physiological induction studies. First, the assessment of panic is based soley on self-report. Interestingly, recent research suggests that subjects are not very accurate about physiological events (Taylor, Sheikh, Agras, Toth, Margraf, Ehlers, Maddock, & Gossard, 1986). Self-reported panic was accompanied by high heart rates only 58% of the time. Further, only four of 14 heart rate episodes identified by monitor as signifying an attack were accompanied by subjective attacks. Although only heart rate was assessed, these combined results argue that physiological arousal may not be the sole determinant of whether or not an attack occurs. Margraf and his colleagues specify that the research is only single blind. Further, there is inadequate criteria for defining panic. Finally, and perhaps most importantly, patient expectations are not controlled. In fact, some patients panic from infusion of saline solution. In comparing normals and panic subjects, panic subjects are often told that they might experience a panic attack while normals are told that they may experience an attack similar to one experienced when speaking in public (Clark, 1986). These different instructions may result in different expectations potentially influencing results. Related to this final point, Ehlers and colleagues (1986) demonstrated that the actual increase in subjective anxiety and in physiological arousal from lactate is approximately equal for panic and control subjects. The difference is dependent on the level of baseline arousal. When this is taken into account, there is little difference in degree of arousal.

Another technique for panic induction is the administration of caffeine. Recent research

suggests that panic patients have increased sensitivity to the effects of caffeine (Boulenger, Uhde, Wolff, & Post, 1984; Charney, Heninger, & Jatlow, 1985). This increased sensitivity includes self-reported anxiety, nervousness, nausea, and palpitations. Charney et al. (1985) report that in panic, but not normal, subjects these symptoms are correlated with caffeine levels. In fact, 71% of panic patients reported that experiences from caffeine were similar to those during panic attacks. A variety of explanations have been provided for caffeine's effect on subjects with panic attacks. Berkowitz, Tarver, and Spector (1970) hypothesized that caffeine increases catecholamine activity. Charney et al. (1985) report that caffeine produces greater anxiety and higher blood pressures in panic subjects. They propose an abnormality in the neuronal system to account for these findings. This proposal remains speculative.

An additional technique for panic induction is hyperventilation. The most commonly occurring symptoms of a panic attack include dizziness, blurred vision, numbness, palpitations, tingling sensations, tachychardia, nausea, and breathlessness (Clark, Salkovskis, & Chalkley, 1985). These are also the most common symptoms occurring during hyperventilation (Clark et al., 1985). Hyperventilation is caused by increased respiratory ventilation. This activity leads to a reduction of carbon dioxide in the lungs and subsequently in the partial pressure of carbon dioxide in the blood. These bodily changes produce the unpleasant symptoms and bodily sensations (Van Den Hout & Griez, 1984). Because of the similarity in symptomatology between hyperventilation and panic attacks, many writers have suggested that hyperventilation may produce panic attacks (Clark & Hemsley, 1982; Hibbert, 1984b; Lum, 1976).

A variety of researchers have demonstrated that a standard hyperventilation provocation can produce panic attack symptoms (Bonn, Readhead, & Timmons, 1984; Garssen, Van Veendaal, &

Bloemink, 1983; Van den Hout & Griez, 1984). Garssen et al. (1983) reported that 61% of their agoraphobic subjects experienced symptoms similar to their usual panic attacks during voluntary hyperventilation. Gorman, Askanazi, Liebowitz, Fyer, Stein, Kinney, & Klein (1984) compared sodium lactate infusions, carbon dioxide inhalation, and hyperventilation in the production of panic-like symptoms. They reported that 8 of their 12 subjects experienced panic symptoms with sodium lactate while 7 of the 12 did so with carbon dioxide inhalation. The less rigorous and challenging procedure of hyperventilation produced similar results both in number of subjects experiencing symptoms as well as number of symptoms experienced. Rapee (1986) compared generalized anxiety and panic disordered subjects on their responses to 90 seconds of voluntary hyperventilation. Panic disorder subjects reported greater distress and more panic like symptoms. They also had lower resting partial pressure of carbon dioxide and higher heart rates. More anecdotically, Rapee questioned the subjects about the similarity of the experienced symptoms and their naturally occurring anxiety symptoms. Only 25% of the generalized anxiety subjects indicated that the symptoms were similar to their naturally occurring ones. Over 80% of the panic disorder subjects responded that the symptoms were similar. Importantly, even with a large majority of the panic subjects reporting that the symptoms were similar, none reported actual panic attacks. When querried, the subjects attributed this to knowledge of why the symptoms were occurring and to being in a "safe" environment. Beyond anecdote, research also suggests the role of attributions in the explanation of the panic-inducing nature of this type of procedure. Using normal subjects, Clark and Hemsley (1982) and van den Hout and Griez (1982) demonstrated that even in normal subjects, the extent to which the subjects experience the induction as aversive was determined

by expectation of prior experiences with induced sensations. In his review of these findings, Clark (1986) suggests that panic provoking procedures may not have a direct induction effect. Rather, they may result in panic with a given set of cognitions.

A common finding in the induction literature is that all these procedures can produce panic-like symptoms. However, they rarely produce what subjects experience as a "panic attack." Evidence (Liebowitz et al., 1986; Rapee, 1986) suggests that the subjects attribute their symptoms to the experimental procedure and thus do not panic per se. The lack of subjective panic attacks in these subjects suggests that symptoms are necessary, but not sufficient, for a panic attack to occur. It would appear that cognitions are integrally related to panic attacks. Further, it would appear that panic subjects are somatically preoccupied (King et al., 1986), but inaccurate in their interpretation of physiological arousal (Taylor et al., 1986). The role of the subjects' attributions and associated cognitions requires more formal investigation.

A final technique for panic induction addresses the difficulties observed in the physical provocation literature. This technique involves the induction of anxiety through imagery. The individual is instructed to imagine his or her panic attack as vividly as possible, Cues are provided to increase the level of anxiety. This approach offers several advantages. First, many panic attack sufferers have idiosyncratic situations or fears which are related to their attacks which may not be readily available for an in vivo exposure treatment. Additionally, induced anxiety states allow the client to recall more details of their attacks. Individuals with panic attacks appear to recall global impressions of their attacks rather than specific details. This is likely related to the traumatic and frightening nature of their experience. Previous research indicates that imagery is a viable mechanism for inducing anxiety (Watkins, Clum, Borden, &

Broyles, 1988). Heart rate and subjective anxiety were compared while subjects imagined a relaxing scene, a neutral scene, a stress scene, and a panic scene. Heart rate and subjective anxiety were significantly higher during scenes containing cues relevant to the subject's panic attack. The utilization of an imaginal induction procedure throughout the treatment of panic allows a unique opportunity to explore the relationship of the experience of a panic attack and an individual's ability to cope with the various components of an attack. This will be further explored after an overview of the current state of cognitive-behavioral treatment for panic attacks.

Cognitive-Behavioral Treatment of Panic

Cognitive-behavioral techniques have been advanced as treatments for anxiety disorders. There are relatively few controlled clinical studies examining cognitive-behavioral treatments of panic. However, the results are fairly encouraging.

Woodward and Jones (1980) compared cognitive restructuring and relaxation training for "clinical anxiety patients." Their population was not defined further. The results indicate that the combination of the two approaches is superior to either alone. However, the study relied on small numbers of subjects and relatively weak dependent measures (such as estimated amount of time spent thinking about problems). Ramm, Marks, Yuksel, and Stern (1981), whose subject population mainly consisted of panic attack patients, reported that positive self-statement training was more effective than negative self-statement training in terms of patient ratings of panic at posttest. More objective measures of improvement were not included in the study. The authors comment that the results are not as impressive as analogue research on self-statement training (e.g. Meichenbaum, 1977).

Barlow and his colleagues (1984) treated panic disorder and generalized anxiety subjects with somatic (EMG biofeedback and relaxation) and cognitive techniques. Cognitive strategies included coping self-statements and evaluation of anxiety-provoking thoughts. Subjects rehearsed the strategies in session and between sessions. Both groups of subjects improved compared to wait list controls suggesting the efficacy of psychological treatments. However, the treatments were not dismantled to examine their relative efficacy.

Waddell, Barlow, and O'Brien (1984) employed a multiple baseline design with three panic disorder individuals. The subjects received cognitive therapy consisting of coping self-statement training and distraction techniques. They also received progressive muscle relaxation training (Bernstein & Borkovec, 1973). Relaxation was included only in a combined treatment package and was never administered alone. The results indicate a decrease in the number and duration of what the authors term "intense anxiety." This was defined as a subjective rating of anxiety greater than 4 on a 1 to 8 scale of anxiety severity. Again, the results point to the efficacy of psychological treatment. Due to a significant amount of missing data and the restricted number of subjects, the relative contributions of the treatments could not be ascertained.

Emmelkamp and Mersch (1982) evaluated three treatments with agoraphobic subjects. They compared exposure in vivo, cognitive restructuring, and a combination of the two treatments. The researchers used a variety of dependent measures including an in vivo measure of time spent walking from the hospital where the research was conducted to the center of town, self and observer rated avoidance behavior, depression, assertiveness, and locus of control. At posttreatment, the exposure and combined treatment groups were significantly improved

compared to the cognitive restructuring group on the in vivo and avoidance measures. The cognitive restructuring group was superior on the other three self-report measures. However, at a one month follow-up, the three treatments were equally effective on the behavioral and avoidance measures. That is, the cognitive restucturing group "caught up" with the improvements of the other groups. The reasons for the change from posttest to follow-up were not identified. While the study supports the efficacy of both cognitive and behavioral treatments for agoraphobics, the lack of specific cognitive assessment makes the results less clear.

Ascher (1981) compared exposure and a cognitive intervention in the treatment of travel restrictions of agoraphobics. He employed paradoxical intention and the treatment of choice for agoraphobia, in vivo exposure. One group received in vivo exposure followed by paradoxical intention instruction. The other group was instructed in paradoxical intention immediately after baseline. The dependent measure was a compiled score based on two target approach tasks. The tasks were individualized for each subject, but the scoring procedure allowed a standard for comparison. The tasks were divided into ten steps. The subject received a point for each step completed. The results clearly indicate that the paradoxical group was superior to the exposure group after the first phase of treatment in which the groups had received only one form of the treatment. The exposure group was then given paradoxical treatment which resulted in significant gains in their approach behavior.

Additional research has pointed to the efficacy of paradoxical intention in the treatment of agoraphobia (Mavissakalian, Michelson, Greenwald, Kornblith, & Greenwald, 1983). This study included a global assessment of cognitions which indicated that after treatment the subjects reported fewer "self-defeating" cognitions. However, they did not report increased coping

statements. Michelson, Mavissikalian, and Marchione (1985) compared paradoxical intention, graduated exposure, and muscle relaxation. All subjects improved on all behavioral approach and self-reported anxiety and panic measures. In fact, 60% of the subjects reported that they were no longer having spontaneous panic attacks following treatment. In a recent study, (Kleiner, Marshall, & Spevack, 1987), agoraphobic patients with panic attacks received either in vivo exposure or in vivo exposure plus problem solving. Both groups improved significantly after treatment. However, improvement was only maintained at follow-up with the problem solving in addition to exposure.

Last, Barlow, and O'Brien (1984) attempted to assess cognitive changes for agoraphobic subjects during cognitive and cognitive-behavioral treatments. Using a multiple baseline design, six subjects received in vivo exposure and three of these additionally received a cognitive treatment component. In the cognitive component, the subjects were taught to replace maladaptive cognitions with coping verbalizations. They were to then apply the strategy in vivo. Only two subjects were improved at post treatment. Cognitions associated with improvement could not be evaluated. Although the results do not consistently demonstrate cognitive changes in either treatment, a trend emerged such that cognitive change was associated with improvement. However, several problems were noted by the authors. First, cognitive changes occurred during baseline periods for most subjects. The cognitions varied considerably during treatment. Finally, several of the subjects had poor clinical outcomes. Given the small sample size, conclusions cannot be drawn from the results.

A second study attempted to examine the process of cognitive change in treatment. Williams and Rappoport (1983) attempted to generalize coping strategies by teaching them in vivo. The

subjects were twenty severe driving phobics. Half of the subjects received an exposure based treatment of gradually increasing their driving distance. In addition, the others were taught coping strategies to deal with their fears. The authors state that this led to teaching the strategy in the situation. However, the coping strategies were taught in between driving tasks thus serving to remove them from the actual situation. While their rationale is appealing, their manipulation does not allow an actual test of the hypothesis that teaching strategies in vivo will enhance treatment efficacy. The results indicated that there were no differences between the two groups on a behavioral driving task following treatment. The cognitive subjects demonstrated more "coping" thoughts after treatment. Based on these findings, the authors conclude that cognitive coping does not add to the efficacy of treatment. Several interpretation difficulties make this conclusion tenuous. The authors define coping as specifically excluding the following: efforts to relax; discussing what was happening bodily; and nonspecific distractions. This leaves a fairly limited category for coping. The results reflect this in that less than 50% of all thoughts can be accounted for in terms of "coping" or "negative" - the only two categories reported. The results are not surprising considering less than 20% of the coded thoughts were "coping." Further, the procedure does not appear to allow conclusions to be drawn about the facilitation of coping in vivo given that the coping strategies were not taught as such.

A recently developed treatment, Guided Imaginal Coping (GIC: Clum, 1986), teaches patients with panic attacks to cope effectively with their attacks and to thereby reduce the frequency and severity of attacks. Briefly, the treatment involves having patients re-experience particular aspects of their attacks within the context of imagery. While reexperiencing these symptoms and thoughts, they are instructed in adaptive coping strategies.

This technique allows the client to practice new coping strategies while he or she experiences anxiety. This provides several potential benefits. First, the individual can test the strategy to determine its efficacy in managing symptoms. This allows an immediate experience of coping. Should the strategy not provide relief, assistance can be provided in the form of additional instruction in using the coping strategy, therapist reinforcement of the process of utilizing the technique, and finally supplemental or additional strategies.

Guided Imaginal Coping (N = 9) has been compared to flooding (N = 5) and to a waiting list no-treatment condition (N = 9) in the treatment of individuals suffering from panic attacks. Although based on few subjects, the results reveal consistent trends in favor of the coping treatment. Pilot results demonstrate a relationship between coping effectiveness and panic thoughts and symptoms (Borden & Hayes, 1986). The effectiveness of coping was analyzed with a 3 x 2 MANOVA with group and time as factors. A marginal effect emerged for time [$F(2,15) = 3.13$, $p < .08$] such that all three groups reported increased effectiveness of coping in terms of symptom reduction at posttreatment evaluation. Although nonsignificant, the GIC group reported more reduction than the other two groups. With a larger sample, this difference may become significant. There is, however, a major confound in the utilization of coping in response to treatment. The effectiveness of coping may diminish, not because of its ineffectiveness in managing symptoms, but rather because the symptoms decrease in frequency and severity. Total self-reported coping frequency, symptom reduction, and thought elimination from coping were correlated with total number of thoughts and symptoms reported by panic attack sufferers. These correlations are summarized below. The subjects indicated that the more coping strategies used, the more symptom reduction achieved. This was consistent at pre ($r = .47$,

$p < .01$), post ($r = .82$, $p < .001$), and follow-up ($r = .65$, $p < .01$). Additionally, the greater the frequency of coping strategies used, the more thought elimination. This was also consistent over time: pre ($r = .51$, $p < .005$), post ($r = .79$, $p < .001$), and follow-up ($r = .54$, $p < .01$). Thought elimination from the use of coping strategies was negatively related to the number of panic thoughts during an attack: pre ($r = -.34$, $p < .08$), post ($r = -.44$, $p < .05$), and follow-up ($r = -.41$, $p < .05$). Total number of coping strategies was negatively related (although not significantly) to total number of symptoms ($r = -.22$, $p > .10$). Symptom reduction was related to fewer panic thoughts during an attack ($r = -.43$, $p < .05$). This was maintained at follow-up ($r = -.38$, $p < .07$). Although the nature of correlations did not allow a test of causality, this relationship between decreased thoughts and symptoms provides indirect support for the above conceptualization which proposes a link between catastrophizing cognitions and symptom escalation.

Panic attacks and related thoughts and symptoms were evaluated more directly in a subsequent study (Borden, Clum, & Broyles, 1987). Subjects treated with GIC reported greater improvement in number of panic attacks following treatment, panic symptom reduction, and catastrophic thoughts reduction. Subjects were compared to determine differences in the number of panic attacks following treatment. Improvement in the number of attacks was defined as no panic attacks or at least a fifty percent reduction in the number of attacks measured in the last month of treatment or in the previous month measured at a point four months posttreatment. From pretreatment to posttreatment, 63% of the GIC subjects, 80% of the flooding subjects, and 45% of the wait list control subjects report improvement. At follow-up, where the same criteria is used, 100% of the GIC subjects, 40% of the flooding subjects, and

78% of the wait list group report improvement.

A 3 x 2 MANOVA was computed to examine total symptom reduction. Group and time were independent variables. Subjects are asked to indicate on a self-report questionnaire which symptoms they experience and to rate them on a 1 to 5 scale where the ratings reflect how long the symptoms last. Time was chosen as an objective criteria of severity. The results indicated that all groups reported decreased symptoms over time [$F(2, 20) = 9.73, p < .01$]. In addition, a marginal group by time interaction emerged [$F(2, 20) = 2.70, p < .09$]. Examination of the change scores for each group revealed that the control group decreases are nonsignificant at all time periods. The flooding group's decrease from pretreatment to posttreatment was marginally significant [$F(1, 4) = 5.23, p < .08$]. The GIC group demonstrated the most change. Their drop from pre-treatment to post-treatment was highly significant [$F(1, 3) = 56.34, p < .01$]. Although nonsignificant, these subjects continued to report decreased symptoms at follow-up.

Another 3 x 2 MANOVA was computed with group and time as factors on the total number of catastrophic thoughts reported during a panic attack. Again, this is based on self-report for the past month. The same pattern of results emerged with a significant effect of time [$F(2, 18) = 6.15, p < .05$] and a significant group by time interaction [$F(2, 18) = 5.75, p < .01$]. Again, the GIC group emerged superior. Their decrease in catastrophic thoughts from pretreatment to posttreatment was significant [$F(1,2) = 50.24, p < .05$]. This was maintained at follow-up. The flooding group had no significant changes while the control group showed a significant drop in catastrophic thoughts at follow-up [$F(1,7) = 7.58, p < .05$].

Although limited in the number of subjects to date, the results point to a consistent pattern

of the GIC group reporting the most improvement. GIC appears to be an efficacious treatment for panic attacks. However, the data still leaves a number of questions unanswered such as the causal relationship between thoughts and symptoms. Additionally, data was collected at three time periods which prevents detailed examination of the process of change. The next logical step in evaluating the efficacy of GIC is to compare it over time with an appropriate control group to examine the process of changes in panic attack symptomatology including panic symptoms, thoughts, and related issues of coping and avoidance. In sum, Guided Imaginal Coping has been demonstrated to be an efficacious treatment for panic attacks although the design of prior studies with the treatment has not allowed detailed examination of the process in which improvement occurs.

Cognitions and Panic Summary

Although clearly limited in both the number of studies and the number of studies focusing on panic per se, the majority of the above research points strongly to the efficacy of psychological, and specifically, cognitive treatment of agoraphobia and panic. However, a common problem exists within the research to date. As Last (1984) discusses, most studies fail to include an examination or assessment of specific cognitions before and as a function of treatment. Yet, various lines of inquiry converge on the importance of cognitions in panic. Support can be found in the etiology literature which suggests that biological explanations are insufficient to account for the research findings. Characteristic ideations and automatic thoughts are consistently found within the panic population. A related line of research involves anxiety (Heide & Borkovec, 1984) and panic (Cohen, Barlow, & Blanchard, 1985) induced during states of relaxation. Cohen and his colleagues (1985) discuss the possible involvement of covert or ideational

stimuli in the production of this paradoxical type of anxiety. They comment that these stimuli may be related not only to stress, but to the unfamiliar bodily sensations which relaxation may produce in chronically anxious subjects. Further credence for the role of cognitions is provided in that experimental panic induction elicits symptoms but not subjective attacks. Although the symptoms are diagnosable by the current criteria for panic attacks, the subjects do not report experiencing attacks per se. Anecdotally, they state that they are aware of the reasons for their symptoms and thus do not "cognitively" panic. A final source of support for the involvement of cognitions comes from the panic treatment literature. Cognitive treatments are effective in the treatment of panic. Assessments which include a cognitive component generally reveal cognitive changes associated with positive treatment outcome. However, cognitions in panic have not been systematically evaluated.

Based on available evidence, it appears that cognitions may serve a mediational role in panic attacks as well as effective treatments of panic. Given that symptoms are necessary, but not sufficient, for panic attacks, subject attributions about their symptoms may provide the necessary link which results in an attack. These cognitions appear to involve catastrophic expectations, the underlying message of which is that the individual feels incapable of dealing with their symptoms or the threatening situation. If cognitions are intimately involved in production of panic, then alteration of these cognitions may offer a viable treatment option for panic attack sufferers. Bandura (1977) has proposed one mechanism for evaluating the mediational role of cognitions in therapeutic change. His theory of self-efficacy, its research literature, and applicability to panic attacks will be reviewed.

Bandura's Theory of Self-Efficacy

Based on social learning theory, Bandura (1977) proposed the theory of self-efficacy to examine the mediational role of cognitions. He hypothesized that individuals' expectations of personal efficacy will determine whether they will initiate coping behaviors in stressful and aversive situations. In addition, the estimate of personal efficacy is predicted to determine how much energy the individual will expend and how long he or she will persist in coping efforts. Explicit in this theory is that cognitive processes mediate behavior change. Bandura further assumes that psychological procedures mainly serve to create and strengthen expectations of personal efficacy. Bandura differentiates expectations of efficacy and outcome. Efficacy expectations are defined as the belief that one can execute a given behavior. Outcome expectations refer to the belief that the given behavior will lead to some predicted outcome. He justifies this differentiation by indicating that individuals can believe that an action will produce a specific outcome without believing that they can perform the necessary actions. Bandura claims that people avoid situations which they believe exceed their ability to cope. It is not that they doubt that successful coping would result in decreasing the aversiveness of the situation. Rather, it is that the people believe they will be unable to effect the necessary coping strategies.

Self-efficacy theory does not reflect broad trait conceptions of abilities, rather it is concerned with expectations of specific interactions with one's environment (Goldfried & Robins, 1982). As such, it is a multidimensional construct. Bandura (1977) proposes that self-efficacy varies on three dimensions: Magnitiude, generality, and strength. An estimate of the magnitude of one's personal efficacy refers to the difficulty of the tasks one feels prepared to

handle. For example, on a list of twenty behaviors which are ordered by level of difficulty, the magnitude of efficacy would be the number of behaviors which the person felt capable of completing. The second dimension, generality, refers to a circumscribed versus generalized sense of ability, that is the extent that similar expectancies are held for other situations. The final dimension along which self-efficacy is proposed to vary is strength. Strength of self-efficacy refers to the extent people believe that they can perform behaviors. Bandura refers to this as the level of perseverence in initiating and sustaining behaviors.

Bandura (1977) also proposes that efficacy expectations are derived from several sources including: Performance accomplishments, vicarious learning experiences, verbal persuasion, and emotional arousal. He claims that performance accomplishments are the most important sources of efficacy information due to the necessity of self-observation as well as the development of coping strategies which can be generalized to other situations. Vicarious experiences provide less personal information concerning one's abilities and are thus less dependable sources of information. Bandura states that verbal persuasion may have some impact on efficacy judgments, but that it is nonexperiential and thus provides no direct feedback. Finally, based on research indicating that performance may deteriorate under conditions of high emotional arousal, Bandura states that individuals learn that their capacity to cope may be diminished in highly stressful or aversive situations.

Self-efficacy theory has been subject to a variety of empirical tests. Bandura presented the first tests of the theory in a series of studies on snake phobics. Bandura, Adams, and Beyer (1977) compared three treatment conditions for snake phobics. The conditions were participant modeling, modeling alone, and no treatment. Subjects privately indicated on a list of

18 behaviors the behaviors that they felt capable of executing. They also indicated on a scale from 10 to 100 their degree of certainty in performing these tasks. The anchors for the scale ranged from "great uncertainty" to "complete certainty." The subjects' approach behaviors were assessed. Treatment was administered and post assessments were completed. These again involved estimates of the magnitude and strength of self-efficacy and behavioral approach tasks. Results indicated, as predicted, that participant modeling based upon performance accomplishments produced the greatest improvement in the strength of self-efficacy. Vicarious accomplishments from modeling alone produced efficacy changes greater than the control group's, but not of the magnitude of the participant modeling condition. Bandura, Adams, and Beyer also introduced the procedure of microanalysis in this experiment. They indicate that this procedure produces a more exact measure of the agreement between efficacy expectations and performance than a correlational analysis. The procedure involves examining the congruence between efficacy judgments and performance on each task. The percentage of total agreements is then calculated. Bandura et al. report high congruence between judgments and performance. In this particular study, the congruence was 80 - 90% across all conditions.

Bandura and Adams (1977) examined the efficacy expectations of snake phobics treated with systematic desensitization. They hypothesized that systematic desensitization altered avoidance behavior by altering efficacy expectations. The researchers report that anxiety was "thoroughly extinguished" in all subjects. However, efficacy expectations varied. Analysis after treatment indicated that self-efficacy estimates were highly predictive of behavior change. The congruence between efficacy estimates and performance was approximately 85%. The second experiment presented by the authors investigated the effect of participant modeling on efficacy judgments

and behavior change. Again using snake phobic subjects, Bandura and Adams sought to examine the process of change during treatment. They divided tasks into blocks which became progressively more difficult and threatening. Subjects received treatment for only the activities within a given block. Treatment continued until they were able to complete the tasks in a block. They were then asked to judge their efficacy for the remaining blocks of tasks. A behavioral avoidance test was administered. Treatment was ended when subjects obtained terminal performances. Again, the results indicated that efficacy expectations were superior to previous behavior in predicting subsequent performance. This suggests that behavior change is not merely based upon prior behavior, but rather has a cognitive mediator. For example, all subjects successfully performed intermediate blocks of tasks. However, the past behavior was not a reliable predictor of behavior on subsequent blocks of tasks. Self-efficacy predictions consistently predicted subsequent behavior.

Bandura, Adams, Hardy, and Howells (1980) continued to examine this critical aspect of the theory which posits a cognitive mediator of behavior change. They presented two studies. The first relied on snake phobics treated with cognitive modeling. According to social learning analysis, repeated imaged scenes of individuals confronting threats enhances performance by increasing perceived efficacy in coping with the threats. Subjects were given 29 graduated tasks of increasing threat with a boa constrictor. Efficacy estimates and behavioral avoidance were measured before and after treatment. Treatment involved imaging a variety of models completing the tasks. The models and their level of success were varied such that they initially experienced difficulty and later achieved success through perseverence, thus representing a coping rather than mastery model. The results indicated higher levels of self-efficacy and

approach behavior at posttreatment assessments. Additionally, there was an 81% congruence between efficacy judgments and performance. There was also a significant relationship between increased efficacy and diminished self-reported fear. Because the treatment employed was a nonenactive one, the authors claim support for a cognitive mediation of behavior change. Their rationale is based on the fact that subjects have no behavioral data upon which to estimate their own competencies. The second study provided a treatment package for agoraphobics. They received group sessions and "field mastery" experiences. Efficacy expectations were related to task completions. The congruence between expectations and behavior was 88%. Additionally, the relationship between subjects' anticipatory fears and efficacy expectations was examined. This involved tasks on which subjects felt efficacious, but varied in the strength of their expectation. A low strength of efficacy was associated with high anticipatory anxiety whereas a high strength of efficacy was related to minimal fear. These findings provide further confirmation that behavior is insufficient to account for the results. These subjects performed all of the behaviors, yet with different degrees of arousal and fear.

The research supporting self-efficacy theory is not confined to the work of Bandura with snake phobics. The theory has been extended to a variety of other areas including health behaviors. Three areas in particular have received much attention. They are smoking cessation (e.g. DiClemente, 1981), behavioral medicine (e.g. Bandura, Reese, & Adams, 1982), and pain management (e.g. Manning & Wright, 1983; Vallis & Bucher, 1986). DiClemente (1981) reported that posttreatment self-efficacy judgments predicted 5 month follow-up status. Highly efficacious subjects reported less difficulty maintaining abstinence from smoking. In the behavioral medicine literature, Bandura et al. (1982) reported that high efficacy judgments

were related to ability to handle stressors, lower heart rates, lower blood pressure, and lower secretion of catecholamines. Manning and Wright (1983) reported that during childbirth, higher self-efficacy judgments were related to fewer requests for pain medication. Additionally, efficacy judgments were better predictors of request for medication than desire for a medication free delivery. Vallis and Bucher (1986), in an analogue pain tolerance experiment, examined the 3 phases of stress inoculation training (Meichenbaum, 1977) in facilitating pain tolerance. The phases included education, skills acquisition, and application. Subjects were asked to rate the magnitiude and strength of their self-efficacy on a cold pressor task. They were asked how long they believed they could endure the pain and how confident they were of their beliefs. Self-efficacy change scored predicted treatment gains. Pretreatment efficacy scores predicted improvement in the nonskills training group, but not in the skills training group. The authors hypothesized that the subjects who acquired skills did not have to rely on their own resources because they were given explicit instructions in how to cope with the pain. As such, their pretreatment efficacy scores were no longer relevent to their beliefs in their ability at posttreatment.

In a comprehensive review, O'Leary (1985) concluded that the evidence as a whole consistently demonstrates that people's perceptions of their self-efficacy are related to a variety of health behaviors. As such, self-efficacy theory has been extended to a number of areas. Apart from Bandura's work, it has received limited attention in the anxiety disorders literature. Biran and Wilson (1981) examined the relationship between self-efficacy and treatment for simple phobics. They chose three types of phobias: Heights, elevators, and darkness. The authors utilized two treatments, cognitive restructuring and guided exposure.

Cognitive restructuring relied on an examination and attempted alteration of irrational beliefs. Guided exposure was conducted with the experimenters. The results indicated that guided exposure enhanced approach behavior and self-efficacy as compared to cognitive restructuring. Additionally, subjects in the guided exposure reported less fear and demonstrated less physiological arousal when confronted with the phobic stimuli. There was a high correlation between approach behavior and self-efficacy estimations across conditions.

Self-efficacy theory is not without its critics. The two most meaningful and common criticisms are that self-efficacy posits an unnecessary cognitive mediator (e.g. Borkovec, 1978) and that there is no distinction between efficacy and outcome expectations (e.g. Borkovec, 1978; Marzillier & Eastman, 1984; Teasdale, 1978). Borkovec (1978) states that learning theory is a more parsimonious explanation of behavior change than self-efficacy or any cognitively mediated theory. He indicates that self-efficacy is a reflection of change rather than a mediator of such. Bandura (1984) relies on prior research to address this criticism. He cites the results of the Bandura et al. (1980) studies which indicate that a nonenactive treatment can facilitate behavior change. He also responds that even when capable of performing a target behavior, people vary in the strength of their efficacy expectation. This results in differential levels of fear and anticipatory anxiety. Thus, behavior change itself cannot account for all of the research findings.

The second criticism has received more extensive attention. For example, Marzillier and Eastman (1984) claim that a task cannot be defined without reference to some outcome and that outcome expectations determine future behavior regardless of individuals' perceptions of their ability to perform such behavior. They provide no empirical evidence to support these views.

Instead, they rely on intuitive examples. Bandura also responds with intuitive examples. While these are perhaps thought provoking, they merely reflect the theoretical stance of the authors. However, there is some evidence to support the position that the two expectations can be differentiated. Collins (1982, cited in Bandura, 1984) examined mathematical ability in school age children. Students were divided into high, average, and low arithmetic skill on the basis of standardized tests. Within each level, students were divided into high and low perceived self-efficacy. They were then asked to solve difficult arithmetic problems. At all three levels of ability, students of high self-efficacy solved more problems correctly. Thus, perceptions of ability were additionally meaningful beyond ability per se.

When applied to the experience of a panic attack, self-efficacy theory would posit that individuals would feel incapable of managing their symptomatic experiences. Rather than actively coping with the symptoms, the panic attack sufferer would likely focus on his or her inability to control the symptoms resulting in some catastrophic thoughts such as impending death or insanity. Their belief in their personal efficacy to cope with the attack would short circuit this process. Billings, Cronkite, and Moos (1983) have defined coping as "cognitions and behaviors that serve to appraise the meaning of stressors, to control or reduce stressful circumstances, and to moderate the affective arousal that accompanies stress" (p. 120). Although the definition was proposed for coping with depression, it can be readily applied to panic attacks. In general, based on Billings et al.'s definition, coping with a panic attack would include realistically examining the initial aspects of the attack, actively coping in some way to reduce or control the attack while avoiding exacerbating thoughts and/or behaviors. This appears to rely on the belief that one is capable of handling the aversive experience of an attack.

Panic attacks are, by definition, frightening experiences. Coping with such experiences appears an important component of cognitive-behavioral treatment of panic. The mechanisms of this ability to cope with panic attacks as well as the interrelationship of coping and panic symptomatology have been largely unexplored.

Summary and Purposes of the Current Investigation

Panic attacks are quite prevalent (Barlow et al., 1985). As such, they have become a focus of much recent research. Two main, but related, spheres of inquiry concern the etiology and successful treatment of the panic. The etiology of panic has been linked to genetic and biological factors. While there are clearly some biological links (e.g. Noyes et al., 1986; Sheehan et al., 1985), these are insufficient to account completely for the current data on panic attacks. For example, individuals with panic attacks have idiosyncratic stressors (Hibbert, 1984a) and significantly more life stressors preceding the onset of their first attack (Faravelli, 1985). A rapproachment considering both physical and psychological factors appears most appropriate to account for the total body of data. A conceptualization was presented which incorporates these factors (Clum & Pickett, 1984). In this and similar conceptualizations (e.g. Clark, 1986), cognitions emerge as important throughout the data and within all levels of the conceptual model.

A variety of researchers have proposed a link between cognitions and arousal. Specific to anxiety and panic, Lang and his colleagues (1983) demonstrated that phobic subjects could be trained to respond to imaged scenes in varied ways depending on the nature of their training. That is, a cognitive change could alter patterns of responding. This does not address whether subjects would respond similarly in a naturalistic setting. Hibbert (1984a) identified

characteristic ideations of panic subjects. These centered on personal harm and danger. Beck and colleagues (1974) reported that anxiety subjects had automatic thoughts related to dying and danger themes. Last et al. (1985) showed that negative thoughts were correlated with anxiety. These studies consistently support a relationship between cognitions and anxiety. However, the methodologies rely on techniques such as retrospective self-report of a traumatic incident and correlational data between thoughts and anxiety level. Neither technique allows for a clear test of directionality or causality.

A recent methodology emerged which could potentially correct these deficits. It involves the induction of panic symptoms by various means including sodium lactate infusions (e.g. Sheehan et al., 1985), administration of concentrated doses of caffeine (e.g. Charney et al., 1985), and hyperventilation (e.g. Bonn et al., 1984). However, problems are apparent in the induction literature. First, the existence of panic is based on self-report and subjects are not very accurate concerning their physiological experiences. Further, expectancies are not controlled. Thus, while it offers a potentially useful tool for examining "naturally" occurring cognitions, this is not done. A more relevant difficulty is that the induction procedures reliably elicit diagnosable symptoms, but not subjective attacks. Empirical and anecdotal evidence points to subjects' attributions about the source of their symptoms as their reason for not experiencing attacks as they know them (e.g. Liebowitz et al., 1986; Rapee, 1986). Thus, while cognitions are not directly assessed, the induction procedure provides indirect evidence of their importance in panic attacks. Making the induction more realistic in terms of what the subject actually experiences appears a more viable procedure for examining the relationship between cognitions and symptoms. One approach which allows for increased realistic presentation of the panic

experience is induction throught imagery.

The second major body of research concerns treatments for panic attacks. Cognitive-behavioral treatments have been advanced (e.g. Ascher, 1981; Last et al., 1984). These were reviewed with the finding that results are limited, but encouraging. A new treatment, Guided Imaginal Coping (Clum, 1986), specifically targets the coping strategies of panic sufferers. Pilot results (Borden & Hayes, 1986; Borden, Clum & Broyles, 1986) demonstrate that the treatment is efficacious in symptom and thought reduction during an attack as well as in increased coping effectiveness and reduction in number of attacks. Correlational analyses in these pilot studies supported a relationship between coping and improvement.

Results of panic treatment studies to date are limited by several factors. The most important of these are limited numbers of subjects, poorly defined dependent variables and measures, lack of dismantling of treatment packages, and lack of cognitive assessments from treatments which claim cognitive changes. Despite these limitations, some evidence exists that cognitions are involved in panic attacks and may indeed provide the critical link which transforms symptoms into a subjective attack.

One method for examining the mediational role of cognitions is Bandura's (1977) theory of self-efficacy. The theory, research (e.g. Bandura & Adams, 1977; DiClemente, 1981), and criticisms (e.g. Borkovec, 1978) were examined. The theory was then applied to an analysis of panic attacks. Based on a self-efficacy analysis, panic individuals would feel incapable of managing their symptoms, tend to focus on their inability to control these symptoms resulting in catastrophic thoughts and theoretically an escalation of symptoms. A treatment aimed at these thoughts may provide a successful treatment approach in that it would intervene before

symptoms could escalate or peak serving to short circuit the attack.

In sum, a variety of findings converge on the importance of cognitions in panic attacks. However, they have not been systematically evaluated for a number of reasons. First, there are few studies which specifically examine subjects with panic attacks. Second, those studies which do exist have very limited numbers of subjects. More importantly, with the limited numbers of subjects, the studies continue to rely on group methodologies which prevent detailed examination of cognitive change and the improvement process across time. Finally, the research to date has generally excluded specific and systematic attempts to alter cognitions and systematic evaluations of the impact of such intervention.

The purposes of the current investigation were to address these difficulties by systematically evaluating the process of change in the treatment of individuals with panic attacks and to examine the issue of causality concerning thoughts and symptoms of an attack. This was completed with a mixed design to allow more intensive examination of the process of change over time. The efficacy of frequently repeated measures for clinical research problems has been addressed by various authors (e.g. Kratochwill, 1978; Kratochwill & Mace, 1984). They suggest that repeated measures offer a viable alternative to traditional group designs because of their unique ability to evaluate the effects of treatment and because of the quality of information obtained by frequently repeated measures.

The current investigation had five hypotheses. First, it was expected that GIC, as compared to a nondirective treatment not specifically aimed at refining individuals' coping repertoires, would result in decreased number of panic attacks following treatment. Second, it was expected that the self-efficacy of individuals with panic attacks would be increased as a function of Guided

Imaginal Coping treatment as compared to a nondirective treatment. Third, self-efficacy was hypothesized to be predictive of coping. Fourth, based on the data collected over treatment, changes in self-efficacy were expected to precede changes in thoughts and coping and to be related to the total number of catastrophic thoughts and to the total number of adaptive coping strategies. Fifth, changes in self-efficacy were also expected to precede changes in symptoms and avoidance and to be related to the total number of anxiety symptoms experienced and to the level of avoidance, where present.

METHOD

Design

The investigation was conducted using a mixed design with one between subject factor, treatment, and one within subject factor, time. Two different treatments were administered: Guided Imaginal Coping and Panic Education. Subjects received only one treatment. Repeated measures over time were obtained before, during, and after treatment on the following dependent variables: self-reported self-efficacy, evaluator ratings of coping during an anxiety induction procedure, total number of symptoms, total number of catastrophic thoughts, number of attacks, level of avoidance, and subjects' ratings of their own total coping frequency.

Subjects

Recruitment. Potential subjects were recruited through three procedures: a mass mailing of flyers offering free assessments for anxiety and related concerns, a press release concerning the ongoing Anxiety Project of Virginia Polytechnic Institute and State University, and through an Introductory Psychology pool. Subjects from all three sources were required to meet the same criteria as described below.

Individuals interested in the project contacted the project coordinator, an advanced clinical psychology graduate student, by telephone or by writing. They were then contacted and interviewed by the project director, a licensed clinical psychologist, or by an advanced clinical psychology graduate student experienced in the assessment and treatment of anxiety disorders. Approximately 100 individuals were assessed. Assessments were conducted with the Anxiety

Disorders Interview Schedule (ADIS: DiNardo, O'Brien, Barlow, Waddell, & Blanchard, 1983). The ADIS is a structured interview which was designed to make differential diagnoses among the DSM-III anxiety disorders categories. The authors have reported high (range: k = .658 to k = .853) kappa coefficients for specific diagnostic category agreement on all but Generalized Anxiety Disorder (k = .467). All interviewers were trained by the project director or coordinator (who had been trained previously). Training involved a review of diagnostic criteria, role plays, and discussion of cases. All diagnoses of subjects were reviewed by either the project director or coordinator. Concensus diagnoses were then made. Because the goal of assessments was to identify individuals suffering from Panic Disorder or Agoraphobia with panic attacks, finer diagnostic discriminations between other anxiety disorders were not required. Panic is a relatively clear phenomenon and can be clearly reported by most patients. As such formal reliability checks were not conducted. Instead, the consensus review process was utilized. No disagreements in diagnosis occurred for patients diagnoses as Panic Disorder or Agoraphobic in the current study.

Selection. Subjects were selected based on the following criteria: (1) those who received a diagnosis of Panic Disorder or Agoraphobia with Panic Attacks and (2) in addition to the diagnosis, subjects had to report current panic attacks. This procedure yielded 21 subjects. Ten subjects, five per group, were recruited from the press release. Two were recruited from the Introductory Psychology pool with one subject assigned to GIC and one to PE. Finally, seven subjects were recruited from the flyer with four assigned to GIC and three to PE. These individuals met the above criteria and were interested in treatment. The first five subjects were assigned to GIC, the next five to Panic Education, and so on. One subject from each of the

two treatment groups dropped out after beginning treatment. Both of these subjects reported difficulty with the considerable time required for the treatment program.

The final pool of subjects consisted of 19 individuals who were assigned to one of two treatments. Subjects in both groups were quite similar with no significant differences on any demographic measure. The typical subject in either group was female, married, age 35, with 13 years education who had experienced problems with panic for about 5 years. Seven subjects took medication for anxiety throughout treatment. Two individuals from each group took Xanax. Two GIC subjects took Ativan as needed and one PE subject took Valium. The demographics for both groups and statistical tests examining potential differences between the two groups are presented in Table 1.

Procedure

Overview of Procedure. A potential subject contacted the project staff. The subject then completed the ADIS (DiNardo et al., 1983). Subjects meeting criteria for Panic Disorder or Agoraphobia with Panic Attacks were invited to participate in the study. Additionally, these subjects had to report current panic attacks. The subject was assigned to one of the two treatment conditions. The subject completed a questionnaire to assess ability to image. Subjects scoring above the normative mean, indicative of poor ability to image, were trained until able to meet criteria on the imagery inventory. The subject then completed a pretreatment evaluation consisting of a taped anxiety induction procedure and five questionnaires. The subject then began treatment, consisting of six individual and four group treatment sessions. Each week during treatment, the subject completed the evaluation procedure of the taped induction and questionnaires. The subject also completed this evaluation at posttreatment and at one and two

Table 1

Demographic Characteristics by Treatment Groups

	Group					
	GIC		PE			
Variable	X	SD	X	SD	t	chi square
Age	34.9	11.8	35.8	12.5	-0.16	
Gender	9 female		8 female			0.02
	1 male		1 male			
Education (years)	13.8	2.9	13.1	2.2	0.57	
Length Illness (years)	5.1	3.5	5.3	6.2	-0.10	
Marital Status	3 single		2 single			1.39
	4 married		5 married			
	2 divorced		2 divorced			
	1 widowed		0 widowed			

month follow-up periods.

Treatment. Subjects were assigned to one of the two treatment conditions based on order of assessment. One group received Guided Imaginal Coping. The other received a nondirective treatment identified as Panic Education. Both treatments contained 6 individual and 4 group sessions. All treatment was offered free of charge in exchange for the considerable time investment required of each subject. Two group treatments of each condition were conducted.

Guided Imaginal Coping (GIC). The overall goal of the GIC treatment procedure was to teach clients to cope effectively with their attacks and to thereby reduce the frequency and severity of attacks. GIC had four specific goals. First, the treatment sought to identify through imagery stimuli and cues which trigger or exacerbate panic attacks. The second goal was to identify the coping techniques which the client was currently using with the recognition that some of these were adaptive while others were not. Adaptive techniques were defined as those which decrease anxiety, decrease anxiety inducing cognitions, increase self-efficacy, and increase rather than decrease the number of situations in which the individual can function effectively. Avoidant techniques, which were considered nonadaptive, include leaving a stressful situation, avoiding thinking about the situation or stressor, and avoiding the situation itself. Greist, Marks, Berlin, Gournay, and Noshirvani (1980) demonstrated that nonavoidant coping techniques are superior to avoidant ones. In fact, the subjects' phobias in that investigation were worsened somewhat by the avoidant strategies. Avoidant strategies would also be predicted by efficacy theory to be negative in that subjects are not allowed the opportunity to develop the expectations that they can successfully manage their anxiety in stressful situations. The third goal was to develop in the client the continued use of adaptive strategies while substituting for

the nonadaptive ones. Again, this was done in the context of imagery, allowing the subject to examine the impact of the strategies. A variety of strategies fit the above criteria. The selection of which to use varied with the individual based upon the effectiveness for that particular person and the perceived personal cost to the individual. The final goal was to help the patient confront any previously avoided situations.

The GIC procedure can be divided into six distinct components: (1) induction of symptoms, (2) identification of currently used coping stratgies, (3) substitution of more adaptive coping strategies, (4) cognitive examination of the strategies, (5) induction of increased symptoms, and (6) repetition of steps 2-4.

Symptoms can be induced in a variety of ways. After a thorough assessment, the therapist has performed an analysis of the situations, symptoms, and cognitions relevant to each individual client's panic attacks. With this information, the client can be instructed to image the type of situations which directly or indirectly predispose him or her to panic attacks. Using idiosyncratic stressors, the imaged situations can be made more relevant to the individual. Symptoms can be induced with the individual's idiosyncratic description of symptoms. Finally, their report of their cognitive interpretation can be added to augment the induced symptoms.

Following the symptom induction, subjects were asked to identify their coping techniques. This was still within the confines of imagery. The ineffective were then sorted from the effective. Clients were thus given an immediate demonstration of the ineffectiveness of a portion of their strategies. Providing this demonstration in imagery allowed the client to observe rather than to be merely told about the difference in types of coping. Their reaction to the ineffective strategies, their feelings about how they managed the stressor, and their reactions to

avoidance can all be identified and employed as rationale for attempting more adaptive techniques. These techniques were substituted into the imaginal scene through direct instruction by the therapist. Clients were then asked to cognitively evaluate the effectiveness in managing their symptoms and their affect to the imaginal scene. Symptoms were then increased through similar mechanisms as above and the process was repeated. A typical session involved a number of escalations. With each repetition, the patient was instructed to take a more active role in providing coping strategies and realistically evaluating them.

The group component of GIC was generally aimed at demonstrating to patients that they are not alone in their experience of panic, providing the patients with information about the cause and maintenance of panic attacks, and teaching them to cope effectively with the situations that may be particularly stressful and thus perhaps increase the risk for the occurrence of an attack. The first group session involved introductions of therapists and group members. Next, the etiological model (see Appendix A) was presented and applied to each member's experience of panic. Predisposing psychological factors were identified where possible. Biological factors were also discussed, but treated as insufficient to account for existing data on the disorder. Cognitive interpretations of the panic symptoms were also discussed within the conceptualization of the attacks. The presently used coping strategies were then discussed. The second and subsequent groups further identified these strategies in terms of their success in reducing panic symptomatology. Adaptive strategies were encouraged, nonadaptive discouraged, and the group was used to help identify straegies for those members who had not been successful in identifying adaptive means to handle their panic attacks. The remainder of the group time was spent examining environmental factors such as the role of significant others and ways to manage

stressful life events and other factors which may be predisposing to panic attacks.

Panic Education (PE). The purpose of Panic Education was to discover the origins of panic. This involves answering the question of why the subject has panic attacks in both historical and immediate terms. Their first panic attack as well as current attacks were analyzed to determine where their panic originated. Panic Education was based on the rationale that most people with panic attacks lack knowledge about the antecedents of their panic. Rather, they experience panic as coming "out of the blue." Subjects were told that being able to connect stressors and conflicts to their panic would make the panic easier to manage. The research literature supporting the relationship between stressors and the onset of panic attacks was shared with the subjects. There are 5 basic goals of Panic Education. The first 3 are based on Yalom's (1975) discussions of group processes. The goals are as follows: (1) to instill the notion of universality - by having the subjects understand that they are not alone in their problems; (2) to instill hope of improvement - by focusing on previous treatment success and general improvement rates as well as the improvement of group members over time; (3) to instill a group norm of acceptance - by emphasizing the right of members to discuss whatever they desire; (4) to provide an opportunity for each member to uncover the origins of their panic from an historical perspective; and (5) to examine relationships between current attacks and life circumstances.

Sessions were conducted in a nondirective manner with no explicit focus by the therapist upon coping or providing coping strategies. During the first group meeting, subjects were told that the group belonged to them and that the therapist was there to facilitate it. At the end of each session, members were asked to evaluate the meeting to reinforce the notion that the group was

theirs. The therapist emphasized their role in changing those aspects of the group. During the first group meeting, all members were asked to identify themselves in whatever manner they desired. They were also asked to indicate why they were participating in treatment as well as what they expected to achieve as a function of treatment. During this initial meeting, the therapist suggested a format for conducting future sessions. The therapist discussed the literature supporting the relationship between stressors and panic. This was provided as the basis for uncovering life stressors at the time subjects' panic attacks began. The 3 additional sessions involved similar material with the continuation of uncovering, discussing current attacks and stressors, and a focus upon the group processes.

The focus of individual sessions was based on the same rationale. All 6 sessions were conducted in a nondirective manner with no prompts concerning coping strategies from the therapist. The general format included discussing how the subject's week was and whether they had a panic attack. If they had an attack, they were encouraged to describe it including why they thought it may have happened. They were also encouraged to examine the situation in which it occurred to detect possible cues. The session then focused upon uncovering historical conflicts or stressors to examine why the person has panic attacks. The first attack was used as a starting point with the subject encouraged to examine the process of their problem. The protocol for PE is presented in Appendix B.

Therapists. Six therapists conducted the individual treatment sessions. Therapists treated at least one subject from both treatment groups. The therapists were four females and two males. Three of the therapists, the project director, the project coordinator, and another advanced clinical psychology graduate student, were experienced with the GIC treatment. The

other three therapists, also advanced clinical psychology graduate students, were trained and then supervised at least weekly by the project director and coordinator. Tapes of sessions were reviewed to insure therapists were following the respective protocols. The project coordinator conducted all group therapy.

Credibility. The credibility of both treatments was evaluated at three points during treatment: pretreatment, midtreatment (after the fourth individual session), and posttreatment (after the sixth individual session). Subjects were asked to indicate their degree of confidence in the treatment, their expectations of mastery of treatment skills, and the logic of the treatment rationale. These questionnaires are presented in Appendix C.

Further Treatment. When clinically possible, subjects were asked to refrain from further treatment until the last follow-up had been completed. This was possible for all but two subjects. One subject, from the GIC group, experienced considerable depression and suicidal ideation. She required several additional sessions to address these concerns. Another subject, as a function of treatment, decided that after 40 years of not driving due to fear, she wanted to learn to drive. She was seen briefly to assist her in this new endeavor.

Evaluations. During the initial interview, each subject was asked to recount a recent panic attack. The details of the situation, their symptoms, and cognitions were recorded. The project coordinator constructed a script based upon this report for each individual subject. These were recorded on audiotape and used for all evaluation sessions. The tapes were of course unique for each subject, but followed a standard outline of imaging the scene, initial experience of symptoms, initial cognitive attributions about symptoms, intensification of symptoms, intensification of cognitions, a full-blown attack of 15 minutes duration, a 30 minute attack, an

attack lasting several hours, and an attack lasting all day which does not appear to be abating. A sample transcript is presented in Appendix D.

In order to participate in the taped induction procedure, subjects were required to demonstrate the ability to adequately image. This was measured by a standard imagery questionnaire (QMI: Sheehan, 1967). Two subjects in the current study received a total score greater than the normative mean on this measure and were thus considered poor imagers. They were trained by their therapist to image more vividly through prompts and repeated practice conducted before the evaluation process and treatment began. Both subjects met criteria on the QMI following two sessions of training. There were no differences between the two treatment groups on the QMI (GIC: X=65.8, SD=13.0; PE: X=67.1, SD=11.0).

In addition to treatment, each subject met weekly with a trained evaluator. The evaluators were five females and two males, ages 20-24. They were trained by the project coordinator in the proper administration of questionnaires and in judging adaptive coping strategies. They were trained to a criterion of .80 in agreement of what constituted adaptive coping strategies. This required approximately 10 hours of training. The training manual for judging coping efficacy is presented in Appendix E. The evaluators were blind to specific hypotheses and to subjects' treatment condition. Evaluations were conducted 9 times: one pretreatment, once weekly during treatment (five times), posttreatment, and one and two month follow-ups. Each evaluation included a self-efficacy measure, the taped anxiety induction procedure conducted by the evaluator, and completion of weekly questionnaires. The evaluator began the induction procedure by playing the audiotape constructed by the project coordinator. The subjects were asked to visualize the scene as clearly as possible. During the evaluation, the evaluator stopped

at each step (e.g. imagining being in situation, experiencing initial symptoms, etc.) in the tape and asked the subject what he or she does at that point in an attack. The subject's response were recorded in writing and on audiotape and rated by the evaluator as coping or not coping adaptively. Reliability estimates were conducted by the evaluators on one-third of the subjects' data selected randomly and scored blind to time of evaluation and treatment group.

Dependent Measures.

Bett's Questionnaire Upon Mental Imagery - Revised (QMI). Subjects' ability to image was measured using a standard imagery questionnaire, the revised version of the Betts' Questionnaire upon Mental Imagery (QMI: Betts, 1909; Sheehan, 1967). The revised QMI (Sheehan, 1967) is a 35 item questionnaire tapping 7 sensory modalities (see Appendix F). The measure has adequate construct validity (White, Ashton, & Law, 1978) and is not correlated with social desirability (Hiscock, 1978). The total scores from the QMI range from 35 to 245 where lower scores reflect increased vividness of imagery. White, Ashton, and Law (1974) reported a normative mean total score of 92.

Evaluation Questionnaires. Subjects completed the following questionnaires at each evaluation period. These were completed before beginning the taped induction each time so as not to confound experiences from the induction and from their week (or month at follow-up). Questionnaires were administered in a random order during each evaluation. The following questionnaires were administered.

Self-Efficacy Questionnaire (SEQ). The SEQ is an 11 item questionnaire concerning subjects' perceptions of their abilities to cope with the various aspects of a panic attack. Each item has two parts. The first part assesses whether or not the individual believes

that he or she can cope with various aspects of the attack. The second part assesses on a 9-point Likert-like scale their degree of confidence that they can do such. These are what Bandura (1977) has referred to as magnitude and strength. The questionnaire was designed to parallel self-efficacy questionnaires in other areas of research (see Appendix G).

Avoidance Questionnaire (AQ). The AQ is a 21 item checklist of situations people with panic and anxiety often find difficult to enter. Subjects were asked to indicate on a 5-point scale how often they avoided these situations in the previous week (or month for follw-up evaluations). This questionnaire is based on a portion of the ADIS (DiNardo et al., 1983). A total score is obtained by summing the rated score for each item (see Appendix H).

Panic Attack Cognitions Questionnaire (PACQ). This scale contains 25 negative cognitions associated with panic attacks. Each item is rated on a 4-point scale indicating degree of preoccupation with each cognition during a panic attack. Scores range from 1-not at all to 4-totally dominates. A total score is obtained by summing the scores for the 25 items. Previous validation of the questionnaire has demonstrated that the total score on the PACQ can reliably differentiate panic sufferers from anxious subjects who do not experience panic. Internal consistency reliabilities are adequate with a Cronbach alpha of .88 for the entire PACQ and alphas for parts 1 and 2 of .84 and .71 respectively. Further, discriminant function analyses indicate that the PACQ contributes uniquely to the differentiation of individuals with panic attacks and those without panic attacks (Clum, Broyles, Borden, & Watkins, 1987). (see Appendix I).

Panic Attack Symptoms Questionnaire (PASQ). The PASQ is a 36 item scale reflecting symptoms frequently experienced during a panic attack. Each item is rated on a

6-point scale indicating duration of the symptoms, ranging from 1- do not experience to 6-protracted period of 24 hours to 2 days or more. The total score is derived by summing the ratings across all times yeilding a total rating of severity for the panic attack symptoms. This questionnaire also asks subjects to indicate the number of panic attacks they experienced during the past week (or month for follow-ups). The subjects responded with a frequency count of attacks experienced during either the past week or month for follow-up periods. Previous validation of the questionnaire has shown that the PASQ also reliably differentiates panic and anxious, but nonpanic, sufferers. Internal consistency is also high, with a Cronbach alpha of .88 for the entire PASQ and alphas for parts 1 and 2 of .82 and .78 respectively. Discriminant function analyses also demonstrated that the PASQ contributes uniquely to the differentiation of panic and anxious nonpanic subjects (Clum et al., 1987). (see Appendix J).

Coping Questionnaire (CQ). The CQ is a 27 item self-report questionnaire designed to assess how individuals cope with panic and extreme anxiety. Each coping strategy is rated on a 4-point utilization scale where 1 = do not use and 4 = use often. A total score is obtained by adding the rated utilization for each strategy. Previous validation (Borden, Clum, Broyles, & Watkins, 1987) has demonstrated that the CQ reliably differentiates panic and anxious, but nonpanic, subjects. Internal consistency is high with a Cronbach alpha of .77 and a Spearman-Brown of .78. In the current investigation, an abbreviated version of the questionnaire was utilized. Items which had been identified a priori as nonadaptive coping were excluded in order to obtain a measure of adaptive coping. As this version relied upon the previously validated one, additional psychometric properties were not computed (see Appendix K).

RESULTS

Preliminary Analyses

Reliability. Reliability estimates were conducted on coping judgments during the imaginal scene on one-third of the subjects' data selected randomly. All evaluators completed reliability estimates. Evaluators were blind to subjects' status in terms of treatment group and time of evaluation. A stringent method of reliability computation was employed. The total number of agreements was divided by the total number of agreements and disagreements (Hartmann, 1977). This was multiplied by 100 to reflect an overall percentage of agreement. Reliability was 93% indicating that the evaluators were highly consistent in what they rated as adaptive coping.

Credibility. Subjects were asked a number of questions concerning the credibility of treatment at pretreatment, midtreatment, and posttreatment. Credibility was examined in a number of ways. First, total scores were obtained by summing scores on all three credibility questionnaires for each subjects. A t-test compared total scores for the GIC and PE groups. There was no difference between the groups on total credibility. Next, each item was analyzed to determine whether there were any relevant differences between groups. Four items were assessed at all three time periods. The first item examined the logic of the treatment. There were no main effects or interaction indicating that both groups found their respective treatments equally logical. Next, subjects were asked if they would recommend the treatment to a friend. A main effect of time emerged [$F(2,45)=3.8$, $p<.05$] with both groups reporting

higher probability of recommending the treatment to a friend as treatment progressed. There was no main effect of treatment nor was there a significant interaction. Next, subjects were asked if the treatment they received should be made available to others. There was a main effect of group with the PE group consistently indicating that it was more important to make the treatment available to others [$F(1,45)=5.72$, $p<.05$]. There was no main effect of time nor significant interaction. Interestingly, there was no variance within the PE group. All subjects at all time periods rated the item as a 9, the highest score. Although there were small differences between groups at each time period (8.4 vs 9.0; 8.9 vs 9.0; 8.7 vs 9.0), this lack of variance may have resulted in the significant F. Finally, subjects were asked to rate the level of mastery that they expected to achieve from the treatment. There were no differences between groups or over time.

Two items were assessed at pretreatment and midtreatment. Accordingly, 2 x 2 ANOVAs were conducted. Subjects were asked how confident they were that the treatment would reduce panic. There were no differences between groups or over time. Subjects were also asked how successful the treatment would be in decreasing other problems in the future. Again, there was a main effect of group with the PE group indicating higher belief in the success of the treatment in reducing future problems [$F(1,32)=5.05$, $p<.05$]. Because the PE treatment was nonspecific to panic attacks and dealt with areas of life conflicts, this difference is not surprising.

Finally, two items were assessed only at posttreatment. Subjects were asked the likelihood of their problems worsening following treatment and the likelihood that their problems would continue to diminish. T-tests between groups were conducted and revealed no differences on

these two items.

Taken together, the credibility measures suggest that the treatments were in general equally credible. Two item differences did occur. Overall, the total credibility did not differ strongly arguing that any differences between groups over time in treatment cannot be attributed to credibility differences.

Pretreatment Dependent Measures. Scores on each dependent measure were compared at pretreatment between GIC and PE to examine potential differences before treatment. Subjects in the two groups did not differ on any of the dependent measures before beginning treatment. The scores and statistical analyses are presented in Table 2.

Treatment Outcome. Because the symptoms and catastrophic thoughts of a panic attack are the most salient aspects to panic sufferers, the changes in these variables were examined between the two treatment groups. Examination of Figure 2 revealed somewhat different patterns of change in the two treatment groups. Both groups showed similar patterns of change over time. However, closer examination of the changes in symptoms and cognitions presented subtle differences. GIC subjects demonstrated a smooth, continuous reduction in symptoms. This is contrasted with the PE group who experienced an abrupt increase in symptoms from week 3 to 4 which does not return to prior levels until week 6. They also did not show continued reduction in symptoms during follow-up periods as did the GIC subjects.

Similarly with cognitions, the GIC group showed a rapid, continuous reduction with a slight increase and then stabilization. The PE group also dropped immediately, but quickly increased (week 2 - 3) before continuing to decrease. Posttreatment to follow-up represented subtle increases in cognitions rather than maintenance. While the sample size limits definitive

Table 2

Pretreatment Scores on Dependent Measures for GIC and PE Groups

	Group				
	GIC		PE		
Dependent Measure	X	SD	X	SD	t
Self-Efficacy (SEQ)	26.4	8.97	31.11	12.79	-0.94
Rated Coping	4.1	2.56	2.7	2.55	1.22
Total Thoughts (PACQ)	54.0	19.43	49.4	13.49	0.59
Total Symptoms (PASQ)	84.7	20.72	93.0	23.15	-0.83
Total Coping (CQ)	33.9	6.90	37.11	3.89	-1.23
Avoidance (AQ)	37.2	11.10	39.33	17.79	-0.32
Number Attacks (in past week)	3.5	3.24	2.4	1.59	0.88

statements, these results may point to the differences in the two treatments. GIC systematically aimed to decrease symptoms and catastrophic thoughts through focused intervention while PE allowed subjects to "find their own way." The lack of smooth curves paralleled this approach. Perhaps by trial and error, the PE subjects learned what was helpful and what was not.

More formally, the improvement of both groups was compared on the following measures: PASQ, PACQ, CQ, AQ, and number of attacks. These measures were compared at four time periods: pretreatment, posttreatment, 1 month follow-up, and 2 month follow-up yielding a 2 (tx: GIC vs PE) X 4 (time) MANOVA. A significant effect of time emerged for all variables taken together [$F(15,128)=7.26$, $p<.001$]. Further, a significant group by time interaction was found [$F(15,128)=1.95$, $p<.05$]. The main effect of group approached significance [$F(5,12)=2.58$, $p<.08$]. Given the significance of the MANOVA, post hoc analyses were conducted on each dependent measure. First, a group x time ANOVA was conducted on each dependent variable.

The group x time ANOVA for symptoms (PASQ) revealed a significant main effect of time [$F(3,64)=14.82$, $p<.0001$]. There was no main effect of group nor significant interaction of group and time. This indicates that both groups experienced symptom reduction over time. The overall one-way ANOVAs for each group over time were significant [GIC: $F(3,36)=7.98$, $p<.005$; PE: $F(3,29)=7.74$, $p<.005$]. Scores within group at each time period were then examined to reveal where significant changes occurred. All post hoc comparisons were conducted using Tukey's test to help control for experimentwise error rate. An alpha level of .05 was used for all Tukey comparisons. The GIC group reported significant decreases from pretreatment to posttreatment, from pretreatment to both follow-up periods, and continued reduction from the 1

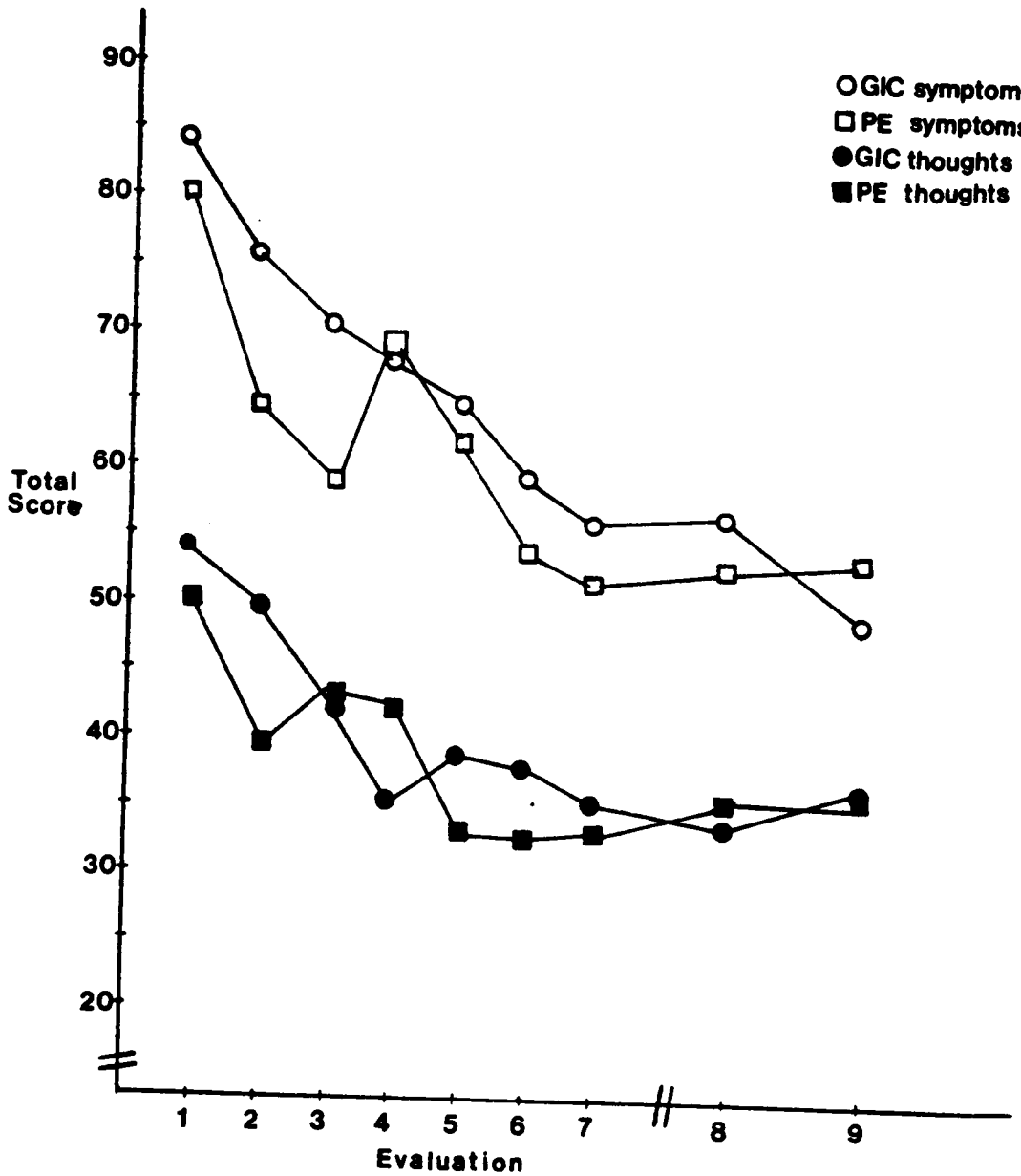

Figure 2

Changes in Symptoms and Catastrophic Thoughts Between Treatment Groups

month follow-up to the 2 month follow-up. The PE group reported significant decreases from pretreatment to posttreatment and from pretreatment to both follow-up periods. There was no further significant reduction from the 1 month follow-up to the second follow-up. In fact, the PE group increased slightly in their total symptoms while the GIC group continued to improve on this variable. These results are presented in Table 3.

Total thoughts during a panic attack (PACQ) were next examined. The group by time ANOVA demonstrated the same pattern as was seen with the PASQ. The only significant finding was a main effect of time [$F(3,64)=8.14$, $p<.005$]. Examination of the within group scores revealed similar results for both groups. Both groups showed significant effects of time [GIC: $F(3,36)=4.81$, $p<.01$; PE: $F(3,29)=3.49$, $p<.05$]. Using Tukey tests, both groups demonstrate significant reductions in catastrophic thoughts from pretreatment to posttreatment. The GIC group also demonstrated significant differences between pretreatment and both follow-up periods. These results are presented in Table 4.

Total number of attacks were then examined. Again, the only significant effect was one of time [$F(3,64)=10.88$, $p<.0001$]. Both groups showed a significant reduction in the number of attacks over time [GIC: $F(3,36)=5.63$, $p<.005$; PE: $F(3,29)=9.91$, $p<.005$]. The means at each time period are presented in Table 5. Tukey tests show that both groups reported significant reductions in number of attacks from pretreatment to posttreatment and from pretreatment to both follow-up periods. No other comparisons were significant. The percent of subjects in each group who reported no panic was also examined at posttreatment and at the two follow-up periods. At posttreatment, 70% of GIC subjects and 63% of PE subjects reported zero panic attacks. At the first follow-up, 60% of GIC and 67% of PE subjects reported no

panic. At the second follow-up, 60% of GIC and 63% of PE subjects reported having no panic attacks. The proportions between groups was no significantly different at any of these time periods.

The next dependent measure was total avoidance. The group by time ANOVA revealed main effects of group [$F(1,64)=4.12$, $p<.05$] and time [$F(3,64)=4.23$, $p<.01$]. There was no significant interaction. The GIC group reported significantly less avoidance overall. Further, only the GIC group had significant change over time [$F(3,36)=5.39$, $p<.005$]. The changes from pretreatment to posttreatment and from pretreatment to both follow-up periods were significant. These results are presented in Table 6.

Finally, total self-reported coping was examined. Interestingly, a main effect of group emerged [$F(3,64)=14.82$, $p<.005$]. There was no main effect of time nor was there a significant interaction. Examination of Table 7 revealed that the PE group employed significantly greater number of coping strategies than did the GIC group. To examine this difference further, total evaluator rated coping during evaluations was examined. A repeated measures 2 (group) X 9 (time) ANOVA revealed a significant main effect of group [$F(1,144)=27.87$, $p<.0001$] and of time [$F(8,144)=24.99$, $p<.0001$]. The interaction was nonsignificant. The means for each time period are presented in Table 8. The scores at time 1 are not significantly different (p's $>.1$). Together, these results suggest that subjects in both treatment groups increase in their ability to cope during the evaluations. However, the GIC subjects more quickly improved their coping. The PE subjects "caught up" by the second follow-up.

Table 3

Symptoms Over Time Within the GIC and PE Groups

Group	Pretreatment	Posttreatment	1 Month Follow-Up	2 Month Follow-Up
GIC	84.7*	56.5	56.4	49.9
(SD)	(20.72)	(19.43)	(15.52)	(12.76)
PE	93.0	51.4	52.4	53.5
(SD)	(23.15)	(17.99)	(14.73)	(27.82)

* values with adjoining lines are significantly different ($p < .05$)

Table 4

Catastrophic Thoughts Over Time Within the GIC and PE Groups

Group	Pretreatment	Posttreatment	1 Month Follow-Up	2 Month Follow-Up
GIC	54.0*	35.8	33.7	35.2
(SD)	(19.43)	(11.71)	(9.02)	(13.01)
PE	50.3	32.8	35.8	35.0
(SD)	(13.49)	(9.39)	(9.84)	(14.15)

* values with adjoining lines are significantly different ($p < .05$)

Table 5

Number of Attacks Over Time Within the GIC and PE Groups

Group	Pretreatment	Posttreatment	1 Month Follow-Up	2 Month Follow-Up
GIC	3.5*	0.4	1.0	0.9
(SD)	(3.24)	(0.70)	(1.25)	(1.10)
PE	2.3	0.4	0.3	0.5
(SD)	(1.59)	(0.52)	(0.46)	(0.76)

* values with adjoining lines are significantly different ($p < .05$)

Table 6

Total Avoidance Over Time Within the GIC and PE Groups

Group	Pretreatment	Posttreatment	1 Month Follow-Up	2 Month Follow-Up
GIC	37.2*	24.6	26.3	26.1
(SD)	(11.10)	(5.02)	(7.07)	(7.26)
PE**	39.3	34.9	32.5	27.4
(SD)	(17.79)	(14.96)	(14.79)	(6.74)

* values with adjoining lines are significantly different ($p < .05$)

** nonsignificant univariate ANOVA

Table 7

Total Self-Reported Coping Frequency Over Time Within the GIC and PE Groups

Group	Pretreatment	Posttreatment	1 Month Follow-Up	2 Month Follow-Up
GIC	33.9*	28.3	28.2	29.2
(SD)	(6.90)	(10.12)	(10.75)	(10.78)
PE	36.3	40.5	38.6	35.4
(SD)	(3.89)	(4.93)	(5.42)	(10.53)

* values with adjoining lines are significantly different ($p < .05$)

Primary Analyses

The following analyses addressed the primary hypotheses directly. Hypothesis #2 predicted that Guided Imaginal Coping would increase self-efficacy more than a nondirective treatment Panic Education, not directly aimed at increasing subjects' coping repertoire. Self-efficacy ratings were obtained at 9 data points (pretreatment, weeks 2-6 of treatment, posttreatment, 1 month follow-up, and 2 month follow-up) over the course of the investigation. A repeated measures ANOVA was conducted with treatment group and time as factors. A main effect of time emerged [$F(8,144)=9.31$, $p<.0001$]. There was no significant main effect of group nor was there a significant group by time interaction. The means and standard deviations for both groups over time are presented in Table 9. Both groups' efficacy scores increased. The scores appeared to increase at approximately the same rate with both groups showing significant increases in their perceived ability to cope with panic attacks.

Hypothesis #3 proposed that self-efficacy would be predictive of evaluator rated coping. Coping was defined as a dichotomous variable as rated by the evaluators. Subjects' responses were rated by the evaluators as either adaptive coping or not with a yes or a no. A point-biserial correlation was computed at each time period. The data was collapsed across treatment groups. The results are presented in Table 10. Examination of the correlations reveals that they are all significant (p's $< .01$). Further, the correlations show a general increasing trend suggesting that the relationship between self-efficacy and rated coping increased over time. Further examination of the correlations showed that while they were all significant, they account for a relatively limited amount of the total variance. Indeed, other factors besides self-efficacy and ability to cope seemed meaningful to explore.

Table 8

Rated Coping by Treatment Group Over Time

	Evaluation								
Group	1	2	3	4	5	6	7	8	9
GIC	4.1	6.0	8.0	9.7	9.5	10.0	10.4	10.9	10.9
(SD)	(2.6)	(2.4)	(2.5)	(1.4)	(2.1)	(1.6)	(0.3)	(0.3)	(0.3)
PE	2.7	3.6	7.0	7.5	8.4	8.1	8.4	9.6	10.4
(SD)	(2.4)	(2.1)	(2.1)	(2.4)	(2.3)	(2.6)	(2.7)	(2.7)	(1.4)

Table 9

Self-Efficacy Scores by Group Over Time

	Evaluation								
Group	1	2	3	4	5	6	7	8	9
GIC	26.4	29.5	38.0	49.3	49.7	53.5	59.4	68.5	71.0
(SD)	(8.5)	(17.4)	(18.4)	(26.9)	(23.9)	(26.0)	(24.8)	(23.9)	(25.6)
PE	29.0	33.4	48.8	51.4	54.4	58.5	66.8	76.9	79.5
(SD)	(12.1)	(12.7)	(27.8)	(22.5)	(22.8)	(24.9)	(22.1)	(27.5)	(19.1)

Table 10

Point-biserial Correlations: Efficacy and Evaluator Rated Coping Over Time

Time	Correlations
1	.24**
2	.23**
3	.22**
4	.40**
5	.37**
6	.36**
7	.40**
8	.37**
9	.41**

** p<.01

catastrophic thoughts and to adaptive coping strategies. Total scores were obtained on all three measures at each evaluation period. Pearson product-moment correlations were computed on the interrelationship of efficacy and the other two variables. These correlations were computed with all nine data points such that correlations were obtained with variables at the same evaluation period and with variables one week apart (e.g. self-efficacy 1 - thoughts 2; thoughts 1 - self-efficacy 2). Correlations were then examined using a cross-lagged panel analyses. This procedure allowed an examination of the direction of causality in the relationship between variables. It was based on time precedence. Thus, if changes in one variable reliably preceded changes in another, it can be more strongly suggested that the first was causally related to changes in the second. The analysis used 9 panels allowing increased confidence in the findings.

To visually examine the relationship between self-efficacy and catastrophic thoughts, total scores were plotted over time. These are presented in Figure 3. As can be seen in Figure 3, self-efficacy and catastrophic thoughts are negatively related such that self-efficacy increases as catastrophic thoughts decrease. More specifics on this relationship were obtained from the cross-lagged panel analyses. These panels are presented in Figure 4. Examination of the panels provided a variety of information. First, the stability of the self-efficacy measure clearly increased over time suggesting that changes in efficacy are lasting. For example, if subjects were efficacious at time 6, they remained efficacious at time 7. This pattern was not clearly seen with the catastrophic thoughts which tended to fluctuate more. However, with one exception, the scores remained significantly related over time. Next, examining contemporaneous measures of efficacy and thoughts, there appeared to be a consistently negative relationship between the two variables. Six of the nine time periods had significantly negative

correlations indicating the higher the self-efficacy, the fewer catastrophic thoughts. This supported the earlier visual representation of this relationship. The causal pattern was next examined by comparing the crossed portions of each panel (e.g. efficacy 1 - thoughts 2 vs thoughts - 1 efficacy 2). Six of the eight comparisons were in the predicted direction with the relationship between efficacy and catastrophic thoughts at the following time period being greater than the relationship between catastrophic thoughts and efficacy at the next period. The differences between these correlations was not significant.

To further explore the relationship between self-efficacy and catastrophic thoughts, partial correlations were computed to partial out the effects of the contemporaneous variable. For example, to examine the effect of efficacy at time one on thoughts at time two, the contribution of thoughts at time one was partialed out. This allowed a purer measure of the impact of efficacy at time one on thoughts at the next assessment period. These results are presented in Table 11. Table 12 presents similar analyses examining the effect of thoughts at time one on efficacy at time two, controlling for efficacy at time one. Again, the directional relationship of efficacy to thoughts was consistently stronger then the directional relationship of catastrophic thoughts to self-efficacy.

The relationship between self-efficacy and coping frequency is presented in Figure 5. As can be seen, the relationship of coping frequency at one time period was significantly related to coping frequency at other time periods. This relationship, in general, appeared to increase over time. Efficacy and coping frequency did not appear to be significantly related either contemporaneously or over time. These results are likely confounded by the issue of differential coping frequency observed in the two treatment groups. The two groups appeared to respond

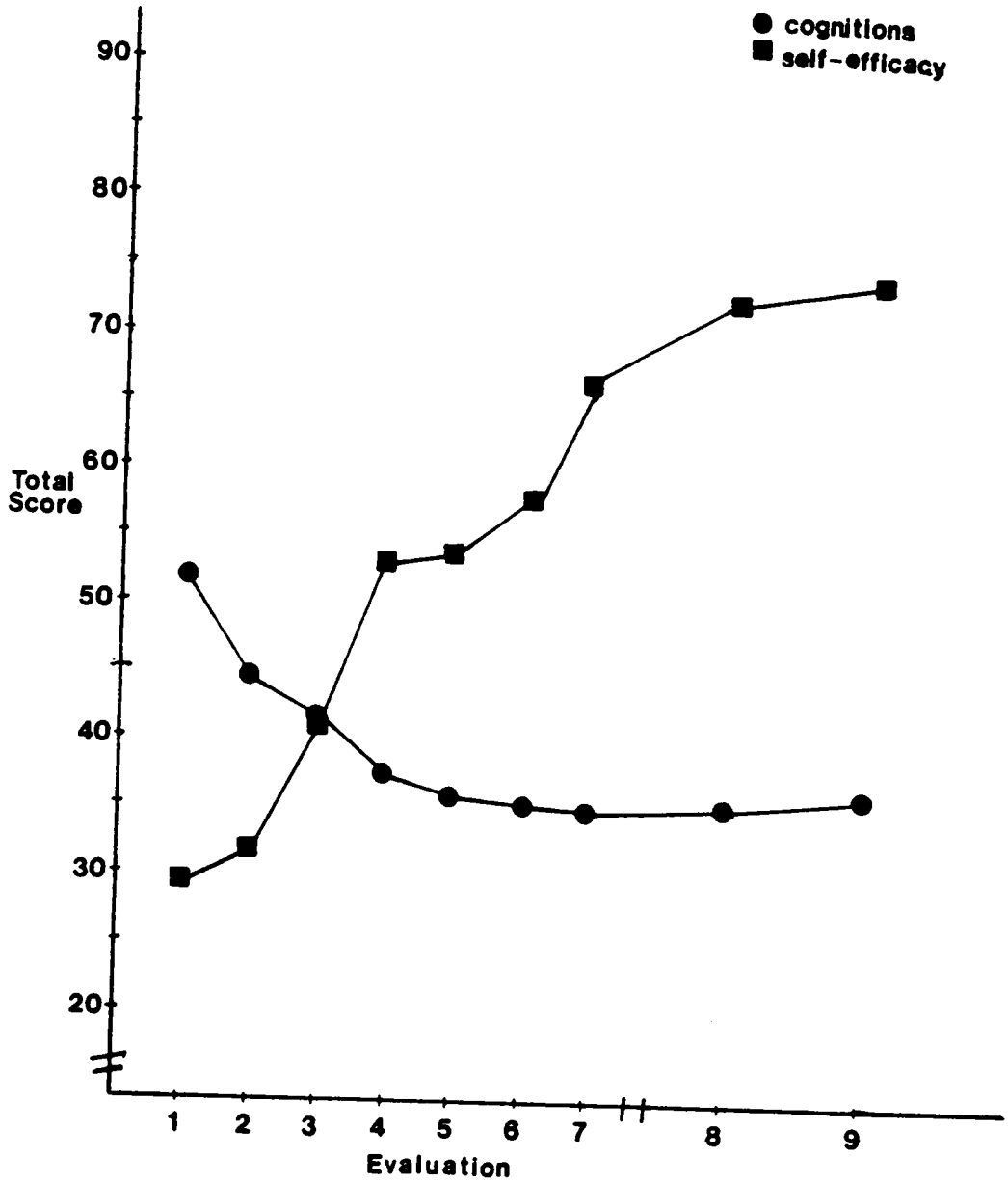

Figure 3

Relationship of Self-Efficacy and Total Catastrophic Thoughts Over Time

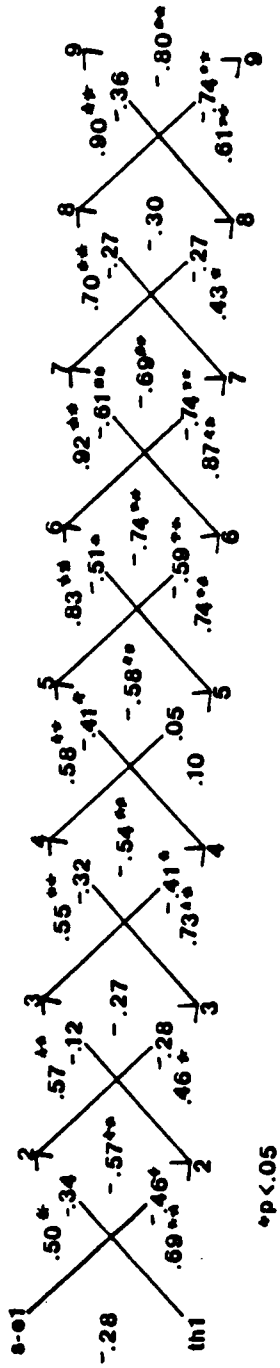

Figure 4

Cross-Lagged Panel Analyses of Self-Efficacy and Catastrophic Thoughts

Table 11

Partial Correlations:

Self-Efficacy and Thoughts, Partialing Effect of Contemporaneous Thoughts

Efficacy 1 - Thoughts 2	-0.39
Efficacy 2 - Thoughts 3	-0.03
Efficacy 3 - Thoughts 4	-0.51*
Efficacy 4 - Thoughts 5	0.12
Efficacy 5 - Thoughts 6	-0.44*
Efficacy 6 - Thoughts 7	-0.30
Efficacy 7 - Thoughts 8	0.05
Efficacy 8 - Thoughts 9	-0.75**

* $p < .05$

** $p < .01$

Table 12

Partial Correlations:

Thoughts and Self-Efficacy, Partialing Effect of Contemporaneous Self-Efficacy

Thoughts 1 - Efficacy 2	-0.24
Thoughts 2 - Efficacy 3	0.30
Thoughts 3 - Efficacy 4	-0.21
Thoughts 4 - Efficacy 5	0.15
Thoughts 5 - Efficacy 6	-0.07
Thoughts 6 - Efficacy 7	0.27
Thoughts 7 - Efficacy 8	0.41
Thoughts 8 - Efficacy 9	-0.21

differentially in terms of their coping frequency. Rated coping during the evaluations increased for both groups, with the GIC group apparently increasing faster. However, GIC subjects' frequency of self-reported coping decreased. Although subjects in general appeared to become more adept at coping, the GIC subjects decreased their frequency of coping. Further, self-efficacy increased in both treatment groups. Thus, the relationship between these two variables was not particularly clear. Efficacy increased and coping skills increased. However, coping frequency did not appear related to these increases.

Next, the relationship between self-efficacy and symptoms was examined. A visual representation of this relationship is presented in Figure 6. From this representation, a negative relationship seemed to exist such that increases in self-efficacy were related to decreases in total symptomatology. To better characterize this relationship, cross-lagged panel analyses were conducted. These are presented in Figure 7. Interestingly, the correlations of symptoms over time were not consistently high. Although six of the nine correlations were significant, they showed a great deal of variability. Examining contemporaneous scores between efficacy and symptoms, six of the nine were significant. All of these correlations were negative suggesting that the higher the self-efficacy, the fewer symptoms experienced. It is clear that these variables were consistently related. However, addressing the issue of causality did not resolve the nature of this relationship. Five of the eight crossed panels were in the expected direction with changes in self-efficacy preceding changes in symptoms. However, these correlations were mostly nonsignificant. Further, they were not significantly greater than correlations in the opposite direction. Together, these results suggested that self-efficacy and symptoms were contemporaneously related. That is, changes in efficacy led to changes in

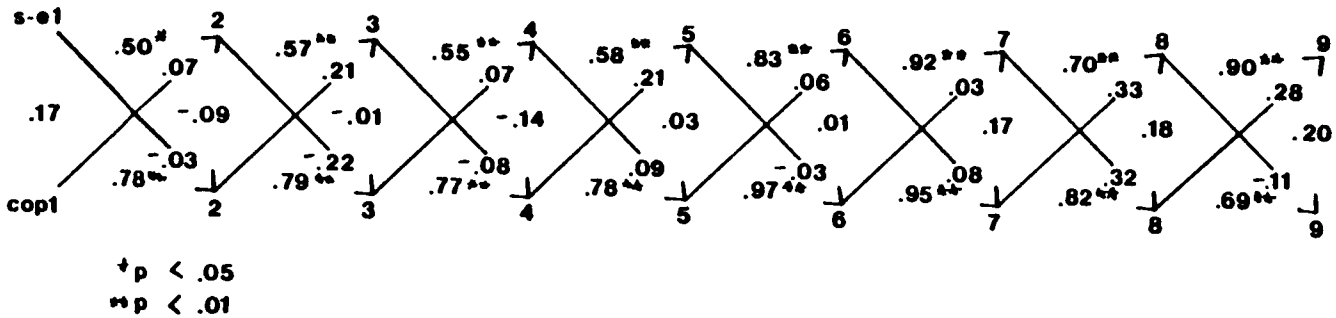

Figure 5

Cross-Lagged Panel Analyses of Self-Efficacy and Self-Reported Coping Frequency

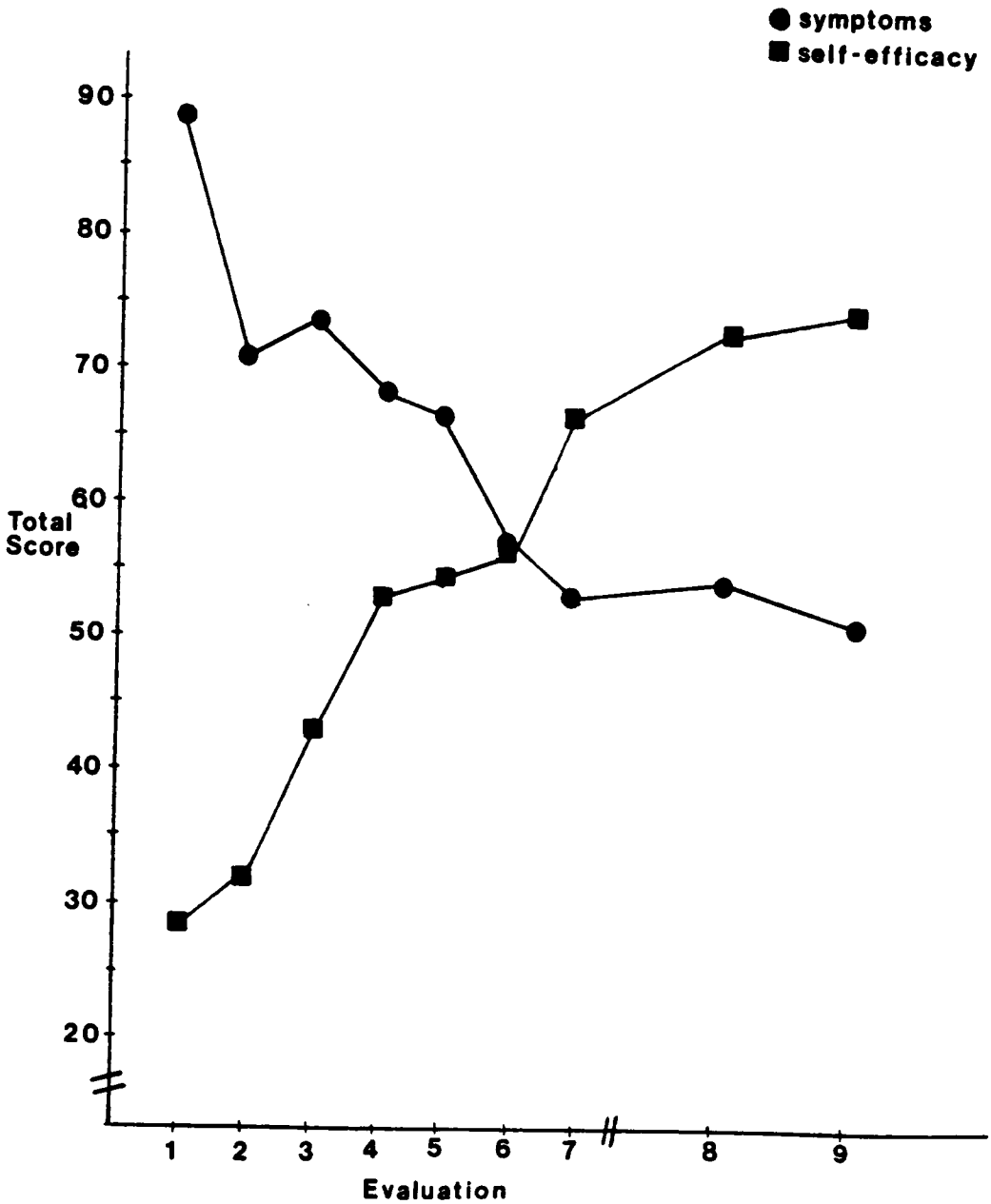

Figure 6

Relationship Between Self-Efficacy and Total Symptoms Over Time

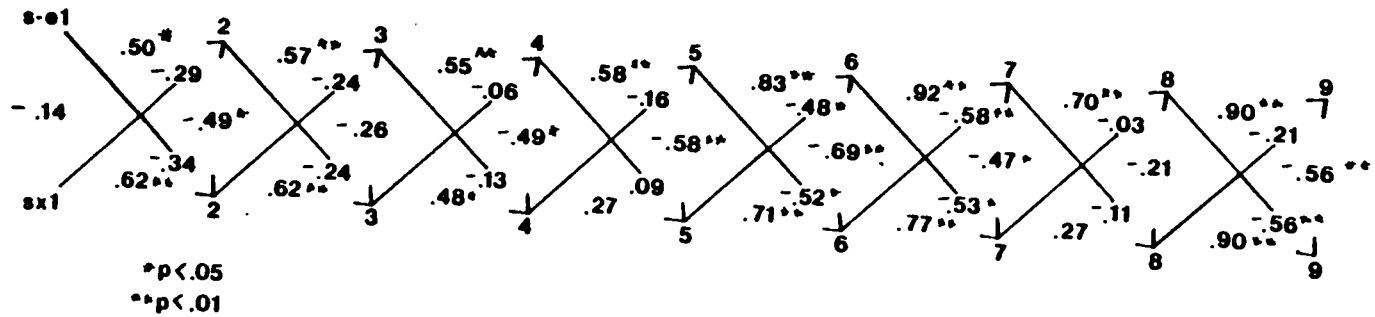

Figure 7

Cross-Lagged Panel Analyses of Self-Efficacy and Total Symptoms

symptoms and changes in symptoms then led to changes in efficacy. Interestingly, the pattern of the relationship from posttreatment to follow-up became negligible, Symptoms and efficacy did not appear related at that time. However, the relationship became stronger from one month follow-up to the two month follow-up period.

The relationship between self-efficacy and avoidance was next examined. These correlations are presented in Figure 8. Over time, avoidance remained highly stable. Overall levels of avoidance decreased over time in both groups. These results combined with the correlations suggested that subjects who began treatment with high levels of avoidance remained relatively more avoidant over time. Thus, avoidance may have been decreased, but subjects' relative status in level of avoidance remained fairly constant as compared to other subjects. Avoidance and self-efficacy did not appear consistently related either contemporaneously or over time. Only two of the eight cross panels were in the expected direction. However, they were nonsignificant. None of the contemporaneous scores reached significance. Another interesting finding was that the correlations between self-efficacy and avoidance began as negative and became positive. To allow closer inspection of the relationship between self-efficacy and avoidance, the cross-lag panels were recomputed for only those subjects who could be classified as avoiders (AQ score > 21). These correlations are presented in Figure 9. The relationship between efficacy and avoidance remained positive at most time periods even with just the avoiders examined. A moderate contemporaneous relationship was present. Further, the crossed panels again suggested that the higher the efficacy, the higher the levels of avoidance. No consistent pattern emerged to suggest causality, although at posttreatment and follow-up periods, it appeared that avoidance led to changes in efficacy.

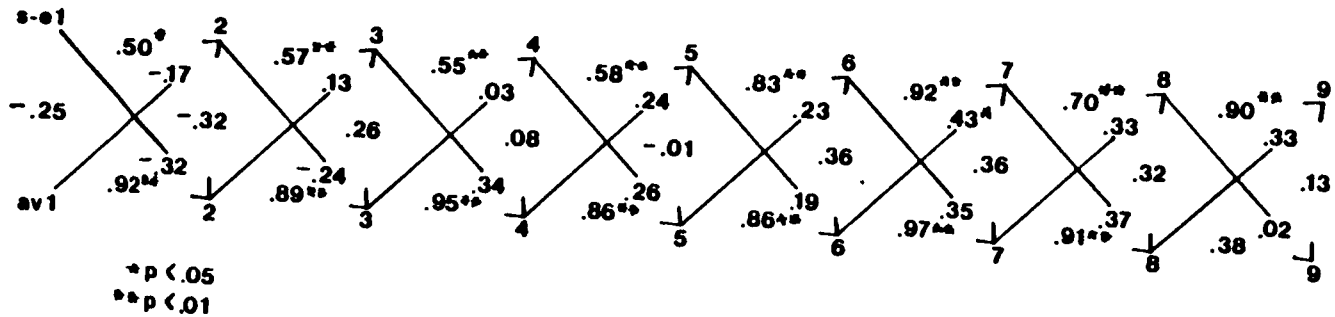

Figure 8

Cross-Lagged Panel Analyses of Self-Efficacy and Avoidance

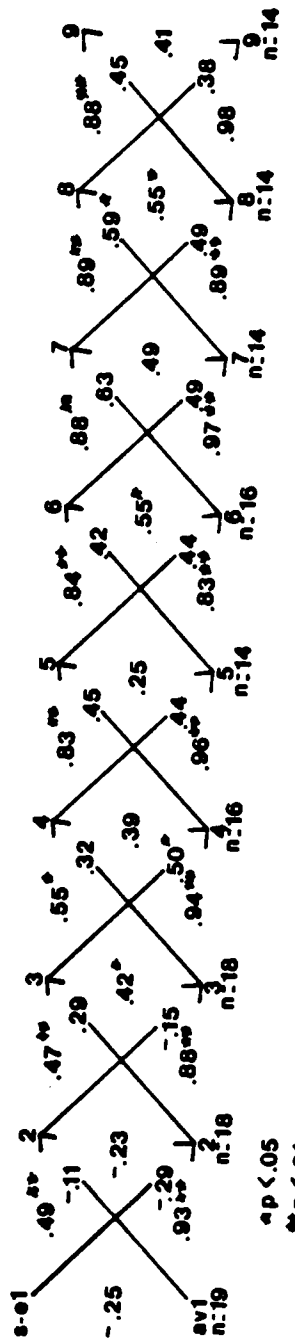

Figure 9

Cross-Lagged Panel Analyses of Self-Efficacy and Avoidance for Avoiders Only

DISCUSSION

The purposes of the current investigation were: (1) to examine whether a cognitive-behavioral treatment specifically aimed at altering the coping repertoires of panic sufferers would yield better treatment outcome than a treatment not aimed specifically at altering coping and (2) to examine whether the concept of self-efficacy would be helpful in explaining the changes in panic sufferers over the course of treatment. In examining these issues, the current study employed a design examining the changes in two treatment groups over time.

This methodology resulted in several advantages over prior research in the area of the cognitive-behavioral treatment of panic. First, the study allowed careful examination of changes over time. This allowed collection of considerable information beyond the traditional measures before and after treatment. Related to this, the evaluation procedure employed in the current study provided a means to more objectively evaluate patient progress through treatment. Progress was not only based upon self-report, but also by comparison with rated performance over time. Finally, inclusion of a reasonable control group permitted examination of how subjects would change without directive intervention to the changes seen in a group treated with specific cognitive-behavioral approaches.

Several disadvantages of the investigation must also be acknowledged. First, a no treatment control group was not included due to the considerable time required of subjects. It was decided that asking subjects to simply come for evaluations without treatment offered over the course of

several months was not appropriate. As such, the results must be interpreted with some caution. Both treatment groups improved, yet it is not clear how patients who did not receive treatment would respond on the same measures over time. Previous research suggests that on some measures of panic, a group not receiving treatment also showed some degree of improvement (Borden et al., 1987). Secondly, the current investigation utilized a relatively small number of subjects. Although some trends appeared fairly clear throughout the data, an increased sample size may have yielded other significant differences not apparent in the present results. Finally, a large number of analyses were conducted to address the hypotheses of this study relative to the number of subjects. Given the exploratory nature of this study in examining changes over time, the number of analyses exceeded the number traditionally performed with a sample of this size. When appropriate, controls were utilized in an attempt to reduce the chance of an inflated experimentwise error rate. Further, the significant findings of the correlational analyses were fairly consistent suggesting that they are likely not merely due to chance. Overall, the design and controls of this investigation permitted an expansion of current knowledge about changes over the course of treatment of panic. The weaknesses of the study were consistent with its exploratory nature and call for a somewhat cautious interpretation of the results.

The first question to be addressed by the current study is does Guided Imaginal Coping work as a treatment of panic? Visual inspection of the changes in catastrophic thoughts and symptoms suggests that GIC allowed a smoother reduction in these components of a panic attack over time than did the nondirective treatment of Panic Education. GIC also resulted in apparent continued improvement through follow-up periods whereas PE did not. Both groups clearly improved.

However, the pattern of these results may point to different mechanisms underlying change. GIC may result in a more focused and consistent alteration of panic symptoms and thoughts whereas PE may allow patients to find their own way to improvement through a trial and error process. The differences in the visual patterns representing change would support this type of difference in the mechanism of change.

Specific analyses of change in the concomitants of panic revealed several interesting findings. In general, both groups presented similar patterns of change on the majority of variables examined. For example, the analysis of symptom level indicated that both groups changed over time with a reduction of symptoms throughout treatment. Both groups showed a significant reduction from pretreatment to posttreatment. However, GIC subjects continued to show improvement from the first to the second follow-up period. PE subjects did not show significant continued improvement. As Michelson and his colleagues (1985) suggested, subjects treated with cognitive-behavioral techniques are learning skills which can be carried with them beyond the time they are in treatment. GIC subjects may have developed new specific skills which allowed them to continue to improve on this single measure of panic.

A similar overall pattern of results was found when examining the changes in catastrophic thoughts over time. Again, both groups demonstrated significant reductions in catastrophic thoughts from pretreatment to posttreatment. Surprisingly, the GIC group did not continue to improve on this measure through follow-up periods as it did with total symptoms. Examination of the scores from posttreatment to the follow-up periods suggested that subjects were consistently reporting very low scores on this measure of thoughts. A score of 25 was received when subjects reported no catastrophic thoughts. As such, the scores were almost demonstrating

a floor effect after posttreatment. Interestingly, scores on the catastrophic thoughts questionnaire almost reached their lower limit whereas scores on the symptom measure did not. Perhaps these thoughts are not as refractory to treatment as are physical sensations. Recent research maintains that normal subjects also experience the physiological sensations of panic (Norton et al., 1985). Yet, they are not necessarily panic sufferers. Perhaps these sensations, in a milder form, stay with patients long after treatment is over. Subjects did report decreased symptoms, but not to the apparent magnitude of the decreased catastrophic thoughts. An intriguing question emerges from these results. Can what is termed a panic attack occur in the absence of catastrophic thoughts? One exception to the need for thoughts to have an attack is the occurrence of nighttime panic in which a patient is awakened with an attack (Craske & Barlow, 1986). These may represent a qualitatively different phenomona from panics which occur while one is awake. Comparison of the two require further investigation. Yet, the specific differences observed in the current study would argue that symptoms can persist beyond reduction of panic attacks.

Examination of the number of attacks over time showed a pattern similar to that observed with catastrophic thoughts. Both groups demonstrated significant reductions in the number of attacks from pretreatment to posttreatment. Again, the number of attacks became very small with a limited number of subjects reporting any attacks during follow-up periods. Both treatments appeared successful in eliminating the presence of what subjects subjectively term an attack.

The avoidance comparison presented a different pattern. The main effect of group indicated that the GIC subjects reported less avoidance. The GIC group also demonstrated significant

reduction on avoidance over time whereas the PE group did not. The scores of the PE group would appear to represent a significant increase. However, they did not, most likely the result of a large error term. The GIC subjects became less avoidant as a function of treatment. When combined with other measures, this underscores the efficacy of this treatment approach. Subjects become less avoidant, but also demonstrated reductions in the measures of panic. One would expect that as individuals began to explore previously avoided situations, that their level of anxiety and panic would increase. The finding that the GIC group became less avoidant and demonstrated significant reductions in the panic measures was particularly encouraging. GIC subjects may have learned to better cope with the situations which they previously avoided. The focused approach to augment their coping abilities may be related to their ability to encounter these situations.

Accordingly, the degree to which subjects reported using adaptive coping strategies was examined. Surprisingly, a main effect of group revealed that PE subjects actually utilized a greater number of coping strategies than did the GIC subjects. Examination of coping as rated by the evaluators at each evaluation period suggested that the GIC subjects more quickly developed adaptive coping strategies. Together, these two results may provide an indication that the impact of a directive treatment aimed at improving the coping abilities of patients is to narrow the types of coping that the patients utilize. It may be that treatment does not necessarily expand the coping repertoires of patients, but rather refines them. GIC subjects were clearly coping adaptively, but using a limited number of strategies. On the other hand, PE subjects also learned to cope adaptively, but continued to utilize a number of approaches. The nondirective nature of their treatment may have led them to try a number of strategies without explicit assistance

from a therapist. Interestingly, those treated without this explicit focus eventually seemed to "find their own way."

The question then remains, does GIC work as a treatment of panic? The answer from the results of the current investigation must be yes. Subjects treated with GIC show reduced numbers of symptoms, catastrophic thoughts, and attacks over time as well as a reduced level of avoidance. Clearly, the treatment was effective in assisting these patients manage their anxiety attacks. They became proficient at coping. They reduced their level of avoidance. Overall, they appeared less anxious, at least as far as their attacks were concerned. Yet, a secondary question is whether GIC is a better treatment than any other treatment for panic. The answer to this can only be a qualified yes. When examining specific measures of avoidance and continued symptom reduction, subjects treated with GIC showed reduction over time whereas subjects treated with PE did not. Yet, the PE subjects also improved in a number of meaningful ways. Perhaps most importantly, they reported fewer panic attacks over the course of treatment. One question still remaining is how would patients who did not receive treatment compare with these two groups? Previous research (Borden et al., 1987) demonstrated that even when a group of subjects did not receive treatment, that the number of panic attacks in the group decreased over time. Future research will need to examine the process of change in the absence of treatment.

The second major issue addressed by the current investigation was whether or not Bandura's (1977) notion of self-efficacy could help to explain the changes in patients as they progress through treatment. A possible means to view the improvement in panic sufferers is to invoke Banura's idea of cognitive mediation. That is, that individuals who suffer from panic attacks may feel unable to control their attacks. In that case, they would be unlikely to effectively cope with

their somatic experiences and instead would perhaps focus upon their inability to control what was happening to them. These types of thoughts would likely be catastrophic in nature such as believing that an acceleration in one's heart rate denoted an impending heart attack and likely death. The belief that one can manage the somatic sensations may serve to prevent or minimize these catastrophic thoughts. This belief is what Bandura termed self-efficacy.

The results of the current study demonstrated that both groups became more efficacious over the course of treatment. The results also pointed to increases in adaptive coping for both groups. The third hypothesis of the current investigation proposed that self-efficacy would be predictive of coping. That is, as self-efficacy increased and a person became more confident in his or her ability to cope with the various aspects of panic, did actual improvement in coping occur? The point-by-serial correlations presented a pattern of these two variables being related over time with a relationship that increased somewhat in strength over time. Self-efficacy and rated coping were related in a positive fashion such that the higher the efficacy, the higher the rated coping and visa versa. However, the correlations did not provide an answer to the question of causality. Thus, both appeared to be relevant factors in improvement, but whether or not self-efficacy led to improved coping remains questionable. Further, the correlations were significant at every time period, but did not account for a large amount of the total variance. Clearly, these were not the only two meaningful factors.

Logic argues that the level of symptoms experienced and the degree that one catastrophizes about these symptoms would also affect the level of self-efficacy and one's ability to cope. The analyses with self-efficacy and catastrophic thoughts provided a fairly clear picture of their relationship. First, visual inspection of the data showed that as efficacy increased, these

frightening thoughts decreased. The converse is also true that as thoughts decreased, efficacy increased. This is intuitively logical in that the fewer frightening thoughts one has, the more confident one is apt to feel and the more confident one feels about managing his or her panic, the fewer catastrophic thoughts. This finding was in general consistent with prior research examining the relationship between anxiety and "negative thoughts" such as that done by Last and her colleagues (1985) where the more negative thoughts, the more anxiety. This again did not provide an answer to the question of causal relationships. Inspection of the cross-lagged panels revealed a number of interesting findings. First, the relationship of efficacy scores over time remained very stable, thoughts tended to show more fluctuation. Second, the relationship between efficacy and catastrophic thoughts was consistently negative as also apparent from the visual representation of the data discussed above. Third, based on the logic of the cross-lagged panel procedure, the causal pattern strongly argued that changes in self-efficacy led to changes in catastrophic thoughts with six of the eight panels in the predicted direction. A major limitation in interpreting these findings was that the correlations were not statistically different. This was likely related to the limited sample size which made it difficult to obtain significant differences between correlations. This limitation should be kept in mind. However, the consistency of the relationship was compelling and difficult to explain without reference to the likelihood that indeed self-efficacy preceded changes in these thoughts. To further examine the relationship between self-efficacy and catastrophic thoughts, partial correlations were computed to obtain a purer measure of the effect of each of these variables on the other at the next evaluation period. Again, these results pointed to changes in efficacy preceding changes in catastrophic thoughts.

The relationship between self-efficacy and the other variables was not as clear as it is with self-efficacy and catastrophic thoughts. For example, the cross-lagged panels of efficacy and total coping frequency did not provide a consistent pattern. Reasons for this have been alluded to previously. One problem with correlating coping frequency over time with efficacy was that patients were presumably becoming better copers, but not necessarily copers who used more coping strategies. That is, they may have been using fewer strategies, but in a more effective manner. Overall, the results argued that both self-efficacy level and adaptive coping improved over time. However, the relationship between these two variables was not necessarily a straightforward one. One hypothesis is that as patients became more efficacious, their experience of panic was not as traumatic and thus utilization of coping strategies could decrease. Alternately, patients may have developed such good coping abilities that they became more efficacious and less symptomatic. A more refined measure of coping, perhaps in a more naturalistic type of study, could address this issue.

The relationship between self-efficacy and symptom level was also not as clear as that between self-efficacy and catastrophic thoughts. Inspection of the visual representation of the relationship did suggest the same type of relationship as observed with the thoughts. That is, a negative relationship was seen where higher levels of efficacy were related to lower levels of symptoms. However, several interesting findings emerged with the cross-lagged panels. First, level of symptoms was not consistently related over time. A high level of symptoms at one time period did not necessarily imply a high level at another. The relationship over time showed a great deal of variability. This may reflect the observed waxing and waning pattern of panic (DSM-III-R, 1987). A second interesting finding was that the relationship of self-efficacy and

symptom level was not consistently high. At times, these variables were not even contemporaneously related. They were also not always strongly related in the lagged panels. In fact, when present, their relationship appeared to be more mutual than causal in one direction or the other. This was particularly interesting given the strong and consistent relationship between efficacy and catastrophic thoughts.

Several issues may account for this difference. Self-efficacy and catastrophic thoughts are both types of thoughts. One might expect a more consonant relationship between variables of similar modality than between variables of two different modalities. Response fractionation is a well established finding in the anxiety literature (e.g. Lang, 1977). Beyond this difference however, one hypothesis is that physiological sensations may not be eradicated by treatment or by increasing one's confidence in the management of symptoms. This relationship between sensations and confidence may be more obscure. Collectively, the results of this study suggested that symptoms did decrease in intensity. Findings from the induction studies also showed a decrement in physical sensations (e.g. Guttmcher & Nelles, 1984). Yet, other data suggested that physical sensations were not eliminated and were present in many people who did not truly have panic attacks (Norton et al., 1984). The combined results of the current and prior investigations is that elimination of symptoms is not necessary for improvement. The physical sensations can be reduced and that may be an important treatment element. However, elimination may not be a reasonable goal. What then accounts for the difference in subjects over the course of treatment? One factor is obviously that the intensity of their symptoms decreased. Another factor may be that they learned to alter the interpretation of what they continued to experience. As such, the relationship between self-efficacy and symptoms became convoluted.

Increased efficacy apparently led to less catastrophic interpretations which may have led to less intensely experienced symptoms. This is similar to what Hibbert's (1984) data suggests. A viable way to better address this issue would be to induce panic and utilize some type of thought listing procedure during the induction process. This way, reports of thoughts and symptoms are not merely retrospective given the likely biases inherent in any retrospective questioning about the experience of panic. This type of approach would allow a more refined analysis of the relationship of panic thoughts and symptoms.

A final variable examined in relation to self-efficacy was level of avoidance. As discussed above, avoidance decreased over time, particularly within the GIC group. However, the cross-lagged panels showed that the level of avoidance remained consistent over time. Together, these results may suggest that if a subject began treatment as an avoider, his or her level of avoidance may decrease, but he or she may remain relatively more avoidant than a subject who does not start as an "avoider." This points to the possibility that there is a "type" of person who is an avoider. Avoidance has been traditionally characterized as a secondary characteristic of panic (e.g. Foa, Steketee, & Young, 1984; Klein, Ross, & Cohen, 1987; Sanderson & Barlow, 1986). Presumably then, when the panic ended, so would the avoidance. Although preliminary, the results of the current study could be interpreted somewhat differently. That is, that there may be some people who are characteristically avoidant, perhaps as a function of some personality characteristic. Although DSM-III-R subsumes Agoraphobia with panic attacks under Panic Disorder, these avoiders may continue to represent a subsample whose treatment needs remain somewhat different.

Related directly to the current investigation, the relationship of self-efficacy and avoidance

began as negative and became positive. The meaning of this was unclear. A negative relationship is conceptually reasonable. The higher the self-efficacy, the lower the amount of avoidance. The more confident a person becomes in his or her abilities to cope with the experience of a panic attack, presumably the less need to avoid situations which were previously related to panic episodes or fear of panic episodes. However, the change from a negative to a positive relationship posed a problem of intepretation. The most direct interpretation would suggest that higher levels of self-efficacy were associated with higher levels of avoidance. That is, subjects may have felt efficacious because they were avoiding situations which induced anxiety. Yet, this interpretation is difficult to support given other data from this study which shows increases in self-efficacy and decreases in avoidance throughout treatment. Another interpretation is that self-efficacy and avoidance were simply not related. Intuitively, one might expect this relationship that as one becomes more confident, one no longer feels prone to avoid situations. However, the results of the current investigation did not provide any indication of a compelling relationship between the two. It may be that both were important in the successful treatment of panic yet they remained somehow orthogonal. Further explicit examination of the role of avoidance appears warrented.

In sum, did the concept of self-efficacy help explain the process of improvement in the treatment of panic attacks? Overall, the results pointed to a clear pattern when examining efficacy and catastrophic thoughts. Self-efficacy appeared to mediate, in Bandura's (1977) term, the changes that occurred in catastrophic thoughts over time. Likely when one becomes increasingly confident, he or she has less reason to catastrophize the sensations that are experienced from time to time. The patient apparently learns to better evaluate what is

happening physiologically. Through this process, catastrophic interpretations are decreased and perhaps the sensations do not further escalate. The relationship between efficacy and symptoms was not as clear. This relationship appeared more mutually causal rather than unidirectional. It appears that decreased symptoms may allow a person to become more efficacious while feeling more efficacious may result in experiencing less intense symptoms. Possibly this relationship was not as clear because individuals will likely continue to experience the somatic sensations of an attack while they probably will not continue to consistently catastrophize these sensations. Prior research shows that these sensations are present, even in the normal population (e.g. Norton et al., 1985; Rapee, Ancis, & Barlow, 1986). That leaves the question, are these sensations a panic attack? Normal subjects would likely not classify an accelerated heart rate and sweating as a panic attack. What may distinguish sensations from panic per se is the interpretation of the sensations. That is, without catastrophic interpretations, are physical sensations a panic attack? Perhaps not. The most consistently shown and compelling role of self-efficacy is to change these catastrophic interpretations.

In conclusion, Guided Imaginal Coping appeared to be a viable treatment for panic. Results revealed improvement in total symptoms experienced, total number of catastrophic thoughts, total number of panic attaacks, total level of avoidance, and rated ability to cope. These results were consistent with and extend preliminary results of the efficacy of the treatment approach (e.g. Borden et al., 1986). However, when comparing the treatment over time with a reasonable control group, the results did not indicate that GIC is significantly better than this other treatment. Several individual variables provided examples of where GIC subjects showed significant changes over time while PE subjects did not change over time. For example,

avoidance levels changed over time with GIC and patients continued to show improvement in the number of symptoms they experienced during follow-up periods. Yet, overall, most dependent measures revealed significant effects of time and not group. Both treatments appeared to work fairly well. Did they work better than nothing? Likely they do given prior results with a no treatment group (Borden et al., 1986). The addition of a no treatment group might provide a clearer answer. What emerges from these results, however, is that the problem of panic may not be as refractory as thought. A nondirective treatment where patients are encouraged to explore the antecedents of their attacks is basically as effective as a specific cognitive-behavioral intervention. This is counter to the notion that specific cognitive-behavioral strategies are the essential ingredients in an effective treatment of the disorder. A treatment designed specifically to eliminate these elements clearly worked and resulted in reduced problems with panic.

The second major question of the present investigation concerned the influence of self-efficacy. Consistent with prior research (Beck & Emery, 1985; Hibbert, 1984; Last et al., 1985) cognitions appeared an important component of panic attacks. Self-efficacy appeared to provide a framework for understanding what happened with catastrophic thoughts over the course of treatment. The relationship between self-efficacy and symptoms of panic attacks requires further examination. It appears interactive. Combining this with the relationship of efficacy and thoughts, a clearer understanding of the conceptual model of panic is permitted. Symptoms occur for whatever reason. Individuals may feel incapable of managing these sensations. They then appear to catastrophize, tending to make a feedback loop of increasingly catastrophic thoughts and increasing physical sensations, leading to what might be termed a

panic attack. This is consistent with previously presented models (e.g. Clark, 1986; Clum & Pickett, 1984). The present investigation suggets that one means to intervene in this process is to increase self-efficacy.

Further refinement of this model can be addressed in future research. The explicit impact of thoughts upon physical sensations needs to be further explored. The suggested thought listing procedure may be one way to accomplish this. Other refinements include an increased sample size yielding more power for statistical analyses and the possible inclusion of a no treatment control group. A further refinement would be the monitoring of physiological response during an induction procedure. Prior research has examined this important component in a limited fashion. Evaluations over the course of treatment appear to provide sufficiently more information to justify their continued use in empirical investigations. Extended examination of physiological responses and thought processes appear to be the next step in continuing to investigate the components of panic and mechanisms of treatment efficacy of Panic Disorder.

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APPENDIX A

Conceptual Model of Panic Attacks

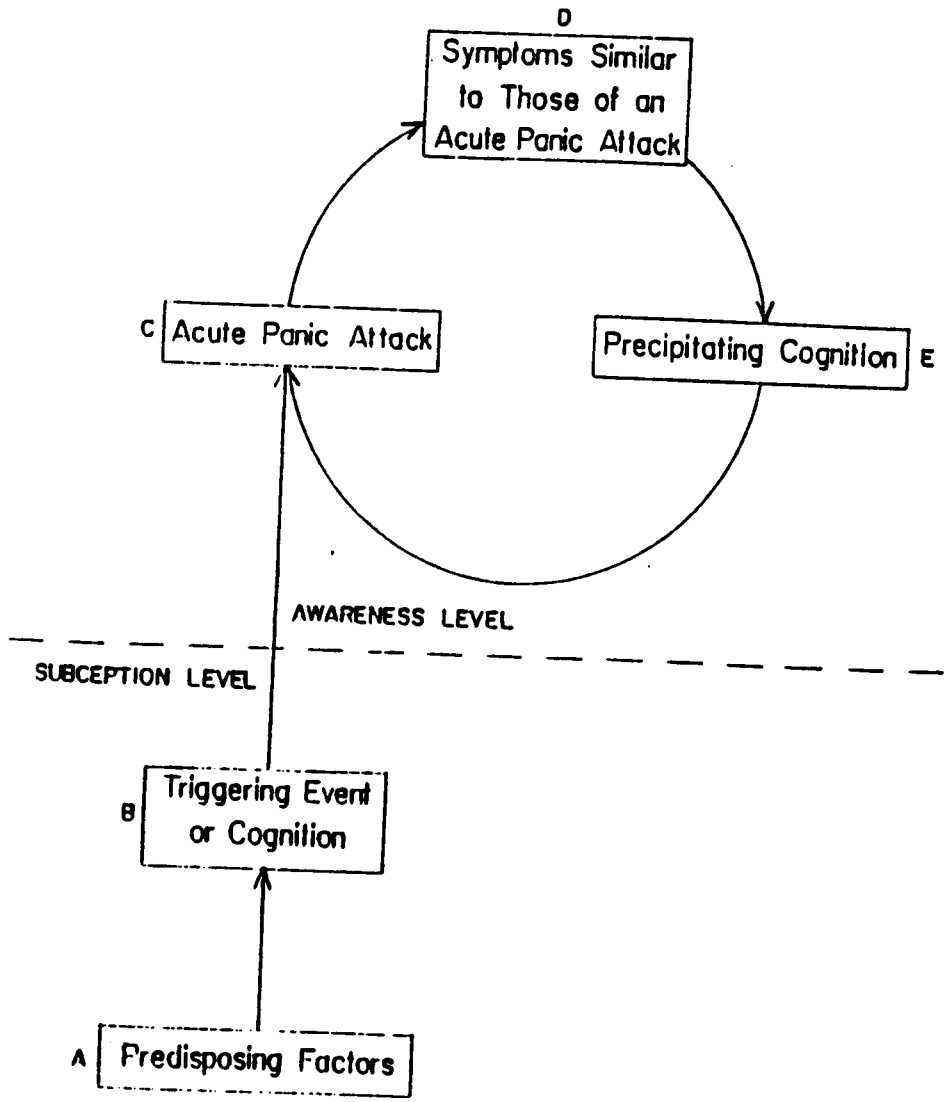

Figure 1

Conceptual Model of Panic Attacks

APPENDIX B

Treatment Protocol for "Panic Education"

Treatment Protocol for Panic Education

Part I: Group Sessions

1. Purpose of Treatment

Subjects will be told that the purpose of treatment is to discover why they have panic attacks, meaning why the attacks began and why they continue to happen. This will be done in both an historical and immediate perspective by uncovering stressors and conflicts within the individual subject's life.

2. Rationale

Subjects will be told that most people do not understand why they have panic attacks. Instead, the panic seems to come from "out of the blue." Relying on the literature demonstrating a relationship between stressors and the onset of panic attacks, subjects will be told that understanding their conflicts and stressors will make the attacks more understandable and also more manageable.

3. General Goals of Panic Education

Panic Education is based on 5 goals:

(1) to instill the notion of universality - that is that the subjects are not alone in the experience of their problem

(2) to instill the idea of hope - by examining general improvement rates in subjects with panic attacks and by focusing upon the improvement of group members as treatment progresses

(3) to instill a group norm of acceptance - by emphasizing the right of each member to say whatever they desire while stressing the difference between accepting a person and accepting what they say

(4) to provide an opportunity for each member to uncover the origins of their panic attacks from an historical perspective

(5) to examine the relationship between current stressors and attacks

4. Conduct of Sessions

All sessions will be conducted in a nondirective manner where the therapist provides no explicit instruction in effective coping. The first session will include a self-introduction by each group member in whatever manner is comfortable for them. In addition, subjects will be asked to share why they are participating in treatment and what they hope to gain from it. The purpose and rationale for treatment will be discussed along with supporting research literature. The conduct of sessions will also be suggested. This will include the process of examining current stressors and attacks as well as taking an historical perspective about the origins of panic. During this first session, the therapist will emphasize that the group members are largely in charge of the group and the directions it will take. The therapist will be identified as someone to facilitate the process based upon the knowledge of what would be effective. However, group members will be asked to evaluate each group and to provide feedback for needed changes. The therapist will emphasize their responsibility in making these changes. Sessions 2 through 4 will follow a similar format, but may be altered by group members.

Part II: Individual Sessions

1. Rationale

Subjects will be given the same rationale as they receive in the group setting. The possibility for more intensive examination of the origins of their panic will be highlighted to encourage exploration during individual sessions.

2. Conduct of Sessions

All 6 sessions will utilize a similar nondirective format where the therapist facilitates self-exploration on the part of the client rather than providing any specific instructions in coping or managing panic. Each session will begin with a discussion of the previous week including stressors and life events. In addition, subjects will be asked if they had a panic attack during the week and if so to describe the details of it. They will be asked to explore possible causes for the attack and to consider what aspects about the situation may have led to an attack. In addition, an historical focus will be taken to examine the first attack. This will involve the events surrounding the attack, particular stressors, and any other information the subject can recall. This will be treated as cues for why the attacks occur. The progression of panic over time will also be explored with the same rationale.

APPENDIX C

Treatment Credibility Checks

Rating of Treatment Rationale - Pretreatment

A technique designed to treat your panic attacks and anxiety was just explained to you. We are interested in obtaining your impressions about the treatment procedure at this time. Please answer each question below as honestly and openly as possible. Circle the number which best reflects your feelings right now.

1. How logical does this type of treatment seem to you for helping people alleviate their problems with panic attacks?

1	2	3	4	5	6	7	8	9
not at all logical								very logical

2. How confident are you that this treatment will be successful in reducing your panic attacks?

1	2	3	4	5	6	7	8	9
not at all confident								very confident

3. How confident would you be in recommending this treatment to a friend with panic attacks?

1	2	3	4	5	6	7	8	9
not at all confident								very confident

4. How important do you think it is that we make this treatment available to others who experience panic attacks?

1	2	3	4	5	6	7	8	9
not at all important								very important

5. How successful do you believe this treatment would be in decreasing other problems like general anxiety, depression, etc.

1	2	3	4	5	6	7	8	9
not at all likely								very likely

6. Please rate the level of mastery of the treatment skills you believe you will obtain by the end of the last treatment session

1	2	3	4	5	6	7	8	9
no mastery at all								great mastery

Rating of Treatment Rationale - Midtreatment

A technique designed to treat your panic attacks and anxiety has been explained to you. We are interested in obtaining your impressions about the treatment procedure at this time. Please answer each question below as honestly and openly as possible. Circle the number which best reflects your feelings right now.

1. How logical does this type of treatment seem to you for helping people alleviate their problems with panic attacks?

1	2	3	4	5	6	7	8	9
not at all logical								very logical

2. How confident are you that this treatment will be successful in reducing your panic attacks?

1	2	3	4	5	6	7	8	9
not at all confident								very confident

3. How confident would you be in recommending this treatment to a friend with panic attacks?

1	2	3	4	5	6	7	8	9
not at all confident								very confident

4. How important do you think it is that we make this treatment available to others who experience panic attacks?

1	2	3	4	5	6	7	8	9
not at all important								very important

5. How successful do you believe this treatment would be in decreasing other problems like general anxiety, depression, etc.

1	2	3	4	5	6	7	8	9
not at all likely								very likely

6. Please rate the level of mastery of the treatment skills you believe you will obtain by the end of the last treatment session.

1	2	3	4	5	6	7	8	9
no mastery at all								great mastery

Rating of Treatment Rationale - Posttreatment

We are interested in obtaining your impressions about the treatment procedure you copmpleted for your panic attacks. Please answer each question below as honestly and openly as possible. Circle the number which best reflects your feelings right now.

1. How logical did this type of treatment seem to you for helping people alleviate their problems with panic attacks?

1	2	3	4	5	6	7	8	9
not at all logical								very logical

2. Please rate the overall level of mastery of the treatment skills you believe you obtained as a result of this treatment

1	2	3	4	5	6	7	8	9
no mastery at all								great mastery

3. How confident would you be in recommending this treatment to a friend with panic attacks?

1	2	3	4	5	6	7	8	9
not at all confident								very confident

4. How important do you think it is that we make this treatment available to others who experience panic attacks?

1	2	3	4	5	6	7	8	9
not at all important								very important

5. How likely is it that your problems with panic will become worse in the future?

1	2	3	4	5	6	7	8	9
not at all likely								very likely

6. How likely is it that your problems with panic attacks will diminish in the future (become less frequent, less severe, etc.)?

1	2	3	4	5	6	7	8	9
not at all likely								very likely

APPENDIX D

Sample Anxiety Induction Script

Instructions: In this evaluation, we want to measure your current progress in treatment. We will be asking you to imagine various parts of your anxiety attacks. It is important that you imagine these as clearly as possible. Do not try to avoid thinking about them. We will then be better able to assess your current progress. We will also be asking you what you do at various points in your attack. Again, please respond as honestly as possible. We have begun by placing your finger in the pulse meter. This is so that we may monitor your pulse throughout this evaluation. If you have no questions, we will begin. First, I want you to close your eyes and relax for a period of three minutes. Go ahead.

Situation: Imagine that it is a work day. You have gotten ready to go to work and now you are in the car driving toward Tech. Imagine driving your car as you get closer and closer to the campus. Imagine that there are a number of cars around you. Traffic is heavier than usual. It seems like there are a lot of cars on the road this morning. Imagine this situation as you continue to drive toward the campus.

Initial Symptoms: As you drive toward work, you begin to have trouble breathing. You feel like you have to force yourself to breathe. You notice that it is becoming more and more difficult to breathe. You begin to feel a little weak from all of your efforts to breathe. Your chest starts to feel sore from this effort. Imagine these initial sensations as they occur while you are driving to work.

Initial Cognitions: As these initial symptoms begin, you start to think that something is very wrong with you. You are afraid that you are going to hyperventilate. You think that people i the other cars can see what is happening to you. You imagine that they think you are crazy. They also think about how you are driving. They are getting aggravated with you and think that something must be wrong with you. Imagine these initial thoughts as they run through your mind.

Intensified Symptoms: Imagine that you are turning into the Tech campus. There are a lot of cars and a lot of people around you. You feel your breathing getting worse and worse. You begin to feel hot and a little sweaty. You start to feel tingly and very unsteady. You feel like you can't get a deep breath - like you are just unable to breathe. Imagine all of these symptoms occurring now.

Intensified Cognitions: As all of these symptoms occur, you become more and more upset. You are afraid that you won't find a parking place. You feel like people around you must think that you are crazy. You start to wonder if you are crazy. You begin to think that something is terribly wrong with you. You think that this may never end.

Worst Symptoms: Now imagine that the symptoms have gotten even worse. You are at work, but you feel as if you are hyperventilating. You feel weak and your chest is sore from all of your efforts to breathe. You are as hot as you've ever been. You feel sweaty and extremely unsteady. You begin to feel like you might even pass out. Every breath is so difficult and you don't feel like you are getting nearly enough air to breathe.

Worst Cognitions: As all of this happens, you become extremely upset. You are afraid that something is very wrong with you. You feel like maybe you are crazy. You think that it willalways be this way - that you will never be any better. You feel like you will always hyperventilate. You begin to think about dying. You feel so overwhelmed by all of these episodes that you don't know what to do anymore.

Fifteen Minute Attack: Imagine that this attack continues. You are still experiencing those intense symptoms of not being able to breathe and those panicky thoughts. These seem to last forever. The attack feels like a full-blown one - one of the worst you've ever had. It's already lasted several minutes and it does not appear to be ending.

Thirty Minute Attack: Imagine that all of your problems continue. You feel as if you are hyperventilating. You are sore in the chest. You feel hot and very weak. You feel faint. You think that you must be crazy; that you will always be this way. You think about dying and putting an end to all of this pain. Imagine how you feel as this episode goes on and on.

Several Hour Attack: Imagine that the attack has now lasted for several hours. Unlike your prior attacks, this one continues without signs of ending. You've never experienced anything this intense and this scary before. You feel your symptoms at their peak level. You are as frightened as you've ever been. You feel completely out of control. Imagine these feelings as they go on and on.

Twenty Four Hour Attack: Now imagine that the attack has lasted late into the night. No matter what you do, the attack continues. Nothing you do seems to make any difference. You are feeling completely exhausted and drained by the length and intensity of this attack. Imagine your feelings now that this attack has lasted into the night and into the next day.

ADDENDIX E

Training Manual For Coping

Training Manual for Coping

Part 1: Judging Coping Efficacy

Coping strategies are considered adaptive or efficacious if they:

(1) Do not result in avoidance of the stressful situation.

(2) Reduce anxiety-arousing cognitions (e.g. I must be having a heart attack; I'm going crazy etc.)

(3) Reduce second order somatic symptoms (i.e. those that develop after initial somatic response and cognitions)

(4) Allow individual to deal with first order somatic symptoms

(5) Lead to mastery and self-efficacy

(6) Reduce fear

These 6 categories are not mutally exclusive nor or they ranked in order of importance. The guiding criteria is that the strategy is one which will reduce anxiety and panic in a tripartite model - that is decreased symptoms, catastrophic thoughts, and avoidance - over the short and long term.

Maladaptive strategies include those which promote avoidance or objectively judged potential harm to the individual or others. For example:

1. Avoidance - this may take a variety of forms including*
   leaving the situation
   avoiding the situation totally
   avoiding thinking about the situation

2. Self-injurious behavior
   e.g. slashing wrists, inflicting pain

3. Dangerous behaviors toward others
   e.g. striking someone, inflicting pain upon someone

* All of these behaviors may be avoidant. However, the subject's motive may need to be considered at times with strategies that appear avoidant. Distraction can often be confused with avoidance. The subject's willingness to return to the situation (or alternatively to think about it) can distinguish avoidance and distraction. For example, consider a subject having a panic attack while driving a car. The subject pulls to the side of the road. Avoidance or distraction?

Base decision on the subject's motive and intention. Avoidance - the subject says she stopped and cannot go any farther. She believes if she drives again, the consequences will be catastrophic.

Distraction - the subject says that she needed a moment to pull herself together. She feels that she will then be able to return to driving.

Effective Coping Strategies include any of the following:

1. Distraction
   a. Behavioral - engaging in some activity to refocus attention away from panic attack.
      - examples:
         work; turning on television, radio; exercise;
         relaxation - unstructured or structured (includes yoga,
         focused attention, muscle tension and relaxation)
   b. Cognitive - thinking about something other than panic attack
      - examples:
         thinking about surroundings, future activities

2. Information Increasing - anything which increases the subject's knowledge about their panic attack.
   - examples:
      noticing that people are not paying attention to them as they have feared; differentiating actual symptoms from their perceptions of symptoms (e.g. rapid heart rate versus fears of what that indicates - will be phrased as something like"my heart is not really going to explode, it's just beating a little faster")

3. Cognitive Reassuring - self-talk to compete with negative cognitions
   - examples:
      "this will pass"
      "I'm breathing fast, but I know it's going to be o.k."
      reasoning out what is happening to them

4. Directly Confronting Symptoms - performing an activity which is intended to directly impact upon the symptoms which the subject is experiencing.
   - examples:
      controlled breathing, diaphragmatic breathing, vagus nerve stimulation

Part 2: Experimental Procedure

Subjects will complete 5 questionnaires at each assessment: an avoidance questionnaire, a panic symptoms questionnaire, a catastrophic thoughts questionnaire, a coping questionnaire, and a self-efficacy questionnaire. These are to be given at the beginning of the evaluation in a random order.

Each subject will be evaluated at 10 points - pre-treatment, the 6 weeks of treatment, post-treatment, 1 month follow-up, and 2 month follow-up. For each evaluation, play the tape designed for the particular subject. The tape will consist of instructions and 11 distinct steps of a panic attack. At each of the steps, the evaluator will ask the subject what s/he does at that point. The subject's response is to be rated as coping or not coping adaptively based upon the above criteria. It is not necessary to indicate which type of strategy is being used, only whether it represents adaptive coping. The subject's verbatim responses should be written down for the evaluator's coding purposes. In addition, all evaluation sessions are to be audiotaped for reliability checks. Place only the subject number on tapes and coding sheets.

APPENDIX F

Revised Betts' Questionnaire Upon Mental Imagery

Vividness of Imagery

Instructions: The aim of this inquiry is to determine the vividness of your imagery. The items of the test will bring certain images to your mind. You are to rate the vividness of each image by reference to the accompaning rating scale. For example, if your image is "vague and dim" you give it a rating of 5. Record your answer in the appropriate space next to the question. Before you begin, familiarize yourself with the different categories of the rating scale. Throughout the test, refer to the rating scale when judging the vividness of each image. Try to make each rating on its own merits without reference to what has gone before.

Rating Scale: The image aroused by any one item of this test may be:

Rating to be given	Nature of the image
1	Perfectly clear and as vivid as the actual experience
2	Very clear and comparable in vividness to the actual experience
3	Moderately clear and vivid
4	Not clear or vivid but recognizable
5	Vague and dim
6	So vague and dim as to be hardly discernible
7	No image present at all; just knowing that you are thinking of the object

---

Your rating of pictorial (visual) images

1. The sun as it is sinking below the horizon _____

Think of some relative or friend. Rate the vividness of the following images:

2. The exact contour of the face, head, shoulders, and body _____
3. The characteristic poses of the head, attitudes of the body, etc. _____
4. The precise carriage, length of step, etc. in walking _____
5. The different colors worn in some familiar costume _____

Your rating of sound (auditory) images

6. The sound of the whistle of a locomotive _____
7. The sound of the honk of an automobile _____
8. The mewing of a cat _____
9. The sound of escaping steam _____
10. The clapping of hands in applause _____

Your rating of touch (tactual) images

11. The feel of sand _____
12. Of linen _____
13. Of fur _____
14. The prick of a pin _____
15. The warmth of a tepid bath

Your rating of muscular (kinesthetic) images

16. Running upstairs _____
17. Springing across a gutter _____
18. Drawing a circle on paper _____
19. Reaching up to a high shelf _____
20. Kicking something out of your way _____

Your rating of taste (gustatory) images

21. The taste of salt _____
22. Of white sugar _____
23. Of oranges _____
24. Of jelly _____
25. Of your favorite soup _____

Your rating of smell (olfactory) images

26. The smell of an ill-ventilated room _____
27. Of cooking cabbage _____
28. Of roast beef _____
29. Of fresh paint _____
30. Of new leather _____

Your rating of bodily (somesthetic-organic) images

31. Sensations of fatigue _____
32. Of hunger _____
33. Of a sore throat _____
34. Of drowsiness _____
35. Of repletion (as from a very full meal) _____

APPENDIX G

Self-Efficacy Questionnaire

Coping with Attacks

Instructions: Think of how you feel right at this moment. Please indicate which of the following parts of a panic attack you believe you could cope with by employing the coping strategies you currently use. Do this by circling either yes or no for each question. Then, after each question rate how confident you are that you could cope by circling one number from 1 to 9.

1. Being in a situation where you've had an attack yes no

1 2 3 4 5 6 7 8 9
not at all confident — moderately confident — totally confident

2. When you first notice the symptoms of an attack yes no

1 2 3 4 5 6 7 8 9
not at all confident — moderately confident — totally confident

3. Thoughts that come into your mind when you experience symptoms, like thinking you are having a heart attack, dying, etc. yes no

1 2 3 4 5 6 7 8 9
not at all confident — moderately confident — totally confident

4. Intense symptoms that continue to worsen and intensify yes no

1 2 3 4 5 6 7 8 9
not at all confident — moderately confident — totally confident

5. Scary and intense thoughts that continue to occupy your mind yes no

1 2 3 4 5 6 7 8 9
not at all confident — moderately confident — totally confident

6. Symptoms as strong as they have ever been for you yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

7. Thoughts as intense, scary, and real as they've ever been for you yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

8. A full-blown attack that lasts 15 minutes yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

9. A full-blown attack that lasts 30 minutes yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

10. A full-blown attack that lasts several hours yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

11. A full-blown attack that lasts all day which seems like it will not subside yes no

1	2	3	4	5	6	7	8	9
not at all confident				moderately confident				totally confident

APPENDIX H

Avoidance Questionnaire

Avoidance Questionnaire

Instructions: People with panic and anxiety often find it difficult to enter specific situations. Listed below are a number of commonly avoided situations. Please rate each situation as to how often you currently avoid it due to your panic or anxiety using the following scale:

1 = no avoidance or escape
2 = occasional avoidance or escape
3 = moderate: may enter alone
4 = severe: rarely alone, must be accompanied
5 = very severe: never enter even with a "safe person"

1. Driving _____
2. Riding in car _____
3. Grocery store _____
4. Mall _____
5. Crowds _____
6. Bus _____
7. Airplane _____
8. Taxicab _____
9. Waiting in line _____
10. Walking outside your home _____
11. Elevators _____
12. Being at home _____
13. Movie theaters _____
14. Restaurants _____
15. Theaters _____
16. Auditoriums _____
17. Church _____
18. Tunnels _____
19. Small rooms _____
20. Parks _____
21. Work _____

Please list and rate any others not listed above

APPENDIX I

Catastrophic Thoughts Questionnaires

Panic Attack Thoughts Questionnaire - Weekly

Instructions: Frightening thoughts often accompany, precede, or follow panic attacks or other episodes of extreme anxiety. Think of the past week. Using the scale below, rate how much each of the following thoughts is currently likely to preoccupy you before, during, and after a panic attack or other episode of extreme anxiety. Remember to rate each thought in terms of its occurrence before, during, and after episodes of anxiety this week. If you have not had an anxiety episode this week, you would rate each of the thoughts "1."

1 = not at all
2 = some, but not much
3 = quite a lot
4 = totally dominates your thoughts

---

		before	during	after
1. I am going to die	1 2 3 4	____	____	____
2. I am going insane	1 2 3 4	____	____	____
3. I am losing control	1 2 3 4	____	____	____
4. This will never end	1 2 3 4	____	____	____
5. I am really scared	1 2 3 4	____	____	____
6. I am having a heart attack	1 2 3 4	____	____	____
7. I am going to pass out	1 2 3 4	____	____	____
8. I don't know what people will think	1 2 3 4	____	____	____
9. I won't be able to get out of here	1 2 3 4	____	____	____
10. I don't understand what is happening to me	1 2 3 4	____	____	____
11. People will think I am crazy	1 2 3 4	____	____	____
12. I will always be this way	1 2 3 4	____	____	____
13. I am going to throw up	1 2 3 4	____	____	____

(Continue to rate each thought with the same scale)

		before	during	after
14. I must have a brain tumor	1 2 3 4	____	____	____
15. I will choke to death	1 2 3 4	____	____	____
16. I am going to act foolish	1 2 3 4	____	____	____
17. I am going blind	1 2 3 4	____	____	____
18. I will hurt someone	1 2 3 4	____	____	____
19. I am going to have a stroke	1 2 3 4	____	____	____
20. I am going to scream	1 2 3 4	____	____	____
21. I am going to babble or talk funny	1 2 3 4	____	____	____
22. I will be paralyzed by fear	1 2 3 4	____	____	____
23. Something is really physically wrong with me	1 2 3 4	____	____	____
24. I will not be able to breathe	1 2 3 4	____	____	____
25. Something terrible will happen	1 2 3 4	____	____	____

Panic Attack Thoughts Questionnaire - Follow-up

Instructions: Frightening thoughts often accompany, precede, or follow panic attacks or other episodes of extreme anxiety. Think of the past month. Using the scale below, rate how much each of the following thoughts is currently likely to preoccupy you before, during, and after a panic attack or other episode of extreme anxiety. Remember to rate each thought in terms of its occurrence before, during, and after episodes of anxiety this week. If you have not had an anxiety episode this month, you would rate each of the thoughts "1."

1 = not at all
2 = some, but not much
3 = quite a lot
4 = totally dominates your thoughts

---

		before	during	after
1. I am going to die	1 2 3 4	____	____	____
2. I am going insane	1 2 3 4	____	____	____
3. I am losing control	1 2 3 4	____	____	____
4. This will never end	1 2 3 4	____	____	____
5. I am really scared	1 2 3 4	____	____	____
6. I am having a heart attack	1 2 3 4	____	____	____
7. I am going to pass out	1 2 3 4	____	____	____
8. I don't know what people will think	1 2 3 4	____	____	____
9. I won't be able to get out of here	1 2 3 4	____	____	____
10. I don't understand what is happening to me	1 2 3 4	____	____	____
11. People will think I am crazy	1 2 3 4	____	____	____
12. I will always be this way	1 2 3 4	____	____	____
13. I am going to throw up	1 2 3 4	____	____	____

(Continue to rate each thought with the same scale)

		before	during	after
14. I must have a brain tumor	1 2 3 4	____	____	____
15. I will choke to death	1 2 3 4	____	____	____
16. I am going to act foolish	1 2 3 4	____	____	____
17. I am going blind	1 2 3 4	____	____	____
18. I will hurt someone	1 2 3 4	____	____	____
19. I am going to have a stroke	1 2 3 4	____	____	____
20. I am going to scream	1 2 3 4	____	____	____
21. I am going to babble or talk funny	1 2 3 4	____	____	____
22. I will be paralyzed by fear	1 2 3 4	____	____	____
23. Something is really physically wrong with me	1 2 3 4	____	____	____
24. I will not be able to breathe	1 2 3 4	____	____	____
25. Something terrible will happen	1 2 3 4	____	____	____

APPENDIX J

Panic Attack Symptom Questionnaires

Panic Attack Symptom Questionnaire - Weekly

Instructions: The symptoms listed below are frequently experienced during anxiety and panic attacks. Base your responses only on the past week. If you have had panic attacks, rate your worst one. If you have not had an attack, rate the period when you were most anxious during the week. Please use the following scale and rate each item where:

1 = Did Not Experience This
2 = Fleeting (lasted 1 second to 1 minute)
3 = Briefly (lasted 1 minute to 10 minutes)
4 = Moderately (lasted 10 minutes to 1 hour)
5 = Quite Long (lasted 1 hour to 1 day)
6 = Protracted Period (lasted 1 day or longer)

(remember to rate each item by circling only one number per item)

---

1. Heart beats rapidly or pounds	1	2	3	4	5	6
2. Pain in chest	1	2	3	4	5	6
3. Heart pounding in chest	1	2	3	4	5	6
4. Difficulty swallowing (lump in throat)	1	2	3	4	5	6
5. Feeling of suffocation	1	2	3	4	5	6
6. Choking sensation	1	2	3	4	5	6
7. Hands or feet tingle	1	2	3	4	5	6
8. Face feels hot	1	2	3	4	5	6
9. Sweating	1	2	3	4	5	6
10. Trembling or shaking	1	2	3	4	5	6
11. Hands or body trembling or shaking	1	2	3	4	5	6
12. Hands or feet feel numb	1	2	3	4	5	6
13. Feeling that you are not really you or that you are disconnected from your body	1	2	3	4	5	6

Continue to rate each symptom with the same scale where:
1 = Did not experience this
2 = Fleeting (1 second to 1 minute)
3 = Briefly (1 minute to 10 minutes)
4 = Moderately (10 minutes to 1 hour)
5 = Quite Long (1 hour to 1 Day)
6 = Protracted Period (1 day to 2 days)

---

14. Feeling that things around you are unreal - as if in a dream	1	2	3	4	5	6
15. Vomiting (not induced)	1	2	3	4	5	6
16. Nausea	1	2	3	4	5	6
17. Breathing rapidly (unable to catch breath)	1	2	3	4	5	6
18. Hands or feet feel cold	1	2	3	4	5	6
19. Mouth feels dry	1	2	3	4	5	6
20. Sinking feeling in stomach	1	2	3	4	5	6
21. Nerves feel "wired"	1	2	3	4	5	6
22. Physically immobilized	1	2	3	4	5	6
23. Vision becomes blurred or distorted	1	2	3	4	5	6
24. Pressure in chest	1	2	3	4	5	6
25. Numbness in body other than hands or feet	1	2	3	4	5	6
26. Shortness of breath	1	2	3	4	5	6
27. Dizziness	1	2	3	4	5	6
28. Feeling faint	1	2	3	4	5	6
29. Butterflies in stomach	1	2	3	4	5	6
30. Knot in stomach	1	2	3	4	5	6

(Continue to rate your symptoms with the same scale)

31. Tightness in chest	1	2	3	4	5	6
32. Wobbly or rubber legs	1	2	3	4	5	6
33. Disorientation or confusion	1	2	3	4	5	6
34. Cold clamminess	1	2	3	4	5	6
35. Sensitivity to loud noises or ears ringing	1	2	3	4	5	6
36. Ears ringing	1	2	3	4	5	6

Panic attacks are defined by having 4 or more of the above symptoms and also by having the attacks come on suddenly. Based on this criteria,

Did you have a panic attack this week? 1 yes 2 no

If yes, how many attacks did you have? (circle only one number)
1 2 3 4 5 6 7 8 9 10 or more

Panic Attack Symptom Questionnaire - Follow-up

Instructions: The symptoms listed below are frequently experienced during anxiety and panic attacks. Base your responses only on the past month. If you have had panic attacks, rate your worst one. If you have not had an attack, rate the period when you were most anxious during the month. Please use the following scale and rate each item where:

1 = Did Not Experience This
2 = Fleeting (lasted 1second to 1 minute)
3 = Briefly (lasted 1 minute to 10 minutes)
4 = Moderately (lasted 10 minutes to 1 hour)
5 = Quite Long (lasted 1 hour to 1 day)
6 = Protracted Period (lasted 1 day or longer)

(remember to rate each item by circling only one number per item)

---

1. Heart beats rapidly or pounds	1	2	3	4	5	6
2. Pain in chest	1	2	3	4	5	6
3. Heart pounding in chest	1	2	3	4	5	6
4. Difficulty swallowing (lump in throat)	1	2	3	4	5	6
5. Feeling of suffocation	1	2	3	4	5	6
6. Choking sensation	1	2	3	4	5	6
7. Hands or feet tingle	1	2	3	4	5	6
8. Face feels hot	1	2	3	4	5	6
9. Sweating	1	2	3	4	5	6
10. Trembling or shaking	1	2	3	4	5	6
11. Hands or body trembling or shaking	1	2	3	4	5	6
12. Hands or feet feel numb	1	2	3	4	5	6
13. Feeling that you are not really you or that you are disconnected from your body	1	2	3	4	5	6

Continue to rate each symptom with the same scale where:
1 = Did not experience this
2 = Fleeting (1 second to 1 minute)
3 = Briefly (1 minute to 10 minutes)
4 = Moderately (10 minutes to 1 hour)
5 = Quite Long (1 hour to 1 Day)
6 = Protracted Period (1 day to 2 days)

---

14. Feeling that things around you are unreal - as if in a dream	1 2 3 4 5 6
15. Vomiting (not induced)	1 2 3 4 5 6
16. Nausea	1 2 3 4 5 6
17. Breathing rapidly (unable to catch breath)	1 2 3 4 5 6
18. Hands or feet feel cold	1 2 3 4 5 6
19. Mouth feels dry	1 2 3 4 5 6
20. Sinking feeling in stomach	1 2 3 4 5 6
21. Nerves feel "wired"	1 2 3 4 5 6
22. Physically immobilized	1 2 3 4 5 6
23. Vision becomes blurred or distorted	1 2 3 4 5 6
24. Pressure in chest	1 2 3 4 5 6
25. Numbness in body other than hands or feet	1 2 3 4 5 6
26. Shortness of breath	1 2 3 4 5 6
27. Dizziness	1 2 3 4 5 6
28. Feeling faint	1 2 3 4 5 6
29. Butterflies in stomach	1 2 3 4 5 6
30. Knot in stomach	1 2 3 4 5 6

(Continue to rate your symptoms with the same scale)

31. Tightness in chest	1	2	3	4	5	6
32. Wobbly or rubber legs	1	2	3	4	5	6
33. Disorientation or confusion	1	2	3	4	5	6
34. Cold clamminess	1	2	3	4	5	6
35. Sensitivity to loud noises or ears ringing	1	2	3	4	5	6
36. Ears ringing	1	2	3	4	5	6

Panic attacks are defined by having 4 or more of the above symptoms and also by having the attacks come on suddenly. Based on this criteria,

Did you have a panic attack this month? 1 yes 2 no

If yes, how many attacks did you have? (circle only one number)
1 2 3 4 5 6 7 8 9 10 or more

APPENDIX K

Coping Strategies Questionnaires

Coping Strategies Questionnaire - Weekly

Instructions: For each of the strategies listed below, first indicate how often you have used the strategy in the past week. If you have had more than 1 attack this past week, rate your worst one. Next, indicate how effective each strategy was in relieving your anxiety symptoms and then your thoughts associated with the anxiety. Use the following scales:

FREQUENCY:
1 = not used
2 = rarely used
3 = sometimes used
4 = often used

SYMPTOM REDUCTION:
1 = not effective
2 = somewhat effective
3 = moderately effective
4 = totally effective

THOUGHT ELIMINATION:
1 = not effective
2 = somewhat effective
3 = moderately effective
4 = totally effective

Remember to rate each item using one number from each of the three scales.

---

	frequency	symptom reduction	thought elimination
1. I tell myself it will pass or it is nothing to worry about	____	____	____
2. I talk to a physician, psychologist, etc.	____	____	____
3. I talk to a friend or relative	____	____	____
4. I engage in structured relaxation	____	____	____
5. I exercise	____	____	____
6. I sit or lie down	____	____	____
7. I engage in some work activity	____	____	____
8. I try to reason out what is making me anxious	____	____	____
9. I try to distract myself mentally by thinking of something else (please specify _____)	____	____	____
10. I try to distract myself by doing some activity (please specify _____)	____	____	____

(Continue to rate each strategy using the same scale)

11. I take medication for anxiety (name_____) _____ _____ _____

12. I try to control my breathing _____ _____ _____

13. I let the symptoms happen rather than struggle with them _____ _____ _____

Coping Strategies Questionnaire - Follow-up

Instructions: For each of the strategies listed below, first indicate how often you have used the strategy in the past month. If you have had more than 1 attack this past month, rate your worst one. Next, indicate how effective each strategy was in relieving your anxiety symptoms and then your thoughts associated with the anxiety. Use the following scales:

FREQUENCY:
1 = not used
2 = rarely used
3 = sometimes used
4 = often used

SYMPTOM REDUCTION:
1 = not effective
2 = somewhat effective
3 = moderately effective
4 = totally effective

THOUGHT ELIMINATION:
1 = not effective
2 = somewhat effective
3 = moderately effective
4 = totally effective

Remember to rate each item using one number from each of the three scales.

---

	frequency	symptom reduction	thought elimination
1. I tell myself it will pass or it is nothing to worry about	____	____	____
2. I talk to a physician, psychologist, etc.	____	____	____
3. I talk to a friend or relative	____	____	____
4. I engage in structured relaxation	____	____	____
5. I exercise	____	____	____
6. I sit or lie down	____	____	____
7. I engage in some work activity	____	____	____
8. I try to reason out what is making me anxious	____	____	____
9. I try to distract myself mentally by thinking of something else (please specify _____ _____)	____	____	____
10. I try to distract myself by doing some activity (please specify _____)	____	____	____

(Continue to rate each strategy using the same scale)

11. I take medication for anxiety (name_____) _____ _____ _____

12. I try to control my breathing _____ _____ _____

13. I let the symptoms happen rather than struggle with them _____ _____ _____

APPENDIX L

Consent Forms

Consent Form

Assessment and Treatment of Panic Attacks

I, _____, freely and voluntarily consent to participate in a research program entitled "Assessment and Treatment of Panic Attacks" to be conducted by Janet W. Borden, M. S. and George A. Clum, Ph. D. The procedures to be followed have been explained to me and I understand them. They are as follows:

1. I understand that I will be interviewed by a graduate student in clinical psychology to: (a) determine if I have a panic disorder and am appropriate for treatment; and (b) determine underlying conflicts which are related to the onset of panic attacks. I will be asked to complete several questionnaires including a measures of my symptoms, thoughts, and anxiety. Assessment will take approximately one and one-half hours.

2. If I am approriate for treatment and desire treatment, I understand that I will be assigned to one of two treatment groups for a period of 6 weeks. The treatment group I will be assigned to will involve the identification and resolution of stresses and conflicts likely underlying the panic attacks. These stressors and/or conflicts will have been identified during the assessment procedure. Participants will be encouraged to share conflicts and possible solutions with other group members, although this will not be insisted upon. It will be possible to problem solve on these conflicts in a general rather than personal way. Some discomfort and anxiety may arise during this process.

3. I understand that I will also be evaluated on a weekly basis during treatment to determine if gains are being made. These evaluations will last approximately one half-hour and will involve completion of the same questionnaires as those I complete before beginning treatment. I understand that 1 month and 2 months after treatment I will be asked to come in for 2 final assessment sessions to determine if gains have been maintained.

4. I understand that all information obtained from me will be held strictly confidential by the treatment staff. Furthermore, in any scientific report of this project, there will be no way to identify me.

5. I understand that I may experience some discomfort when discussing conflict or stressful situations during the treatment sessions. However, it is anticipated that the overall effects of treatment, if completed will be beneficial.

6. I understand that I may withdraw my consent at any time without penalty. Further, I acknowledge that I have a duplicate signed copy of this form.

_____
Signature

_____ _____
Phone number Date

If you have any questions about this study, contact any of the following people:

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