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Testing a Brief Treatment to Reduce the Frequency of Panic Attacks in a Clinical Outpatient Population

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TESTING A BRIEF TREATMENT TO REDUCE THE FREQUENCY OF PANIC ATTACKS IN A CLINICAL OUTPATIENT POPULATION

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Dissertation Approval

This is to certify that the thesis presented to us by Benjamin Daniels

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Abstract

Panic attacks, the key symptom of panic disorder and an associated feature of various anxiety disorders, are extremely distressing events that can negatively impact an individual's mental health, physical health, and quality of life. This study validated a brief treatment for panic attacks, designed to reduce the frequency of panic attacks after the first session, in an outpatient clinical population. One participant was recruited to participate in this single case experimental ABA design with follow-up, where a reversal was not expected, due to the maintenance of positive effects. The treatment included both cognitive and behavioral techniques. The results were analyzed using simulation modeling analysis, as well as visual analysis. This treatment produced clinically significant effects by reducing the frequency and severity of panic attacks, reducing symptoms of anxiety and panic, decreasing the frequency of cognitive distortions, and increasing the level of functioning. Additionally, these gains were maintained at a 3-month follow-up. It is hoped that this intervention can help clinicians treat panic disorder and improve their effectiveness and efficiency by reducing the time needed to significantly decrease panic attacks. It is also hoped that this intervention might be expanded for use with other panic-related anxiety disorders. Finally, it is possible that this study will encourage efforts toward briefer treatments for other disorders.

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Introduction

Statement of the Problem

Many people in the United States meet criteria for panic disorder (American Psychiatric Association [APA], 2000). According to a national epidemiological study, the lifetime prevalence rates for panic disorder with and without agoraphobia are 1.1% and 3.7%, respectively. Moreover, 22.7% of the population will experience panic attacks without meeting the criteria for panic disorder. An additional 0.8% also meets criteria for agoraphobia without a history of panic attacks (Kessler et al., 2006). Furthermore, in a given year, 2.7% of the population suffers from panic disorder (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Taken together, it is apparent that over a quarter of the national population has suffered from panic symptoms (Kessler et al., 2006).

Many of those who suffer from panic disorder also may meet criteria for comorbid disorders. A national study found statistically significant comorbidity between panic disorder and many disorders, such as major depressive disorder, bipolar disorder, posttraumatic stress disorder (PTSD), social phobia, and generalized anxiety disorder, among others (APA, 2013; Kessler et al., 2005). Additionally, comorbidity of panic disorder, for example with depression, is associated with more severe and persistent symptoms (Roy-Byrne et al., 2000).

Panic attacks and panic disorder are both associated with increased suicidal ideation (Goodwin & Roy-Byrne, 2006). For example, Weissman, Klerman, Markowitz, and Ouellette (1989) found that 20% of those with panic disorder had attempted suicide. This risk for suicidality is further increased with the presence of comorbid depression (Roy-Byrne et al., 2000). Moreover, patients with panic disorder are found to have a

much higher incidence of medical conditions, such as irritable bowel syndrome, than non-panic disorder patients in a general medical setting (Kaplan, Masand, & Gupta, 1996). Panic attacks are also associated with increased alcohol and drug use, impaired perception of physical and emotional health, and poor social, occupational, marital, and financial functioning (Klerman, Weissman, Ouellette, Johnson, & Greenwald, 1991; Markowitz, Weissman, Ouellette, & Lish, 1989).

Despite the high prevalence of panic disorder, many individuals do not seek treatment. One study followed emergency department patients who were identified as having panic disorder over a 2-year period and found that only 22% had sought mental health treatment for their symptoms (Fleet, 2003). Generalizing those results, more than three quarters of those who suffer from panic disorder do not seek treatment.

Moreover, within the course of treatment, a large percentage of patients drop out before the treatment can be completed. The dropout rate from treatment for panic disorder ranges from 13% to 41%, with lower rates for behavior therapies and higher rates for pharmacological treatments (Clum, 1989; White et al., 2010). This means that of the small percentage of individuals who seek treatment, many will not be effectively treated. Not only does this mean that panic disorder is frequently untreated, but this treatment failure also may discourage these individuals from seeking treatment again in the future if they believe that such intervention will ultimately be ineffective. This would not be surprising, given the relationship seen between hopelessness and treatment avoidance and drop-out (Björk, Björck, Clinton, Sohlberg, & Norring, 2009; Salmoiraghi & Sambhi, 2010).

Those who meet criteria for panic attacks and panic disorder, as compared to those who do not, have higher rates of healthcare utilization, including ambulance services, hospital emergency departments, and other healthcare facilities (Katerndahl & Realini, 1997; Klerman et al., 1991; Markowitz et al., 1989). This may be due to the fact that the somatic symptoms of panic disorder are similar to symptoms of medical conditions. The distressing nature of panic symptoms leads many people to go to hospital emergency departments. For example, chest pain, which may be catastrophically misinterpreted as a heart attack, is a common symptom of panic attacks. One study found that approximately 20% of those who seek treatment for chest pain at emergency departments have panic disorder, as opposed to a medical condition. The study also found similar rates of panic disorder at outpatient cardiology clinics (Huffman & Pollack, 2003). Previous studies have found even higher rates of panic disorder in patients presenting with chest pain without a medical cause, ranging from 34% to 43% (Beitman et al., 1989; Katon et al., 1988).

Most of these patients do not seek treatment at mental health clinical settings. Instead, they often present to already overwhelmed emergency departments and clinics that may not have the resources to deal with these problems. Fortunately, cognitive behavioral therapy (CBT) for panic disorder can significantly reduce the unnecessary use of medical resources, both in terms of mental health care and overall medical healthcare utilization (Barlow, Gorman, Shear, & Woods, 2000; Klosko, Barlow, Tassinari, & Cerny, 1990; Roberge, 2005). This in turn reduces healthcare costs (Roberge, 2005).

While pharmacological treatments have also shown to be effective in treating panic disorder, cognitive behavioral therapies are preferred for their lower rates of

relapse, absence of side effects, and lower dropout rates (Beamish et al., 1996; Clum, 1989). A widely accepted CBT treatment for panic disorder was developed by Craske and Barlow (Craske & Barlow, 1993, 2007). Barlow proposed a cognitive behavioral treatment protocol lasting 12 sessions, consisting of components including psychoeducation about anxiety and the nature of panic, self-monitoring, developing a hierarchy of agoraphobic situations, breathing retraining (diaphragmatic breathing and autogenic training), cognitive restructuring, as well as graded in vivo and interoceptive exposure (Barlow, Craske, Cerny, & Klosko, 1989; Craske & Barlow, 2008). The protocol offered by Barlow reduced attrition rates to 17% in the original study, in comparison to rates of up to 41% in pharmacotherapy patients. However, this still means that almost one in five CBT patients terminated treatment prematurely (Barlow et al., 1989; Clum, 1989).

Consequently, even Barlow's 12-session treatment may be perceived as too long in duration for some patients, leading to dropouts, as well as incomplete and, consequently, ineffective treatment. Therefore, an effective treatment protocol of lesser duration may reduce attrition rates, offer faster relief, and be more cost effective in treating patients with panic disorder.

Purpose of the Study

The purpose of this study was to determine if the efficiency of the current empirically supported treatment regimens for panic disorder can be improved. Specifically, our study evaluated the effectiveness of a four-session treatment protocol for panic disorder. This protocol was intended to greatly reduce the frequency and severity of panic attacks after the first session and reduce residual panic-related anxiety with three

additional booster sessions. This treatment protocol included the following components in the first session: psychoeducation, diaphragmatic breathing retraining, autogenic training, release-only relaxation, imagery, cognitive restructuring, instructions for interoceptive and in vivo exposure, and cognitive rehearsal. Currently, the standard protocol for treating panic disorder is 12 sessions (Craske & Barlow, 2007). A briefer treatment may reduce attrition by providing significant relief after the first session. This intervention may also be more appealing to clients who may be hesitant to utilize mental health care or are worried about missing time from other obligations at work and home. It was further hoped that an effective, brief intervention could ultimately reduce panic disorder prevalence in the general population, and in turn, reduce unnecessary healthcare use and cost. Follow-ups were completed posttreatment to establish the long-term effectiveness of the treatment protocol at 3 months.

Literature Review

Panic disorder is classified as an anxiety disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR;* American Psychiatric Association, 2000). It is mainly characterized by the presence of panic attacks. A panic attack is defined as "a discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes" (APA, 2000, p. 432). These symptoms can be physiological and psychological. The physiological symptoms include chest pain, shortness of breath, increased heart rate, shaking, sweating, dizziness, numbness, and nausea, among others. The psychological symptoms include feelings of unreality, depersonalization, and a fear of losing control, going crazy, or dying (APA, 2000). Because this study predates the current DSM-5, this literature review refers to the *DSM–IV–TR*. However, the diagnostic criteria for panic attacks and panic disorder are sufficiently similar for the purposes of this study (APA, 2000, 2013).

A diagnosis of panic disorder requires the presence of repeated and unexpected panic attacks. Additionally, at least one of these panic attacks has to have been followed by at least 1 month of change related to the panic attacks. This change can be one or more of the following: "persistent concern about having additional attacks, worry about the implications of the attack or its consequences (e.g., losing control, having a heart attack, 'going crazy'), or a significant change in behavior related to the attacks" (APA, 2000, p. 440). Furthermore, to ensure an accurate diagnosis of panic disorder, the direct physiological effects of substance use (legal or otherwise) and general medical conditions must be ruled out as causes for the symptoms. Additionally, these symptoms must not be

better accounted for by other mental illnesses, such as PTSD, obsessive-compulsive disorder, separation anxiety disorder, social phobia, or specific phobia (APA, 2000).

Sometimes, panic disorder can be accompanied by agoraphobia, which is defined in the *DSM–IV–TR* as "anxiety about being in places or situations from which escape might be difficult (or embarrassing) or in which help may not be available in the event of having an unexpected or situationally predisposed panic attack or panic-like symptoms" (APA, 2000, p. 433). These situations include crowds, bridges, and being alone outside, among others. These situations are either avoided or endured with great distress. When endured, they are often accompanied by anxiety of having a panic attack and may require the presence of another person. The final aspect of the diagnostic criteria requires that the symptoms are not better accounted for by other mental illnesses, such as the ones mentioned above (APA, 2000).

Cognitive behavioral model of panic disorder.

According to Clark (1986), the cognitive behavioral model of panic disorder generally begins with the experience of physiological arousal and anxiety-related bodily sensations. These symptoms of physiological arousal are then combined with cognitive misappraisals of autonomic arousal symptoms. These sensations are usually germane to anxiety responses, such as shortness of breath, increased heart rate, heart palpitations, sweating, and dizziness (Clark, 1986; Levitt, Hoffman, Grisham, & Barlow, 2001). Individuals engage in cognitive misappraisals, where physiological arousal symptoms are interpreted as dangerous (Clark, 1986). For instance, the symptoms of increased heart rate or chest pain may be interpreted as signs of an imminent heart attack, or the feeling of dizziness might be interpreted as a sign that the individual will faint

(Clark, 1986; Levitt et al., 2001). Other thoughts include fears that the individual is going crazy, losing control, or dying (Barlow, 1992).

The catastrophic misappraisals further increase anxiety, which in turn increases the intensity of the physiological arousal symptoms. These increased levels of arousal are then interpreted as further proof that the catastrophic misappraisals are actually correct. This vicious cycle continues to build the state of arousal until the threshold of panic is reached. Furthermore, the environmental situations and/or physiological stimuli that precede this state of panic are associated with the panic attack itself. In other words, triggers or discriminative stimuli can include both internal cues (interceptive conditioning) and external cues (exteroceptive conditioning). In cognitive terms, such conditioning can lead to anticipatory fear of these stimuli, due to the belief that the stimuli will lead to another panic attack and avoidance (Clark, 1986).

According to this theory, this fear of subsequent panic, or fear of fear, is what causes the panic attack to develop into panic disorder. The fear of another panic attack leads the individual to become hypervigilant to anxiogenic physiological sensations that may signal the start of an attack, selectively focusing on the symptoms, often innocuous physiological sensations, which have become associated with panic (Barlow, 1992; Clark, 1986). This hypervigilance also can have the unintended effect of increasing the actual intensity of these physiological sensations and the perception of threat (Clark, 1986).

Subsequent panic attacks can be triggered by a variety of stimuli. Some stimuli may be internal bodily sensations, perhaps caused by other activities, such as exercise or caffeine intake. Other stimuli might be external, such as locations or objects associated

with previous panic attacks. Sometimes these external stimuli trigger the internal stimuli related to the onset of panic (Clark, 1986). Conversely, panic attacks can be completely unexpected and can appear to be spontaneous. However, the cues for these "unexpected" panic attacks, whether they are internal or external, can often be discovered with further investigation (Barlow, 1992).

In either case, the stimuli are perceived as a threat, which in turn creates apprehension. This apprehension increases the intensity of the bodily sensations, which are interpreted catastrophically. This then increases the level of perceived danger and threat, further perpetuating the vicious cycle that eventually leads to a panic attack. This collectively develops into a continual state of fear of stimuli, internal and external, associated with a panic attack (Clark, 1986). In some cases, this can lead to agoraphobia, which can be conceptualized as behavioral response to avoid feared stimuli (Clark, 1986; Levitt et al., 2001).

Panic disorder can also be thought of as a learned fearfulness of particular physiological sensations (Levitt et al., 2001). This takes place via classical conditioning. First, an individual experiences uncomfortable or distressing physiological sensations, which may be due to hyperventilation or internal biochemical processes. These distressing sensations can cause anxiety, which builds into the initial panic attack. These sensations serve as the unconditioned stimulus that leads to the unconditioned response of the initial panic attack (Craske & Barlow, 2007; Wolpe & Rowan, 1988).

These two experiences, physiological sensations and high anxiety, are temporally proximate to each other, leading to a classically conditioned association between them. When these sensations are paired with the panic attack, it increases the association

between them, such that the individual believes that there is a causal relationship and that the sensations are dangerous. This explanation asserts that the development of panic disorder "is due to the conditioning of the evoked anxiety to contiguous stimuli" (Wolpe & Rowan, 1988, p. 445), whether they are interoceptive or exteroceptive cues. These stimuli can then trigger the panic attacks (Wolpe & Rowan, 1988). The physical, cognitive, and behavioral components of panic each propagate and collectively interact to further intensify the feelings of anxiety (Levitt et al., 2001).

These internal sensations can also become associated with the external stimuli that were present during the panic attack. These external stimuli then become discriminative stimuli, which increase autonomic arousal. These stimuli, while perhaps specific in the beginning, can also become more generalized. For example, if an individual's external stimulus associated with panic attacks is being on a bus, and being on a bus induces a panic attack, this reaction may also generalize to being in an airplane, a car, or just being outside of the home (Craske & Barlow, 2007; Domjan, 2010).

Prevalence.

The prevalence of disorders is determined through a variety of means. One such tool for establishing the prevalence of panic disorder in the population is epidemiological study, such as the replication of the National Comorbidity Survey (Kessler et al., 2005). This replication was completed in 2003 and included 9,282 participants. The World Health Organization's Composite International Diagnostic Interview (CIDI) was used as the assessment tool for this national survey conducted in the United States. All data were gathered using face-to-face interviews (Kessler et al., 2005).

The initial results of this study found that 2.7% of the population suffered from panic disorder in the previous 12-month period, according to *DSM–IV* criteria (Kessler et al., 2005). Further analysis of the results determined that 0.4% of the population also experienced agoraphobia in addition to panic disorder in the past year. The total lifetime prevalence of panic disorder was found to be 4.7% of the population. Specifically, the lifetime prevalence for panic disorder with and without agoraphobia were 1.1% and 3.7%, respectively (Kessler et al., 2006).

The total 12-month prevalence of panic attacks, among people who may or may not have met full *DSM–IV* criteria for panic disorder, was 11.2% in this study. After accounting for the portion that qualified for panic disorder (2.7% in Kessler et al., 2005, and 2.8% in Kessler et al., 2006), there appears to be an additional 8.4% of the population that reported experiencing panic attacks in the past year without meeting the criteria for panic disorder. Furthermore, this study found that 22.7% of the population has experienced panic attacks in their lifetime without meeting the criteria for panic disorder, with an additional 0.8% also suffering from agoraphobia concomitant with their panic attacks. Collectively, 28.0% of the national population has suffered from symptoms of panic over the course of their lifetime (Kessler et al., 2006). This is over a quarter of the population of the United States, roughly 87 million people.

An epidemiological study of 43,093 participants in a community setting sought to establish the prevalence of panic disorder, with and without agoraphobia, in a nationally representative sample. The presence of panic disorder was established using the Alcohol Use Disorder and Associated Disabilities Interview Schedule–IV (AUDADIS–IV). The total 12-month prevalence for panic disorder was 2.1%, including 1.6% for panic disorder

without agoraphobia and 0.6% for panic disorder with agoraphobia. The total lifetime prevalence for panic disorder was 5.1%, including 4.0% for panic disorder without agoraphobia and 1.1% for panic disorder with agoraphobia.

This study also found that panic disorder was significantly more common in women, people with low incomes, middle-aged people, and people who were divorced, separated, or widowed. Ethnically, Native Americans were at higher risk for panic disorder than Caucasians, whereas Asians, Hispanics, and African Americans were at lower risk than the population average (Grant et al., 2006).

One study surveyed a general practice population for possible panic symptoms using a screening questionnaire, with a total of 1,152 respondents. Those who had positive scores for panic were given the Structured Clinical Interview for the *DSM–IV* (SCID–I) to assess for the presence of panic attacks and panic disorder. The results indicated lifetime prevalence for panic disorder and panic attacks of 6.2% and 8.6%, respectively, within the sample (Birchall, Brandon, & Taub, 2000).

Panic disorder is also quite prevalent in other countries around the world. In Sweden, a postal survey with 591 respondents found a 2.2% past-year prevalence for panic disorder, as measured by the panic disorder module of the CIDI (Carlbring, Gustafsson, Ekselius, & Andersson, 2002). A Canadian community health survey found that 1.7% of the sample reported symptoms of panic disorder, while an additional 6.4% reported having a panic attack in the past year, as measured by self-report (Kinley, Cox, Clara, Goodwin, & Sareen, 2009). A German community-based sample of 5,000 participants reported a 4.6% prevalence for panic disorder using the panic module of the Patient Health Questionnaire (PHQ; Wiltink et al., 2011). A Spanish epidemiological

study of primary care patients reported a 7.0% past-year prevalence and an 8.81% lifetime prevalence for panic disorder, as measured by the Spanish version of the SCID–I (Serrano-Blanco et al., 2010).

Not only have panic attacks been prevalent across the globe, they appear to have also become more prevalent across time. Goodwin (2003) sought to establish the temporal variance in panic attack prevalence in the United States. Two epidemiological samples were compared, one from 1980 and another from 1995. The former was from the Epidemiologic Catchment Area Program (ECA), while the latter was from the Midlife Development in the United States survey (MIDUS). The study compared the age group of 25 to 74 years old. The ECA used the National Institute of Mental Health Diagnostic Interview Schedule (DIS), while the MIDUS used the CIDI short form. The lifetime prevalence of panic attacks in 1980 was 5.3%, while the rate in 1995 was 12.7%, a statistically significant increase. While this study had a number of limitations, including a restricted age range in the sample and different wording of the questions, the results still indicate that the rate of panic attacks appears to have increased significantly over the last few decades (Goodwin, 2003).

Taken collectively, the prevalence of panic disorder appears to vary widely. The 12-month prevalence of panic disorder ranges from 1.7% to 7.0% (Kinley et al., 2009; Serrano-Blanco et al., 2010). The lifetime prevalence of panic disorder ranges from 4.7% to 8.81% (Kessler et al., 2006; Serrano-Blanco et al., 2010). Reviewing the two national epidemiological studies, the 12-month prevalence of panic disorder with and without agoraphobia appears to be 0.5% and 2.0%, respectively. Moreover, the lifetime

prevalence of panic disorder with and without agoraphobia appears to be 1.1% and 3.9%, respectively (Grant et al., 2006; Kessler et al., 2006).

Also relevant is the prevalence of panic attacks. The 12-month prevalence of panic attacks, as opposed to panic disorder, ranges from 6.4% to 11.2% (Kessler et al., 2006; Kinley et al., 2009). The lifetime prevalence of panic attacks ranges from 8.6% to 28.0% (Birchall et al., 2000; Kessler et al., 2006). Additionally, the lifetime prevalence of panic attacks appears to be growing over time, from 5.3% in 1980 to 12.7% in 1995, and finally to 28.0% in 2003 (Goodwin, 2003; Kessler et al., 2006). It should be noted that some of these differences in prevalence may be the result of varying study designs and procedures for data collection.

Comorbidity.

There is significant comorbidity accompanying panic disorder and panic attacks. Comorbidities include mental health disorders, suicide, medical conditions, and quality of life issues. The Canadian community health survey, mentioned earlier, further discovered that people who have panic attacks also have more serious mental illnesses, lower mental and physical functioning, higher levels of disability, and higher rates of suicidal ideation and suicide attempts than those who do not suffer from panic attacks (Kinley et al., 2009).

Mental health. An American national study found statistically significant comorbidity between panic disorder and specific mental disorders, including major depressive disorder, dysthymia, bipolar disorder, oppositional defiant disorder, attention deficit/hyperactivity disorder, intermittent explosive disorder, PTSD, separation anxiety disorder, generalized anxiety disorder, specific phobia, and social phobia (Kessler et al.,

2005). For example, using this same national comorbidity survey replication data, Cougle, Feldner, Keough, Hawkins, and Fitch (2010) reported that 35% of sample with PTSD also had experienced panic attacks in the past year. They found that the presence of panic attacks in patients with PTSD was associated with higher levels of global and PTSD-related disability, less time spent at work, and higher rates of substance use/dependence, depression, and other anxiety disorders (Cougle, Feldner, Keough, Hawkins, & Fitch, 2010).

A British study investigating comorbidity with panic disorder found high rates of both depression and generalized anxiety disorder. Rates were significantly higher for those who also had agoraphobia accompanying panic disorder than for those without it. This high comorbidity rate was even found in those study participants who only had subthreshold panic disorder symptoms (Skapinakis et al., 2011). Further complicating potential difficulties with panic disorder is the finding that comorbidity with depression is associated with more severe and persistent symptoms and increased role impairment (Roy-Byrne et al., 2000).

A study of patients with panic disorder and nocturnal panic attacks revealed that this subgroup suffers from more clinical syndrome comorbidity than those with panic disorder without nocturnal panic attacks. Specifically, there was a greater trend toward comorbid depressive and other anxiety disorders, as well as a higher rate of agoraphobia among those experiencing nocturnal panic attacks. Additionally, the group with nocturnal panic attacks also had a higher rate of personality disorders, particularly obsessive-compulsive and avoidant personality disorders. Although of clinical interest,

these group differences did not quite reach the level of statistical significance (Sarísoy, Böke, Arík, & Şahin, 2008).

Alcohol dependence is a significant comorbid issue for those who suffer from panic disorder. One study suggested that almost one in four people with panic disorder with agoraphobia and more than one in ten people with panic disorder without agoraphobia suffer from moderate to severe alcohol dependence. Even those with subthreshold panic symptoms have alcohol dependence at a rate of almost 12% (Skapinakis et al., 2011).

Suicide. Suicidality is a significant risk for those with panic disorder. The relationship between panic and suicidality was analyzed in a national survey with 5,877 participants. The presence of panic attacks and panic disorder was determined using the CIDI, and a history of suicidal ideation and suicide attempts was based on self-report in response to a survey question. This study found that both panic disorder and panic attacks (without a history of panic disorder) are strongly associated with increased suicidal ideation, both lifetime and within the past year, with rates ranging from 11.28% to 38.80% (Goodwin & Roy-Byrne, 2006). Another study, with 18,011 participants, also investigated the relationship between panic and suicidality. This study compared panic attacks and panic disorder to other mental illnesses. The results of the study indicated that both panic attacks and panic disorder were linked to a higher risk of suicidal ideation (Weissman et al., 1989). Furthermore, Skapinakis and colleagues (2011) found that over 21% of those with panic disorder without agoraphobia, and even those with subthreshold panic symptoms, reported suicidal ideation within the past year.

There is also a significant risk of actual suicide attempts in people with panic attacks and panic disorder. Goodwin and Roy-Byrne (2006) found that there was a statistically significant association between panic disorder and a history of suicide attempts within the past year. Weissman and colleagues (1989) found that both panic attacks and panic disorder were linked to a higher risk of suicide attempts. Specifically, the results found that 20% of those with panic disorder and 12% of those with panic attacks had attempted suicide. Again, these results remained even when accounting for confounding variables, such as depression or drug and alcohol abuse (Weissman et al., 1989).

Often, there are additional factors commonly germane to panic disorder that further increase the risk of suicidality. Specifically, the rate of suicidal ideation in a study sample was twice as high for participants with panic disorder with agoraphobia than for those without agoraphobia. This suggests that agoraphobia significantly increases the risk of suicide in clients with panic disorder (Skapinakis et al., 2011). In hospital emergency department settings, Foldes-Busque and colleagues (2011) found that comorbid psychiatric disorders were three times more likely and suicidal thoughts were twice more likely in patients with panic-like anxiety than in those without panic-like anxiety. Additionally, the presence of comorbid depression further increased the risk for suicidality (Roy-Byrne et al., 2000).

Medical conditions. Individuals with panic disorder are also at higher risk for certain medical conditions than the general population. A matched-control study in a general physician's office with 81 participants compared the rates of irritable bowel syndrome (IBS) in patients with panic disorder to those with no Axis I disorders.

Diagnoses were made using a semi-structured interview. The group with panic disorder reported a much higher rate of IBS (46.3%) than the group without panic disorder (2.5%). Additionally, the combination of panic disorder and IBS appears to put those patients at higher risk of comorbid back pain (Kaplan et al., 1996).

A study of 357 participants sought to determine the frequency and severity of IBS symptoms in patients with particular mental illnesses in comparison to the general population. Panic diagnoses were made using the SCID–IV, and patients were grouped according to their principal diagnosis. Results of this study indicated that patients with panic disorder had more frequent and severe IBS symptoms than the general population (Gros, Antony, McCabe, & Swinson, 2009).

A high rate of comorbidity has additionally been found between panic disorder and migraines. One study reported that 61.1% of patients with panic disorder experienced migraines, compared to 8.4% in the general population (Yamada, Moriwaki, Oiso, & Ishigooka, 2011). Another study in a headache clinic found that 27% of the patients met panic disorder criteria (Mehlsteibl, Schankin, Hering, Sostak, & Straube, 2011).

Quality of life. Panic disorder is also associated with lower levels of mental and physical functioning, higher levels of disability, and substantial impairment in quality of life (Barrera & Norton, 2009; Kinley et al., 2009). This higher level of disability and impairment is further elevated with the addition of agoraphobia (Skapinakis et al., 2011). Having panic attacks alone, without the full set of symptoms required for panic disorder, is associated with increased alcohol and drug use, impaired perception of physical and

emotional health, and poor social, occupational, marital, and financial functioning (Klerman et al., 1991; Markowitz et al., 1989).

Healthcare resource utilization.

Individuals with panic attacks and panic disorder often use healthcare resources to cope with the symptoms of panic attacks. Of the people with panic attacks who seek care for their attacks, about half will go to a medical setting. Only about a quarter of those who seek care go to a mental health setting. That means that medical settings receive almost twice the number of people with panic attacks than mental health settings. Emergency departments and family physicians' offices were the two most frequent sites of presentation (Katerndahl & Realini, 1995).

Emergency departments. A common symptom of panic attacks is chest pain (APA, 2000). People experiencing chest pain often report to hospital emergency departments to seek treatment (McCaig & Nawar, 2006). However, between 52% and 78% of all cases of chest pain in emergency departments remain without a clear cause or explanation at discharge (Christenson et al., 2004; Dumville, MacPherson, Griffith, Miles, & Lewin, 2007).

Foldes-Busque et al. (2011) noticed that symptom profiles of patients with unexplained chest pain were similar to the symptom profile of patients with panic attacks, including increased heart rate and the belief that they were having a heart attack. These authors sought to identify the prevalence of panic attacks and panic disorder, referred to collectively in this study as panic-like anxiety, in those patients who came to the emergency department with unexplained chest pain. The study used the emergency departments in two hospitals in Quebec, Canada. Together, these hospitals received

about 100,000 emergency department visits each year, from both rural and urban populations. Participants were screened for possible medical causes for their chest pain. Those patients without medical explanations for their chest pain were given the Anxiety Disorder Interview Schedule for DSM–IV (ADIS–IV) to identify the presence of panic-like anxiety. A total of 4,750 patients were screened for eligibility, with a total of 771 patients meeting criteria, which included being over 18, speaking English, consenting to complete the interview, and having chest pain without an objective medical explanation. Of those 771 patients with unexplained chest pain, 339 patients, or 44% of the sample, were found to have panic-like anxiety. Unfortunately, despite the fact that nearly one half of patients with unexplained chest pain had panic-like anxiety, only 7.4% of them were correctly diagnosed and given proper referrals. The authors speculate that this low rate could be due to emergency physicians' unfamiliarity with panic disorder, the similarity of panic disorder symptoms to medical conditions, and the tendency for both the physician and patient to focus on organic causes, rather than psychological causes, for physical symptoms (Foldes-Busque et al., 2011).

Other studies have found similar rates of panic disorder among patients with chest pain in medical healthcare settings. Huffman and Pollack (2003) used a meta-analysis to identify the factors that might identify panic disorder within this population. They identified rates of panic disorder among patients in emergency departments with chest pain in the range of 18% to 25%, with a combined average of about 20%.

Medical outpatient clinics. Emergency departments are not the only healthcare resources that individuals with panic disorder are inappropriately utilizing. The aforementioned meta-analysis conjointly investigated patients with panic disorder in

outpatient settings. Among patients with chest pain in outpatient clinics, the rates of panic disorder were found to be in the range of 16% to 57% (Huffman & Pollack, 2003).

In 1989, Beitman and colleagues investigated alternative causes of chest pain in 94 patients in a university hospital setting who had normal epicardial vessels. Using a structured psychiatric interview protocol, it was found that 34% of the sample met the diagnostic criteria for panic disorder. Katon and colleagues (1988) compared the rate of panic disorder in chest pain patients with and without a medical cause for their symptoms. A structured psychiatric interview protocol was used in a sample of 74 patients in a university hospital setting, including some who were referred from a private practice setting. The rate of panic disorder for patients without medical causes for their chest pain was 43%, compared to a 6.5% rate for patients with a medical cause for their chest pain.

A Scandinavian study suggested that panic disorder is three times more likely than a cardiac condition to be the cause of chest pain and palpitations among patients referred to a cardiac outpatient unit for these symptoms (Jonsbu et al., 2009). One study used patients hospitalized for chest pain in India. The inpatients were divided into two groups, those with ischemic heart disease and those with non-cardiac chest pain. Structured psychiatric interviews were used to establish whether they met the diagnostic criteria for panic disorder according to the *DSM–III*. Half of the patients with non-cardiac chest pain were found to meet the criteria for panic disorder, compared to approximately one tenth of the patients with ischemic heart disease. This study demonstrates that patients with panic disorder are utilizing inpatient hospitalizations. Additionally, this high number of panic disorder patients inappropriately utilizing

medical healthcare resources does not appear to be limited to Western cultures (Alexander, Prabhu, Krishnamoorthy, & Halkatti, 1994).

The Panic Attack Care-Seeking Threshold study surveyed 97 individuals with panic attacks to determine where they sought care for their panic attacks. The presence of panic attacks was identified using the SCID–III. Of the places where these individuals sought care, a general/family physician's office was the single most common site. A total of 35% of those with panic attacks had utilized a general/family physician's office to seek care for their symptoms. Emergency departments were used by 32% seeking care (Katerndahl & Realini, 1995).

Within family physicians' offices, it has been suggested that a quarter of all patients presenting with chest pain had panic disorder. Another quarter of patients with chest pain suffered from panic attacks, without the symptoms required for a diagnosis of panic disorder. Taken collectively, approximately half of all patients with chest pain in a family physician's office may be suffering from panic attacks (Katerndahl, 1996).

Chest pain is not the only symptom of panic disorder that may lead patients to utilize medical healthcare resources. Ross, Walker, Norton, and Neufel (1988) examined the records of patients presenting to emergency departments and walk-in clinics who were eventually diagnosed with panic disorder. They found that, along with chest pain, heart palpitations and difficulty breathing were the most frequent presenting physical complaints. Additional symptoms of panic disorder that may lead patients to inappropriately utilize medical healthcare resources include nausea, dyspepsia, lightheadedness, dizziness, and faintness (Kerber, 2009; Sansone & Sansone, 2009). This may be due to the fact that the somatic symptoms of panic mimic symptoms of medical

conditions, such as a heart attack, irritable bowel syndrome, or vestibular disease (Sansone & Sansone, 2009). Patients with panic disorder may also go to medical settings, such as emergency departments or clinics, because of fearful psychological and/or cognitive symptoms, believing that they are in imminent danger of death, disease, loss of control, or going crazy.

Other healthcare resources. In addition to visits to emergency departments, outpatient clinics, and physicians' offices, those who suffer from panic attacks and panic disorder have higher rates of healthcare utilization in terms of, for example, ambulance services, psychoactive drugs prescriptions, and use of general medical professionals (Katerndahl & Realini, 1997; Klerman et al., 1991; Markowitz et al., 1989). Even among those with other anxiety disorders, patients with panic disorder had the highest rates of medical healthcare utilization. Specifically, they had the highest percentage of overall medical visits and use of specialty medical services. Some of the most frequent specialty visits were to cardiology, family medicine, and emergency medicine (Deacon, Lickel, & Abramowitz, 2008).

Financial impact. The fact that many of these patients do not seek treatment at settings that can treat their symptoms and panic disorder results in visits to already busy emergency departments and clinics that may not have the resources to deal with these problems. This unnecessary overuse of healthcare resources can only increase already rising healthcare costs (Coley, Saul, & Seybert, 2009; Swensen et al., 2011).

An investigation of patients reporting to an emergency department with the possible diagnosis of panic disorder was conducted to determine the economic impact of their healthcare utilization. This 2-year retrospective cohort study found 1,099 patients

who had reported to the emergency department and were admitted to the hospital for evaluation, with the chief complaint of chest pain, chest pressure, or palpitations. A total of 155 patients were discharged without diagnoses to account for their symptoms. All met at least one criterion used to predict panic disorder among patients with chest pain, as established by Hoffman and Pollack (2003). Most of the sample met three of more of these criteria (Coley et al., 2009).

The average cost per patient for this one incident (which may have included a hospital admission) was $8,151, with a range of $1,489 to $26,845. The total cost for these 155 patients for their one-time hospital admission was over $1.26 million. Most of the costs were attributed to laboratory tests, nuclear cardiology, the emergency department, and the pharmacy, in that order. These 155 patients accounted for 318 electrocardiograms, 154 chest x-rays, and 137 myocardial perfusions. In the year following their admission to the hospital, 64 of the patients accrued an additional 183 visits to the hospital, resulting in an additional $1.59 million in healthcare costs (Coley et al., 2009).

Individuals with panic also incur losses in their personal finances through lost wages. Symptoms of panic can cause people to be absent from or be less productive at work. In fact, one study found that full-time workers with panic missed more than twice as many days of work (12) than the general population (5.2). This difference resulted in an additional $619 in lost wages per year for each individual with panic (Siegel, Jones, & Wilson, 1990).

Outcomes.

Unfortunately, many people with panic disorder do not receive the appropriate treatment for their symptoms. Two years after being identified and diagnosed with panic disorder in an emergency department, only 22% reported that they had obtained mental health treatment for their symptoms of panic disorder. This means that 78% of those identified with panic disorder still had not sought appropriate treatment for their symptoms, even 2 years later. Another study estimated that 40% of people who experience panic attacks do not seek treatment, which is a more conservative but still significant percentage (Katerndahl & Realini, 1995).

Treatments for panic disorder.

There has been an interesting evolution of the diagnosis of the disorder. Symptoms similar to panic disorder first appeared under the diagnoses of anxiety reaction and phobic reaction in the *DSM–I* and anxiety neurosis and phobic neurosis in the *DSM–II* (APA, 1958, 1968). The *DSM–I* describes anxiety reaction as:

> ... diffuse and not restricted to definite situations or objects, as in the case of phobic reactions. It is not controlled by any specific psychological defense mechanism as in other psychoneurotic reactions. This reaction is characterized by anxious expectation and frequently associated with somatic symptomatology. The condition is to be differentiated from normal apprehensiveness or fear (p. 32) [reprinted with permission]

The *DSM–I* describes phobic reaction as anxiety that has become:

> ... detached from a specific idea, object, or situation in the daily life and is displaced to some symbolic idea or situation in the form of a specific neurotic

> fear. The commonly observed forms of phobic reaction include fear of syphilis, dirt, closed places, high places, open places, animals, etc. The patient attempts to control his anxiety by avoiding the phobic object or situation (p. 33) [reprinted with permission]

This early absence of understanding of the disorder and lack of diagnostic specificity limited the study and treatment of panic disorder. It was not until the *DSM–III* that panic disorder gained its specific nomenclature, allowing it to be studied specifically (APA, 1980). Early treatments lacked empirical support and most were ultimately ineffective (Dittrich, Houts, & Lichstein, 1983; Roberts, 1984; Stampler, 1982).

More recently, many psychological treatments for panic disorder have been developed. These include panic control treatment, cognitive therapy, eye movement desensitization and reprocessing (EMDR), and emotion-focused therapy (Clark et al., 1999; Craske & Barlow, 2008; Goldstein, de Beurs, Chambless, & Wilson, 2000; Shear, Houck, Greeno, & Masters, 2001). However, not all treatments have proven effective.

Research on EMDR did not find significant results (Goldstein et al., 2000). This led Goldstein and colleagues to suggest that EMDR should not be used to treat panic disorder. Emotion-focused therapy was found to be less effective than either CBT or medication management with imipramine. At follow-up, the results for emotion-focused therapy were similar to the placebo control group (Shear et al., 2001). Only panic control treatment and cognitive therapy yielded statistically significant improvement in symptoms of panic disorder (Aaronson et al., 2008; Barlow et al., 2000; Clark et al., 1999; Klosko et al., 1990).

Cognitive therapy. Clark and colleagues (1994) evaluated the effectiveness of what they termed "cognitive therapy" for treating panic disorder. This study compared cognitive therapy with applied relaxation, imipramine, and a wait list control. A total of 246 patients were referred for panic disorder treatment. Of these initial referrals, 72 patients met the study criteria, which included meeting the *DSM–III–R* criteria for panic disorder, experiencing panic attacks currently, not being in concurrent treatment for panic disorder, and not suffering from severe agoraphobic avoidance (Clark et al., 1994).

The cognitive therapy condition included mainly cognitive, but also some behavioral, techniques to "help patients identify and change misinterpretations of bodily sensations" (Clark et al., 1994, p. 761). There were two main behavioral components. The first was inducement of feared sensations that was used to demonstrate for the participants alternative explanations for their symptoms. This was done via hyperventilation, centering attention on the body, or reading word pairs representative of their feared sensations and catastrophes. The second behavioral component was cessation of safety behaviors, which was done to provide the patients with evidence they could use to disconfirm the negative predictions about the consequences of their feared symptoms (Clark et al., 1994).

The cognitive components were identification and examination of the evidence behind the patients' misinterpretations of their symptoms, substitution of more realistic interpretations, and modification of relevant images. Homework assignments were also included in this condition. These included daily recording of negative thoughts and rational responses, as well as behavioral experiments, which were used to evaluate the negative thoughts (Clark et al., 1994).

Patients in the applied relaxation condition were shown how to recognize early signs of anxiety and then were trained to quickly relax. This condition also included homework assignments. In this case, they included practicing the relaxation skills two times a day. To avoid an overlap with the cognitive therapy condition, a behavioral rationale was substituted for a cognitive rationale for the relaxation training. Additionally, exposure to anxiety provoking stimuli began after the fourth session (Clark et al., 1994).

The imipramine condition consisted solely of medication management, with a neurochemical rationale for the treatment. Imipramine was initially administered in a small dose to avoid an increase in tension and arousal. The dose was gradually increased every 3 days until the panic attacks were eliminated or the maximum dose of 300 mg was reached. The average dosage prescribed was 233 mg per day. The effective dosage was maintained until the 6-month assessment (equivalent to the end of the booster sessions for the cognitive therapy and applied relaxation conditions), then it was gradually withdrawn over the next 2 to 3 months (Clark et al., 1994).

All nonpharmacological interventions were administered weekly for 3 months, and then maintained for 3 months with booster sessions. For the imipramine condition, medication administration was continued through this maintenance time period. Sessions for cognitive therapy and applied relaxation were 1 hour in duration. Sessions in the imipramine condition lasted only 25 minutes. A total of 64 patients across all conditions completed treatment. Most were female (78%) and most had some level of agoraphobia avoidance (81%; Clark et al., 1994).

Results indicated that all three treatments were significantly more effective in treating panic disorder than the wait list control condition. When comparing the treatments, cognitive therapy was more efficacious than both applied relaxation and imipramine. Cognitive therapy was rated superior on most measures at the 3-month follow-up. At the 6-month follow-up, cognitive therapy was as effective as imipramine, while both were superior to applied relaxation. By the 15-month follow-up, cognitive therapy again was rated superior to both applied relaxation and imipramine, although this difference was evident on fewer measures than before. The reduction in imipramine effectiveness over time was due to relapse after treatment, defined as an increase of panic attack frequency to the point where patients once again met criteria for panic disorder (Clark et al., 1994).

These results demonstrate that cognitive therapy was the most consistently efficacious treatment for panic disorder. Specifically, the results indicate that the reductions in misinterpretations of bodily sensations were a significant predictor of long-term positive outcome. This suggests that these cognitive changes should be an important focus of treating panic disorder in order to ensure sustained long-term success (Clark et al., 1994).

Brief cognitive therapy. Clark et al. (1999) subsequently developed a brief version of cognitive therapy to treat panic disorder. This was done to create a more cost-effective treatment. The authors compared the effectiveness of the full protocol of cognitive therapy with the brief protocol (Clark et al., 1999). The full 12-session cognitive therapy protocol was identical to the protocol in the aforementioned study (Clark et al., 1994).

The brief cognitive therapy protocol involved five 1-hour sessions of individual therapy over 3 months, the same time period as the full protocol. The authors designed four self-study modules to give to the participants between their sessions. These modules introduced concepts and procedures that would be expanded upon in subsequent cognitive therapy sessions. The modules also contained written exercises and homework assignments for the participants to complete on their own before reviewing them in session with the therapist (Clark et al., 1999).

Both conditions did not have participants engage in exposures to feared situations until after the fifth week. Additionally, participants in both conditions received two booster sessions in the 3 months following the initial 3-month treatment period. Participants in the wait list control group did not receive any treatment during the initial 3-month period, but were assigned to either of the cognitive therapy conditions during the following 3 months (Clark et al., 1999).

Assessments were made pretreatment, posttreatment, and at two follow-up dates, 3 and 12 months after treatment was completed. Of the 49 participants who initially met inclusion and exclusion criteria for entry into the baseline phase, a total of 43 were randomized and 42 completed treatment. Of the seven clients who did not complete treatment, some were withdrawn because they did not report any panic attacks during the baseline phase, while others withdrew voluntarily. Furthermore, similar to the previous study, most of the participants were female (62%) and most had some level of agoraphobia avoidance (85%). There was one notable difference from the previous study among the participants: a significant portion (32%) were also taking psychotropic medication during treatment (Clark et al., 1999).

Results of this study indicated that brief cognitive therapy was efficacious in treating panic disorder, when compared to wait list controls. This effect was found consistently across all measures. These improvements even continued through the 12-month follow-up assessments. Most significantly, there were no significant differences between the brief cognitive therapy and the full 12-session condition. Furthermore, the authors reported that the results from the brief cognitive therapy condition (Clark et al., 1999) were superior to the applied relaxation and imipramine conditions from the previous study (Clark et al., 1994).

A similar cognitive behavioral protocol was instituted in a primary care setting. In this study, Roy-Byrne and colleagues recruited 232 primary care patients who met the *DSM–IV* criteria for panic disorder from six primary care clinics (2005). These participants were randomly assigned to either a 6-session CBT intervention over 12 weeks (with medication if indicated) or treatment as usual (typically just pharmacotherapy). The CBT condition, administered by behavioral health consultants, also included six brief telephone follow-up booster sessions every 6 to 12 weeks. Participants from both conditions were able to take antianxiety medication (Roy-Byrne et al., 2005).

The authors of this study assessed symptoms of panic, anxiety, agoraphobia, and depression, as well as functionality, disability, and quality of life using a variety of measures. The results indicated that the CBT intervention group, with pharmacotherapy, had significantly higher rates of improvement than the treatment-as-usual group. These higher rates of improvement were seen for rates of responding and remittance of panic symptoms at both 3- and 12-month follow-ups. Additionally, significantly higher rates of

improvement in functionality and disability were seen for the CBT/pharmacotherapy group than the pharmacotherapy only treatment-as-usual group (Roy-Byrne et al., 2005).

These results suggest that brief protocols containing cognitive and behavioral components can be effective in treating panic disorder (Clark et al., 1994, 1999). Additionally, these results were likely to be maintained over a long period (Clark et al., 1999).

Panic control treatment. Panic control treatment (PCT) is a cognitive-behavioral protocol developed to treat panic disorder. The main components of PCT are psychoeducation, self-monitoring, breathing retraining, cognitive restructuring, in vivo exposure, and interoceptive exposure. It was originally designed as a 12-session protocol, consisting of one session per week, and included homework to be completed between the weekly sessions. Weekly homework assignments consisted of assigned readings and exercises, such as graded exposures, intended to generalize cognitive and behavioral change (Craske & Barlow, 2007).

The treatment begins with psychoeducation about the nature of panic disorder. Also included in session one is instruction in self-monitoring. During the second session, the patient and clinician collaboratively create a hierarchy of agoraphobic situations, implement breathing retraining, and explore the basics of cognitive restructuring. The third and fourth sessions continue breathing retraining and cognitive restructuring practice, with planning of in vivo exposures and the involvement of others in treatment to increase treatment success. Session five continues in vivo exposure and breathing retraining and introduces the interoceptive exposure. Sessions six through 11 continue in vivo and interoceptive exposure, including planning and problem-solving obstacles. The

final session focuses on accomplishments, maintenance, and relapse prevention (Craske & Barlow, 2007).

PCT was originally validated by Barlow, Craske, Cerny, and Klosko in 1989. In this study, 71 participants with panic disorder were randomly assigned to one of four conditions: applied progressive muscle relaxation, exposure and cognitive restructuring, a combination of the aforementioned treatments, and a wait list control, which consisted of self-monitoring and occasional telephone contact during the treatment period. All three treatment conditions were implemented via individual therapy for 15 weekly sessions, with the application of these skills to anxiety-provoking events in the real world through practice assignments starting during the sixth week. Outcome assessments included measures of trait anxiety, cognitive and somatic anxiety, phobic distress, depression, somatic symptoms, daily functioning interference, and self-report measures of anxiety, depression, pleasantness, and frequency and severity of panic attacks (Barlow, Craske, Cerny, & Klosko, 1989).

The authors found that all three treatment conditions were successful in treating panic disorder. Additionally, all three conditions were superior on most of the measures, as compared to the wait list control group. Only the combined and exposure and cognitive restructuring groups were significantly better than the wait list control. These differences between the relaxation, exposure and cognitive restructuring, and combination groups are best illustrated in the percentage of participants who reported the complete elimination of panic attacks after treatment: 60%, 85%, and 87%, respectively (Barlow et al., 1989).

Two years later, a follow-up with 41 of these participants revealed that participants who had received exposure and cognitive restructuring as their treatment maintained or improved their condition. Specifically, 81% of this group was still free of panic attacks 2 years later. This is compared to 43% and 36%, respectively, in the combined and relaxation groups. These results indicate that relaxation may have detrimental or dilution effect on the exposure and cognitive restructuring procedures (Craske, Brown, & Barlow, 1991).

The effectiveness of PCT has also been evaluated in comparison to medication management. Potential participants were screened using the ADIS–R to ensure that they met the *DSM–III* criteria for panic disorder. Additionally, panic attacks had to have been present in the week preceding the beginning of treatment and had to be at least in the moderate range of severity, as rated by a clinician. Prospective participants were excluded for pregnancy, significant medical problems, history of a psychotic disorder or dementia, recent alcohol or substance abuse, history of bipolar disorder, predominant depression, suicidal ideation, or medication use for panic disorder (Klosko et al., 1990).

Qualifying participants were then randomized into four conditions: PCT, medication management, placebo, and a wait list control. The protocol for PCT was similar to the procedures mentioned in the previous study. Alprazolam (Xanax®) was used as the pharmacological treatment in the medication management condition. All subjects in the treatment conditions received 15 weekly sessions of their respective interventions. The medication management group began gradually increasing dosages daily until the participant reached the therapeutic window, then maintained this dosage until gradually withdrawing the medication at the beginning of the 13th week. It should

also be noted that the wait list control group was not required to withdraw from other medications, so that even the wait list condition may have been receiving some treatment (Klosko et al., 1990).

Assessments were taken pretreatment and posttreatment. Of the 69 participants who met all inclusion and exclusion criteria, a total of 57 completed treatment. Twelve participants, most of whom were from the placebo condition, dropped out during the study (Klosko et al., 1990). The results of this study demonstrated that PCT was significantly superior to the placebo and wait list control conditions, with 85% of participants without panic attacks for 2 weeks at the end of treatment. The panic-free rates for the alprazolam, placebo, and wait list control conditions were 50%, 36%, and 33%, respectively. However, the higher rate for the PCT condition was not significantly different from the frequency in the alprazolam condition. However, PCT was superior to medication management in terms of reductions in somatic panic attack symptom severity (Klosko et al., 1990).

Barlow, Gorman, Shear, and Woods (2000) sought to further demonstrate the effectiveness of PCT in comparison to medication in treating panic disorder. In this study, the medication used was imipramine, and combination conditions were also included. The treatment conditions were imipramine only, PCT only, placebo only, a combination of imipramine and PCT, and a combination of placebo and PCT. A total of 497 patients across four treatment settings passed the initial screening for panic disorder, which was later confirmed pretreatment by the ADIS–R. Three hundred twelve patients completed enough of the treatment to be included in the analysis. Most of these patients were Caucasian (90.8%) and female (62.5%; Barlow et al., 2000).

The PCT sessions were 50 minutes in duration, while the medication/placebo sessions were 30 minutes, and the combination conditions received a total of 75 minutes of session time divided between two therapists. Treatment was divided into two phases: acute and maintenance. Acute treatment included 11 weekly sessions over 3 months. Maintenance treatment included monthly sessions for 6 months. The PCT protocol was from *Mastery of Your Anxiety and Panic, II* (Barlow & Craske, 1994). The medication protocol was similar to the aforementioned Klosko (1990) study, where dosage began small, gradually increased over time to a maximum dosage of 300 mg per day, and eventually was slowly withdrawn (Barlow et al., 2000).

The results of this randomized, double-blind, placebo-controlled clinical trial indicated that both PCT and imipramine were effective in treating panic disorder, when compared to the placebo condition. Imipramine initially appeared to be more effective than PCT at the conclusion of the acute treatment phase. However, later assessments indicated that PCT was a more durable and more tolerable treatment. Moreover, the combination of PCT and imipramine was found to be more advantageous than either treatment alone, but these differences only became apparent after the maintenance treatment phase, and not after the acute phase. Surprisingly, the combination group had the highest relapse rate at follow-up of all of the treatment conditions; however, the authors believe that this may have been due to an undetected selection bias (Barlow et al., 2000).

More recently, Aaronson and colleagues (2008) provided an in-depth analysis of the effectiveness of PCT in treating panic disorder. This study treated participants with 11 sessions of PCT across four treatment settings. Sessions were conducted weekly,

indicating that the treatment should have taken 11 weeks, although missed appointments extended some treatment up to 18 weeks. All participants on medication at the beginning of the study were asked to discontinue these medications by week 9 (Aaronson et al., 2008).

From a total of 454 referrals, 381 people met inclusion/exclusion criteria, completed the baseline diagnostic interview using the ADIS–IV, and came to the first session of treatment. Only 256 of these participants completed all 11 sessions of the PCT protocol. The other 125 either dropped out voluntarily or were removed from the study for reasons such as violating study protocol or continuing medication. This latter group was designated nonresponders for the purposes of the intent-to-treat analysis (Aaronson et al., 2008).

A total of 168 participants were rated by independent evaluators as responders to this treatment, as determined by a reduction in Panic Disorder Severity Scale (PDSS) scores of at least 40% from baseline levels, as well as a score of much or very much improved on the Clinical Global Impression (CGI). This represented 65.6% of the participants who completed treatment, while the intent-to-treat response rate was 44.1% of the total sample that began the study. The best predictors of response were higher levels of panic disorder symptom severity and lower levels of adult separation anxiety, as measured at the beginning of treatment. Neither the presence of comorbid disorders nor higher levels of baseline agoraphobia affected response rates (Aaronson et al., 2008).

Statistically significant differences in reductions in panic disorder severity scores between the responders and nonresponders were evident by the fourth week. Furthermore, by the sixth week, more than three fourths of the responders had two

consecutive weeks of significant reductions in severity of panic disorder symptoms, as opposed to just over one third of nonresponders who met these criteria by the same point in time. These results indicate that those who will respond to PCT will likely show significant progress by week 4 to 6, in this case, in the middle of the treatment process (Aaronson et al., 2008). This further suggests that an abbreviated treatment protocol for PCT may be effective in treating panic disorder.

Brief panic control treatment. A condensed version of the PCT protocol was developed and studied to determine if these components could be delivered effectively in a more succinct package. The authors of the study cited the potentially prohibitive cost of a 12- to 16-session treatment as a reason for seeking a briefer version of PCT. A four-session protocol was implemented with participants who were originally seeking medication to treat their anxiety symptoms (Craske, Maidenberg, & Bystritsky, 1995).

The first session of this brief PCT protocol provided comprehensive psychoeducation about the nature of panic and introduced the basic principles of cognitive restructuring. The second session began the practice of cognitive restructuring and breathing retraining. The third session continued the cognitive restructuring while also introducing and practicing interoceptive exposure. Finally, the fourth session concluded with a review of the concepts and skills learned during treatment. Throughout these sessions, participants were provided written session summaries and asked to practice each of these components on their own between sessions (Craske et al., 1995).

This condensed version of PCT was tested against nondirective supportive therapy (NST) as a control. This condition included the same basic information provided to the PCT condition during the first session. Subsequent sessions of NST did not

include any components of PCT. Instead, therapists provided a nondirective discussion of symptoms and stressors. Therapists were supportive and engaged in reflective listening, but did not encourage or discourage any coping skills presented by the participants (Craske et al., 1995).

Of the possible participants, only 34 met all inclusion and exclusion criteria. This included a positive screen for the presence of panic disorder using the SCID and ADIS–R. Participants were then randomly assigned to one of two treatment conditions: brief PCT or NST. Four rejected randomization, leaving 30 participants, most of whom were Caucasian (80%) and female (68%). Additionally, two thirds of the participants had agoraphobia (Craske et al., 1995).

Measures for this study included the Anxiety Sensitivity Index, the Fear Questionnaire, the Four Dimensional Anxiety Scale, and the Subjective Symptoms Scale. Clinician ratings from the ADIS-R were also included, specifically, the degree of worry of another panic attack occurring (0 to 8) and the number of incidents of agoraphobic avoidance. Furthermore, participants were also asked to record the occurrence of panic attacks and daily ratings of maximum anxiety, average depression, and average worry about having panic attacks (0 to 8). Participants were assessed pretreatment and posttreatment. No follow-up was provided, meaning long-term outcomes could not be determined (Craske et al., 1995).

Of the 30 participants who began treatment, 29 were able to complete all four sessions (one dropped out from the NST condition). The results of this study demonstrated that PCT was significantly more effective than NST. This included the finding that participants in the PCT condition had significantly decreased their levels of

phobic distress (improvement of 3.6 points on agoraphobia subscale of Fear Questionnaire) and worry about the recurrence of panic (improvement of 2.4 points on the ADIS–R), whereas the participants in the NST condition saw no significant effects (improvements of 1.5 points and 0.4 points, respectively). Additionally, a higher percentage of participants in the PCT condition were panic free at the end of treatment than in the NST condition. Thirty-eight percent of PCT participants were improved enough that they no longer met the criteria for panic disorder and/or declined participation in further treatment of panic disorder via medication trials (Craske et al., 1995).

While this brief version of the PCT protocol appears to be effective in treating panic disorder, the improvements seen in this study's PCT condition were not as significant as improvements seen in full 12- to 16-session protocols of PCT (38% vs. 85% panic free at end of treatment; Barlow et al., 2000; Craske et al., 1995). This condensed version of PCT appeared to be less effective than the original, although significant caveats about the nature of this study's sample may explain this difference. These include the preference for pharmacological treatment and the unusual procedure requiring that participants be abstinent from medication for the course of the study. At the very least, further investigation into a brief PCT protocol is warranted. The authors suggested augmenting their condensed protocol with additional components, such as extensive bibliotherapy and homework assignments (Craske et al., 1995).

In summary, many treatments for panic disorder have been found to be efficacious (Aaronson et al., 2008; Barlow et al., 2000; Clark et al., 1994, 1999; Craske et al., 1995; Klosko et al., 1990). These include protocols that use various cognitive and

behavioral components, including cognitive therapy and PCT. Additionally, brief versions of two of the aforementioned protocols have shown positive outcome in as little as four to five sessions (Clark et al., 1999; Craske et al., 1995).

Attrition rates.

It should not be surprising that dropping out of treatment can reduce treatment efficacy and negatively impact mental health. Research indicates that adult psychiatric clients who do not remain in treatment have more severe mental health problems and more impairment in social functioning than those who remain in treatment. These negative consequences are the result of a lack of treatment (Killaspy, Banerjee, King, & Lloyd, 2000). In previous studies with PCT, relatively high attrition rates were found. Klosko and colleagues (1990) began with 18 participants in the PCT condition, while 3 dropped out, indicating a 16.7% attrition rate. In the Barlow and colleagues (2000) study, three conditions included PCT (PCT alone, PCT plus medication, PCT plus placebo pills), with a total of 148 participants. Fifty-seven of these clients dropped out before the study could be completed, leaving an attrition rate of 38.5%. Of the 381 participants who began treatment with Aaronsson (2008), 125 dropped out of treatment for various reasons. This suggested a mean attrition rate of 32.8%.

Conversely, a brief version of panic control treatment, lasting four sessions, had a zero percent (0%) dropout rate (Craske, Maidenberg, & Bystritsky, 1995). Similarly, brief cognitive therapy for panic disorder, lasting only five sessions, also had a zero percent (0%) dropout rate (Clark et al., 1999). On the other hand, in a six-session CBT intervention in a primary care setting, 60% of the participants dropped out before completing the treatment, with an average of 4.1 sessions attended. Additionally, the

response and remission rates were higher for participants who completed all six sessions than those that just completed three to five sessions, although it was not made clear if these were statistically significant differences (Roy-Byrne et al., 2005). It should be noted that these higher rates in primary care settings may simply reflect differences in the study samples. Nonetheless, these results, taken together, indicate that brief treatments for panic disorder, lasting five sessions or fewer, are likely to produce lower attrition rates than protocols of greater duration while still providing positive outcomes in terms of mental health and social functioning.

Treatment components.

Cognitive-behavioral protocols for panic disorder have included various components, including psychoeducation, breathing retraining, relaxation training, cognitive restructuring, autogenic training, and exposure, either alone or in various combinations. These interventions, which are elucidated below, are of current focus as they are part of the protocol under investigation in this study, which evolved independently from clinical experience by Dr. Brad Rosenfield (Philadelphia College of Osteopathic Medicine) and Dr. J. Russell Ramsay (Center for Cognitive Therapy at the University of Pennsylvania).

Psychoeducation. Psychoeducation is a crucial component of treating panic disorder. It is provided to help clients understand their panic attacks, appreciate the genesis and maintaining factors associated with their panic symptoms, and ultimately learn how to manage their panic attacks and associated anxiety. For instance, clients learn about the underlying physiological aspect of panic. This includes understandable information on the autonomic nervous system reframed as the fight-or-flight system,

hormonal and cardiovascular systems, and how natural responses to perceived danger can account for the physical symptoms of anxiety and panic attacks. Furthermore, clients learn about the role of avoidance in maintaining panic and anxiety (Craske & Barlow, 1993; Craske & Barlow, 2008)

The goal of psychoeducation is to teach clients that the physiological sensations associated with anxiety and panic attacks can be tolerated without resulting in any catastrophic consequences, such as a heart attack, going crazy, or losing control. Ultimately, this should help decatastrophize the feared consequences of panic symptoms (Levitt et al., 2001). Psychoeducation also provides the rationale for all of the elements of this brief treatment protocol. Understanding the rationale also provides the client the motivation to learn the rest of the treatment regimen, some of which can be counterintuitive and aversive, particularly the exposure components (Craske & Barlow, 1993, 2007; Westra & Dozois, 2006).

Cognitive behavioral functional analysis. A cognitive-behavioral functional analysis of a specific and most typical panic attack for clients can also be used in the treatment of panic disorder. This includes the setting events, antecedents, behaviors, and consequences for their panic attacks. The purpose of this is to help clients learn what factors influence the onset and maintenance of their panic attacks (e.g., O'Neil, 1997).

Breathing retraining. It has been noted that abnormal breathing plays a role in development and maintenance of panic disorder (Bass, Lelliott, & Marks, 1989; Ley, 1985). Hyperventilation, or breathing more than is necessary for metabolism, causes the body to expel carbon dioxide from the blood at a faster rate than it is created in the body. The lack of carbon dioxide inhibits the ability of hemoglobin to release oxygen. This

decreases the concentration of oxygen and increases respiratory drive, which signals the body to activate the sympathetic nervous system, the fight-or-flight reaction. Ley (1985) theorized that the interaction between fear and hyperventilation spurs panic attacks. This begins with mild overbreathing in response to a stressor, which then escalates via a positive feedback loop between intensifying anxiety and increasing hyperventilation. This activity, along with the other signs and symptoms of panic, correlates with sympathetic nervous system arousal (Ley, 1985). On the other hand, homeostasis is maintained by the parasympathetic nervous system, the counterbalance to the sympathetic nervous system. To this end, controlled diaphragmatic breathing is helpful in activating the parasympathetic nervous system, thereby reducing or preventing the panic cycle (Courtney, 2009; Craske & Barlow, 1993; Recordati & Bellini, 2004)

Ideally, breathing retraining should consist of three components. First, the therapist educates the client about the physiological basis for hyperventilation and its implication in creating panic attacks. Second, the therapist teaches the client diaphragmatic breathing, breathing with the diaphragm as opposed to the chest. Third, the therapist teaches the client to slow the breathing rate to about 8 to 10 breaths per minute. The technique of diaphragmatic breathing activates the homeostasis of the parasympathetic nervous system and can be used to prevent the onset of panic attacks as well as to induce relaxation and adjust everyday breathing patterns (Craske & Barlow, 1993; Levitt et al., 2001).

Breathing retraining as a sole treatment component. Various studies have been conducted in an attempt to determine the effectiveness of breathing retraining in treating panic disorder, either as the sole component or part of a larger treatment. Early studies of

breathing retraining used it as the sole treatment intervention (Clark, Salkovskis, & Chalkley, 1985; Salkovskis, Jones, & Clark, 1986). These researchers found that instructions to slow respiration rate alone were effective in reducing panic attack frequency, as well as in decreasing self-reported levels of anxiety and depression. However, these studies suffered from major limitations, including very small numbers of participants (18 or fewer) and lack of a control group. Additionally, they did not include instructions for abdominal or diaphragmatic breathing as part of their breathing retraining (Clark et al., 1985; Salkovskis et al., 1986).

Hibbert and Chan (1989) addressed these limitations by investigating the effects of breathing retraining in a sample of 40 participants with panic disorder, including a control group. The experimental group included 21 participants who received two sessions of the Clark et al. (1985) breathing retraining, which included voluntary hyperventilation, an explanation of how hyperventilation induces panic, and training in slow breathing. The control group received a placebo treatment which can best be described as psychoeducational in nature. After 2 weeks of treatment, both groups improved in terms of self-reported anxiety, but no other significant differences between the groups were evident (Hibbert & Chan, 1989).

Following these 2 weeks, both groups received three weekly sessions of in vivo exposure therapy. At the 3-week follow-up, the breathing retraining group appeared to have improved more than the control group in terms of observer-rated anxiety, but there were no significant differences in terms of self-reported anxiety between the two groups. Further limiting the strength of this study was the lack of any significant follow-

up to assess long-term effects, as the follow-up took place only 3 weeks posttreatment (Hibbert & Chan, 1989).

A more recent investigation of breathing retraining as the sole treatment intervention for panic disorder used slow paced breathing, but also included instructions for diaphragmatic breathing. Four participants underwent five sessions of breathing retraining over 4 weeks. Outcome measures were the Panic Disorder Severity Scale, Anxiety Sensitivity Index, the Beck Depression Inventory, and the trait version of the State-Trait Anxiety Inventory. Improvements were found across all outcome measures at posttreatment and at the 2-month follow-up. While this study used more methodologically sound assessment techniques, it suffers from the same limitations as the early studies: a very small number of participants and lack of a control group (Meuret, Wilhelm, & Roth, 2001).

Breathing retraining as a component of larger treatment protocol. Other studies have included breathing retraining in combination with other components in a larger treatment protocol. An early study in 12 patients used diaphragmatic breathing retraining in combination with in vivo exposure versus in vivo exposure alone. Seven participants received two weekly sessions of breathing retraining plus seven weekly sessions of in vivo exposure. The control group included five participants who received nine weekly sessions of in vivo exposure. At posttreatment and at the 1-month follow-up, both groups improved in terms of panic attack frequency, phobia scores, and somatic symptoms scores, with no significant differences between them. At the 6-month follow-up, the breathing retraining group was superior in terms of these same outcome measures. These results indicated that breathing retraining is a beneficial component that leads to

improved longer-term positive treatment outcomes than exposure without the breathing component (Bonn, Readhead, & Timmons, 1984).

Conversely, two studies that compared diaphragmatic breathing retraining to other panic interventions indicated that the breathing component was relatively less effective in improving outcomes. One study compared breathing retraining to interoceptive exposure as a third treatment component in combination with in vivo exposure and cognitive restructuring. Both groups received 12 weekly sessions of in vivo exposure and cognitive restructuring lasting 90 to 120 minutes. The variable component, either diaphragmatic breathing retraining for 18 of the participants or interoceptive exposure for the other 20, was incorporated into the treatment starting during session four and continued throughout the rest of the treatment (Craske, Rowe, Lewin, & Noriega-Dimitri, 1997).

Both groups had similar improvements on most of the more than two dozen measures of anxiety and panic-related distress. However, the interoceptive exposure group demonstrated significantly superior results on five measures: panic attack frequency, panic attack severity, agoraphobia fear, social fear, and overall level of impairment. Additionally, 88% of the interoceptive condition reported zero panic attacks at posttreatment, compared to 43% with no panic attacks in the breathing retraining condition. The study was limited by its high attrition rate across both conditions, as more than a quarter of the sample did not complete treatment. Nonetheless, this study appears to indicate that breathing retraining was an inferior treatment component to interoceptive exposure. However, the authors note that these differences could be due to the highly individualized nature of the interoceptive exposure, compared to the more standardized nature of the breathing retraining. Breathing retraining is a standard procedure that is the

same for each participant, whereas interoceptive exposures differ for each participant, depending on the specific nature of his/her fears and thus is more tailored to the individual (Craske et al., 1997; Craske & Barlow, 2008).

A logical next step would be to see if adding breathing retraining to a CBT protocol would provide any incremental benefits. A total of 77 participants with panic disorder were recruited and randomly assigned to one of three treatment conditions: CBT with diaphragmatic breathing retraining, CBT without diaphragmatic breathing retraining, or a wait list control. Treatment took place in group settings of five to seven participants over 12 weekly sessions lasting 2 hours. This common base CBT included psychoeducation, cognitive restructuring, in vivo exposure, and interoceptive exposure. The breathing retraining was added to sessions four and five, which were each 30 minutes longer to account for the extra material (Schmidt et al., 2000).

Outcome measures for this study were the Multicenter Panic Anxiety Scale, the Sheehan Patient-Rated Anxiety Scale, the Mobility Inventory, the Sheehan Disability Scale, and the Beck Depression Inventory. Both treatment groups had similar improvement rates on all of the outcome measures, with no significant differences. However, the CBT group without diaphragmatic breathing retraining scored higher on end-state functioning and sought less additional treatment than the group that received the additional diaphragmatic breathing retraining. This led the authors to conclude that breathing retraining did not provide any incremental benefits in the treatment of panic disorder, and may in fact be detrimental because it appeared to lead to a less substantial recovery and a higher risk of relapse (Schmidt et al., 2000).

Breathing retraining summary. While some studies have indicated breathing retraining is an effective sole treatment component for panic disorder, they all have some severe methodological limitations (Clark et al., 1985; Hibbert & Chan, 1989; Meuret et al., 2001; Salkovskis et al., 1986). In terms of a supplemental treatment component, results of breathing retraining's effectiveness are mixed. While Bonn and colleagues (1984) found it to be a beneficial component in long-term outcomes, more recent studies suggested that breathing retraining is not a necessary component of treatment (Craske et al., 1997; Schmidt et al., 2000) and may, in fact, be perceived as a detrimental safety behavior that, in turn, interferes with effective treatment exposures (Salkovskis, Clark, & Gelder, 1996). However, Meuret and colleagues address this concern as theoretically driven criticism that lacks true empirical evidence to support its negative view of breathing retraining (Meuret, Wilhelm, Ritz, & Roth, 2003). In fact, Wolpe and Rowan (1988) used classical conditioning to postulate that a state of reduced anxiety, such as one caused by breathing retraining, paired with normally anxiety-provoking experiences could actually increase counterconditioning and the rate of extinction of learned fearfulness. Additionally, studies that indicated no benefits of breathing retraining may have suffered from poor timing, small samples, and order effects in teaching this technique. For instance, Schmidt and colleagues (2000) waited until late in their protocol to teach diaphragmatic breathing, which could have limited its effect and contributed to poor outcomes.

Imagery.

Guided imagery is a technique used to enable relaxation by creating mental images (Moffatt, Hodnett, Esplen, & Watt-Watson, 2010). Providing an alternative focus

for cognitive attention can keep an individual from reengaging in anxious thoughts (Harvey & Payne, 2002). Deliberately changing mental imagery can have an immediate impact on autonomic activity (Lee & Olness, 1996). There is empirical support for imagery as effective in reducing anxiety in a variety of populations (Gonzales et al., 2010; Papathanassoglou, 2010). It is believed that imagery can be helpful in assisting the relaxation skills learned through breathing retraining and autogenic training.

Autogenic training.

Autogenic training is a self-induced relaxation technique developed by Johannes Schultz (Schultz & Luthe, 1959). The training consists of six exercises. The first targets muscle relaxation by repeating a verbalization combined with a subtle relaxation technique designed to create a feeling of heaviness. The second exercise uses passive concentration to form the visualization of warmth. The following exercises each use a distinct visualization to focus on calm cardiac activity, slow respiration, warmth in the abdomen, and coolness in the head (Kanji & Ernst, 2000).

Initial investigations by Sakai (1996) found that autogenic training was an effective component in a behaviorally based treatment for panic disorder that included components such as in vivo exposure. Further research discovered that autogenic training is an effective stand-alone treatment for a variety of anxiety disorders. In this study, 55 patients in a psychiatric medical setting with anxiety disorders, most of which were panic disorder (58%), were treated by the author. Results showed that more than three fourths of the sample was significantly improved by the end of treatment (Sakai, 1997). Building on this research, Ashihara, Tsutsumi, Osawa, and Sata (2000) found

autogenic training to be an efficacious treatment for panic disorder in an outpatient group setting.

A systematic meta-analysis of studies of autogenic training for anxiety and stress found that most subjects reported successful outcomes; however, the authors found that the studies all deviated from the accepted technique (Kanji & Ernst, 2000). These deviations from the accepted technique for autogenic training raise questions for virtually every manner of validity and reliability for the aforementioned studies. Additionally, most of these studies suffered from other methodological deficiencies. Therefore, the authors concluded that autogenic training had yet to be properly validated as an effective treatment for anxiety disorders, including panic disorder (Kanji & Ernst, 2000).

Addressing these methodological deficiencies in a randomized control trial, Kanji, White, and Ernst (2006) found that autogenic training was an effective way to reduce anxiety in a population of nursing students. A study conducted by Ota, Majima, Shimura, and Ishikawa consisted of 89 participants who were divided into two groups. Both groups received the same base psychosomatic treatment for panic disorder, but one group of 46 participants also received autogenic training, while the other group of 43 participants did not. Assessments were taken pretreatment and posttreatment in areas such as avoidance severity, agoraphobic anxiety, the level of depression, subjective symptoms, and withdrawal from medications (Ota, Majima, Shimura, & Ishikawa, 2007).

Both groups demonstrated significant improvements in terms of avoidance, agoraphobia, and depression. However, the group that received autogenic training showed significantly more improvement in agoraphobic avoidance, subjective symptoms, and withdrawal from medications than the group that did not receive autogenic training.

The authors concluded that autogenic training should help individuals with panic disorder manage anxiety, agoraphobic avoidance, and other symptoms of panic disorder (Ota et al., 2007). This collection of evidence suggests that autogenic training could be a helpful component in treating panic disorder (Ashihara et al., 2000; Ota et al., 2007; Sakai, 1996).

Release-only relaxation.

Release-only relaxation was originally developed as a component of applied relaxation. Applied relaxation is a coping technique that involves progressively tensing and relaxing muscle groups in order to quickly create a state of relaxation that can be used to counteract sensations of anxiety (Öst, 1987). The protocol for applied relaxation includes four components: progressive relaxation of muscle groups, release-only relaxation, cue-controlled relaxation where classical conditioning occurs, and differential relaxation. A review of controlled studies found that applied relaxation is efficacious for treating various anxiety disorders (Öst, 1987). As a treatment for panic disorder, applied relaxation has been established as more effective than progressive muscle relaxation (Öst, 1988). Additionally, one study found applied relaxation alone to be as effective as cognitive therapy, in vivo exposure (Öst, Westling, & Hellström, 1993), and CBT (Öst & Westling, 1995) in treating panic disorder.

One phase of applied relaxation is release-only relaxation. It differs from the other phases of applied relaxation in that release-only does not include the tensing of muscle groups, as in both the complete protocol for applied variety and progressive muscle relaxation. Instead, clients are instructed to relax their muscle groups directly, going from the top of their head down to their toes. Providing the client with

psychoeducation about anxiety, the autonomic nervous system, and relaxation that includes a rationale for how the exercise will work for them is crucial for successful implementation. The release-only relaxation exercise should take 5 to 7 minutes in order for relaxation to be completed (Öst, 1987).

Currently, no studies have been published that only use the release-only phase of applied relaxation to treat panic disorder, although it has anecdotally been found to be effective as an adjunctive treatment in outpatient clinical populations (Personal communication, Brad Rosenfield, July 9, 2012; Personal communication, J. Russell Ramsey, August 3, 2012). However, release-only relaxation has been studied in isolation as a tool for relaxation. Carlbring, Björnstjerna, Bergström, Waara, and Andersson (2007) compared computer and therapist administered release-only relaxation in a student population. Sixty participants were randomly assigned to three conditions: computer administered relaxation, therapist administered relaxation, and a control condition. Outcome measures included skin conductance, as measured by specialized hardware, and well as the participants' self-reported state of relaxation, as measured by a visual analogue scale. The authors found that both relaxation conditions resulted in significant improvements in relaxation, compared to the control condition (Carlbring, Björnstjerna, Bergström, Waara, & Andersson, 2007). These results indicate that release-only relaxation has the potential to be an effective, and by extension, a helpful intervention in reducing sympathetic nervous system arousal, which is fundamental to the onset and maintenance of panic disorder.

Differential relaxation.

Another component of applied relaxation is differential relaxation. The purpose of differential relaxation is to generalize the ability to relax to other settings than the one in which it was learned. This is accomplished by having the client go through the relaxation procedure in various states and settings, such as standing, talking on the telephone, and walking (Öst, 1987). As differential relaxation is an adjunctive component to learning relaxation skills, it has not been studied in isolation in the treatment of panic attacks. However, as a component in applied relaxation for panic disorder, it has proven to be a part of a larger successful treatment (Öst, 1988; Öst & Westling, 1995; Öst et al., 1993).

Despite the fact that it was removed from the latest protocol for PCT, patients with panic disorder have reported that such relaxation protocols give them a sense of self-efficacy and control of their own autonomic nervous system, that is, bodily sensations related to panic attacks. This sense of self-efficacy and control can thereby speed treatment effects. Relaxation exercises also orient patients to exposure to symptoms and may reduce avoidance (Personal communication, Brad Rosenfield, July 9, 2012; Personal communication, J. Russell Ramsey, August 3, 2012).

Cognitive restructuring.

Cognitive restructuring is the process of identifying, exploring, and correcting the beliefs and misinterpretations that clients have about their panic symptoms. These can include thoughts such as “my heart is beating so fast that I am going to have a heart attack and die” and “I will lose control and go crazy if I have another panic attack.” Using knowledge gleaned from the psychoeducation component of treatment, especially

about the physiology of panic, clients learn that such catastrophic expectations associated with panic attacks are extremely unlikely to occur. They learn that these physical and cognitive symptoms are not an indication of an imminent catastrophe. Specifically, cognitive restructuring helps clients identify and evaluate their catastrophic and unreasonable misinterpretations using coping skills, such as examining the evidence to determine the realistic chance that a catastrophic outcome will occur. For instance, if the individual fears having a heart attack and dying, the clinician might respectfully ask, "How many times have you had a panic attack in your life?" "How much did you believe you would have a heart attack and die at the height of your panic attack?" and then, "How many times have you had a heart attack and died?" Clients then learn to change these thoughts by recognizing unrealistic expectations, brainstorming alternate explanations for their symptoms, and then developing more realistic alternative cognitions (Craske & Barlow, 1993, 2007; Levitt et al., 2001).

Salkovskis, Clark, and Hackmann (1991) studied the effectiveness of cognitive restructuring in treating panic disorder. Seven participants with panic disorder were included in a multiple baseline across subjects design. A comparison was made between two purely cognitive treatment conditions, as neither included breathing retraining, exposure, nor any other behavioral components. The active cognitive treatment included cognitive restructuring aimed at changing the participants' catastrophic misinterpretations of physical symptoms of panic. The second cognitive treatment, serving as a control condition, had the same duration, structure, and cognitive exercises as the main condition, but explicitly did not discuss misinterpretations of physical symptoms of panic. Both

treatment conditions consisted of two sessions, with a total time not exceeding 2.5 hours (Salkovskis, Clark, & Hackmann, 1991).

For outcome measures, Salkovskis and colleagues used the frequency of panic attacks, as reported on a daily panic diary, and the level of belief in their misinterpretations of physical symptoms of panic, rated on a scale from 0 to 100. The authors found that the active treatment that included cognitive restructuring aimed at changing the participants' catastrophic misinterpretations of physical symptoms of panic was efficacious in reducing the participants' level of belief in their catastrophic misinterpretations as well as the frequency of their panic attacks. Additionally, the nonactive cognitive control condition did not result in improvement in either of these measures. Furthermore, two of the participants were completely panic free following the active cognitive treatment. This study appears to indicate that cognitive restructuring can treat panic disorder. However, this study was limited by the small sample size ($N = 7$) and lack of random assignment (Salkovskis et al., 1991).

Beck, Stanley, Baldwin, Deagle, and Averill (1994) compared cognitive therapy and relaxation training in the treatment of panic disorder. Sixty-four participants with panic disorder were randomly assigned to one of three conditions: cognitive therapy, relaxation training, and a minimal-contact control group. The cognitive therapy condition consisted of psychoeducation and cognitive techniques such as cognitive restructuring. The relaxation training included progressive muscle relaxation and breathing retraining. The minimal-contact control group received weekly contact phone calls and empathetic listening, but no formal treatment or psychoeducation was provided (Beck, Stanley, Baldwin, Deagle, & Averill, 1994).

Treatment was administered in a group format with 4 to 6 members per group, meeting for ten weekly 90-minute sessions. Many specific outcome measures were used, including measures for panic symptoms, global psychological functioning, agoraphobic fear, and secondary fears and anxiety. Participants were assessed pretreatment, posttreatment, and at 1-, 3-, and 6-month follow-ups, with some measures also administered at week 5 of treatment. Cognitive therapy and relaxation training were both superior to the control condition in all areas measured. Statistically significant improvements were observed in both treatment conditions for global severity of panic disorder and panic-related worry. Additionally, improvements were also seen in measures of agoraphobic fear and state anxiety. Furthermore, a significantly higher number of treatment responders were found in the cognitive therapy and relaxation training groups, 82% and 68%, respectively, compared to the control group, 36% (Beck et al., 1994).

Bouchard and colleagues directly compared the rate of change of cognitive restructuring with exposure therapy in the treatment of panic disorder. Twenty-eight participants diagnosed with panic disorder with agoraphobia were included in this study. Participants were randomly assigned to receive either cognitive restructuring or exposure therapy. Both conditions included 15 weekly sessions lasting 1.5 hours each. Assessments were taken throughout the treatment period and at a 6-month follow-up in order to determine the rate of change of panic-related symptoms (Bouchard et al., 1996).

The outcome measures, as in the previous study, were numerous. They included measurements of panic attack frequency and severity, self-reports of panic and agoraphobia, and overall functioning, which included anxiety, depression, and level of

impairment. Ultimately, the study found that both treatment conditions were effective in treating panic disorder. Additionally, no significant differences were found between cognitive restructuring and exposure therapy in the rates of change on any of the measures. The results of this study suggest that cognitive restructuring is as efficacious in treating panic disorder to exposure therapy (Bouchard et al., 1996).

Cognitive restructuring has also been included as a component in larger treatment protocols for treating panic disorder. As detailed above, cognitive therapy (Clark et al., 1994, 1999) and panic control treatment (Aaronson et al., 2008; Barlow et al., 2000; Craske et al., 1995; Klosko et al., 1990) are effective treatments for panic disorder that have included cognitive restructuring as a major component. These results suggest that cognitive restructuring is an effective treatment component for panic disorder.

Feared fantasy technique.

The feared fantasy technique is a cognitive intervention designed to identify and change catastrophic beliefs or attitudes, in this case, related to the consequences of panic attacks. The goal of this technique is to have the individual realize that what he or she fears most is highly unlikely to happen. This is accomplished by confronting and exposing the individual to his or her worst fears by asking the individual to fantasize the worst possible experience, and then role play this feared fantasy with the therapist. The purpose is to identify ultimate fears that fuel the panic cycle. Subsequently, patient and clinician may reality test those fears as to their likelihood to occur as a result of panic symptoms and the patient's ability to cope. For example, a clinician might query a patient whose final fear is "dying and leaving my family to starve" by asking "How much do you believe you'll die and leave your family to starve because you have a panic

attack? How many times have you had panic attacks? How many times did you actually have a heart attack? How many times did you actually die?' Then, to challenge underlying helplessness schema, "Eventually, we all die. How can you prepare so that your family won't starve if you die prematurely?" An added technique, externalization of voices, involves the therapist and client switching roles, with the client learning how to talk back to their fears with more rational cognitions (Burns, 1999).

Exposure.

Exposure interventions involve therapeutic techniques that expose an individual with anxiety to an anxiety-provoking stimulus. These interventions have been included as components in effective treatments for panic disorder (Aaronson et al., 2008; Barlow et al., 2000; Clark et al., 1994, 1999; Craske et al., 1995; Klosko et al., 1990). The intention of exposure interventions is to habituate the client to his or her anxiety and teach him or her that the anxiety-provoking stimuli are not dangerous, eventually extinguishing the panic response (Levitt et al., 2001).

Given the extremely distressing nature of panic attacks, individuals tend to avoid stimuli and environments associated with their panic attacks. Accordingly, because exposure can seem so counterintuitive to panic patients, it can often be helpful to start exposure towards the middle or end of treatment, after the clients have received the psychoeducation needed to understand the nature of their anxiety and panic and coping skills, as well as the value of exposure to feared stimuli. This will allow them to more readily remain in their particular anxiety-provoking situations (Levitt et al., 2001). Two prominent types of exposure in effectively treating panic disorder include in vivo and

interoceptive exposures (Aaronson et al., 2008; Barlow et al., 2000; Clark et al., 1994, 1999; Craske et al., 1995; Klosko et al., 1990).

In vivo exposure. In vivo exposure is considered to be a behavioral intervention because it encourages the clients to physically enter into situations that expose them to anxiety-provoking stimuli. This intervention has empirical support in the treatment of panic disorder, PTSD, and specific phobias (Beidel, Frueh, Uhde, Wong, & Mentrikoski, 2011; Michaliszyn, Marchand, Bouchard, Martel, & Poirier-Bisson, 2010; Michelson & Mavissakalian, 1985). This treatment component includes the introduction of the client to anxiety-provoking situations in an organized and controlled manner. This is often done in a graded or hierarchical fashion, but can also be completed in quick, intensive, and concentrated exercises. To accomplish this, clients are instructed to remain in the anxiety-arousing situation until their anxiety reduces significantly. In this process, the anxiety will naturally reduce over time as the client remains in the anxiety-provoking situation (Levitt et al., 2001).

Michelson and Mavissakalian (1985) investigated the effectiveness of in vivo exposure as a stand-alone treatment for panic disorder. Sixty-two participants who met *DSM–III* criteria for agoraphobia with panic attacks were randomly assigned to one of four conditions: prolonged in vivo exposure, medication management via imipramine, a combination of prolonged in vivo exposure and medication management, and a control group. Results of this study indicated that prolonged in vivo exposure alone is effective in reducing the psychophysiological symptoms of panic, including mean and peak heart rates. The other treatment conditions did not result in this significant reduction in psychophysiological symptoms of panic. This study did not measure the other domains

of panic, including cognitive, affective, and behavioral (Michelson & Mavissakalian, 1985).

In vivo exposure was compared to cognitive therapy in treating panic disorder. In this study, 48 participants who met *DSM–III–R* criteria for panic disorder (with 44 also meeting criteria for agoraphobia) were randomly assigned to one of four conditions: in vivo exposure, cognitive therapy, combined in vivo exposure and cognitive therapy, and a control group that received no treatment. While the three treatment conditions were all equally effective in treating panic disorder and were superior when compared to the control group, the in vivo exposure alone condition was significantly superior to the cognitive therapy group in treating generalized phobia, agoraphobic cognition, and panic coping self-efficacy. Also of note, the level of agoraphobia impacted the effectiveness of treatment, as 94% of participants who were designated as having low levels of agoraphobia were panic free at the end of treatment, while only 52% of those who were designated as high levels of agoraphobia were panic free (Williams & Falbo, 1996)

Arch and Craske (2011) noted three important points in treating panic disorder with in vivo exposure. First, experiencing anxiety during exposures, tolerating any fear, and learning that the symptoms of panic can be fully experienced without danger are fundamental to ensuring that the exposures are a successful component of treatment. Second, varying the environment of exposures helps to generalize the learning gained through interoceptive and in vivo exposures. Finally, according to Arch and Craske, an "emphasis on immediate and short-term fear reduction and the controllability of panic symptoms" can contribute "to the treatment's failure to result in long-term learning" (Arch & Craske, 2011, p. 310). In summary, the goal is not to control panic, rather it is

to learn that panic and anxiety can be experienced without danger and that this can and should happen in a number of environments.

Interoceptive exposure. Interoceptive conditioning follows a classical conditioning paradigm in which the conditioned stimulus associated with panic is an internal bodily sensation. Interoceptive conditioning of the initial symptoms of panic attacks (such as chest pain or increased heart rate) with increased anxiety can lead to the development of panic disorder (De Cort, Griez, Büchler, & Schruers, 2012). Consequently, interoceptive exposure to those same stimuli can play a role in the treatment of panic disorder. Interoceptive exposure can be described as recurrent inducement and experience of the feared internal physiological sensations of sympathetic nervous system arousal experienced during panic attacks in order to demonstrate that the symptoms are not dangerous or threatening (Schmidt & Trakowski, 2004; Stewart & Watt, 2008). These exposures are completed via exercises designed to replicate the feared sensations, such as breathing through a straw, hyperventilation (breathing rapidly), breath holding, spinning, using a tongue depressor, doing jumping jacks, running, and head shaking (Antony, Ledley, Liss, & Swinson, 2006; Kiyoe et al., 2006; Schmidt & Trakowski, 2004).

Using the conceptualization that panic disorder is a fear of internal feelings, Griez and van Den Hout (1986) attempted to treat panic disorder with exposure to these interoceptive sensations. Using a cross-over design, 14 participants were randomly assigned to receive either carbon dioxide therapy or propranolol, a beta-blocker, as a control treatment. The carbon dioxide therapy was designed to elicit interoceptive

stimuli associated with panic, while the propanol kept the participants from experiencing these symptoms.

Interoceptive exposure (carbon dioxide treatment) resulted in statistically significant improvements in fear of autonomic sensations, state anxiety, trait anxiety, agoraphobic anxiety, agoraphobic avoidance, and frequency of panic attacks, whereas the control treatment only improved state, trait, and agoraphobic anxiety. Furthermore, the interoceptive exposure improvements were superior to the propranolol control treatment across all measures, except trait anxiety. The superiority of interoceptive exposure was especially pronounced for fear of autonomic sensations and agoraphobic avoidance (Griez & van Den Hout, 1986). Relevant to the current study, it is interesting to note that agoraphobic anxiety was effectively treated by targeting interoceptive conditioning and, by extension, anxiogenic cognitions, in the absence of in vivo exposure.

Similar results were found with other studies using carbon dioxide as an interoceptive exposure (Beck, Shipherd, & Zebb, 1996; van den Hout, van der Molen, Griez, Lousberg, & Nansen, 1987). Additionally, prolonged hyperventilation as an interoceptive exposure was found to be effective in reducing sensations of panic, as compared to a control condition with normal ventilation (van den Hout, de Jong, Zandbergen, & Merckelbach, 1990). In a 12-session treatment with 69 participants, interoceptive exposure was found to be as effective as cognitive therapy in treating panic disorder without agoraphobia, when measured at posttreatment and 1- and 6-month follow-up (Arntz, 2002). Even interoceptive exposure applied via virtual reality has been shown to be effective in significantly reducing symptoms of panic disorder (Pérez-Ara et al., 2010).

Interoceptive exposure has also been studied as a component of a larger treatment protocol for panic disorder. Interoceptive exposure was compared to breathing retraining as a third treatment component in combination with in vivo exposure and cognitive restructuring. Two sessions of either breathing retraining or interoceptive exposure were added to 12 weekly sessions of in vivo exposure and cognitive restructuring. While both groups improved on most of the more than two dozen measures of anxiety and panic-related distress, the interoceptive exposure group had significantly superior results in panic attack frequency, panic attack severity, agoraphobia fear, social fear, and overall level of impairment. This study indicates that interoceptive exposure is an effective treatment component in a larger treatment protocol (Craske et al., 1997). However, it is theorized that prolonged interoceptive exposure may not be a necessary component in treating panic disorder. For example, many interventions have successfully treated panic disorder without including interoceptive exposure (Beck et al., 1994; Bouchard et al., 1996; Öst et al., 1993; Öst & Westling, 1995; Salkovskis et al., 1991). Instead, it is theorized that cognitive restructuring breaks the panic cycle. Specifically, cognitive restructuring allows people to reframe interoceptive stimuli as a normal variation of physiological arousal. This may similarly reduce the catastrophic misinterpretation of symptoms and subsequent further hyperarousal (Clark, 1986).

Cognitive rehearsal. Cognitive rehearsal is the process of having a client identify potentially problematic situations and mentally work through how they might use coping skills to deal with these situations. This mental rehearsal helps the client deal with the problematic situations more effectively by practicing the steps needed to reduce anxiety. The identification of and solution for potential obstacles can also be a part of the

rehearsal. This technique is typically taught after the client has already learned the coping skills that he or she will need to use (Northam, 2000; Wright, Basco, & Thase, 2006).

Previous researchers have determined that cognitive rehearsal is somewhat successful in reducing avoidance in snake phobia (Barrios, Somervill, Henke, & Merritt, 1981). Additionally, cognitive rehearsal was found to be the most efficacious component of a systematic desensitization procedure to reduce stress responses (Folkins, Lawson, & Opton, 1968). Furthermore, results from a meta-analysis determined that mental practice significantly improves task performance (Driskell, Copper, & Moran, 1994). Cognitive rehearsal is also theorized to increase commitment to and completion of therapeutic homework (Freeman & Rosenfield, 2002). Collectively, cognitive rehearsal appears to be an effective tool to tie together previously learned coping skills to increase the probability of successful implementation of coping responses.

Hypotheses

Research Questions

1. Can the frequency and severity of panic be reduced after the first treatment session and be maintained with three additional booster sessions?
2. Will treatment gains be maintained at a three-month follow-up?

The hypotheses for the study were:

1. The frequency and severity of panic attacks would be significantly reduced after the first treatment session of the proposed protocol (reduction of 30% or more).
2. Additional cognitive, behavioral, and physiological symptoms of panic-related anxiety would be reduced at posttreatment and at 3-month follow-up, as

measured by the Beck Anxiety Inventory (BAI) and the Panic Disorder Severity Scale–Self-Report (PDSS–SR; with significance operationally defined as a reduction of 30% or more).

3. Global Assessment of Functioning (GAF) scores would be significantly improved at posttreatment and 3-month follow-up, compared to baseline GAF (operationally defined as an increase of 10 or more; Eisen, Ranganathan, Seal, & Spiro, 2007).
4. The frequency of cognitive distortions would be significantly decreased, as measured by the Inventory of Cognitive Distortions (ICD) at posttreatment and 3-month follow-up, compared to ICD scores at baseline (operationally defined as a reduction of 30% or more).
5. Reduction in panic attack frequency and severity would be maintained at 3-month follow-up.

Thus, it was expected that the four-session protocol would be effective in reducing the frequency and severity of panic attacks after the first session and that these gains would be maintained, operationalized as scores on the BAI, PDSS–SR, ICD, and GAF, as well as in the panic log.

Method

This study used a single-case experimental ABA design with follow-up, where a reversal was not expected, due to the maintenance of positive effects. This design was chosen because it allows for continuous assessment to create stability of information. The use of continuous assessment in this design reduced the impact of testing as a threat to internal validity. Additionally, conducting treatment in the "real world" of an outpatient clinic reduced threats to external validity (Kazdin, 1982).

Participants

Participants for this study were recruited from outpatient mental health facilities located in Southeastern Pennsylvania. Three facilities participated in recruitment. These clinics mainly treat adults and adolescents with anxiety and mood disorders. While many potential participants were screened across the three clinics, only one participant met criteria and was retained for the study.

Inclusion/Exclusion criteria

Inclusion criteria included adult outpatients applying for treatment at three clinics in the Southeastern Pennsylvania region between the ages of 18 and 65 with a primary or secondary diagnosis of panic disorder. Initial diagnoses were be made by intake clinicians at the respective treatment centers using the Structured Clinical Interview for the *DSM–IV–TR* - Clinician Version (SCID–CV; First, Spitzer, Gibbon, & Williams, 1996). Applicable diagnoses, as detailed in the *DSM–IV–TR*, were 300.01 panic disorder without agoraphobia and 300.21 panic disorder with agoraphobia. Participants were required to obtain physician medical clearance to rule out general medical etiology.

Excluded were patients with active psychosis, defined as reports of current delusions or hallucinations via initial screening or self-report during the SCID–CV interview, current suicidal or homicidal ideation per client report, intellectual/developmental disabilities, as demonstrated through patient or external report of a previous diagnosis, severe agoraphobic avoidance, as measured by the Mobility Inventory for Agoraphobia (MIA; Chambless, Caputo, Jasin, Gracely, & Williams, 1985), an experience of release effect during relaxation training (becoming paradoxically more anxious), severe depression, as measured by the Beck Depression Inventory–Second Edition (BDI–II; Beck, Steer, & Brown, 1996), current substance abuse or dependence, current use of anxiolytic medication, or a medical condition that accounted for the symptoms of panic disorder, as indicated by the client's physician report required for medical clearance.

The rationale for these exclusion criteria were: Active psychosis, current suicidal or homicidal ideation, an experience of release effect during relaxation training, intellectual/developmental disabilities, severe agoraphobic avoidance, current substance abuse or dependence, and severe depression can interfere with the treatment for panic disorder and require additional specific treatment outside the scope of this brief intervention. Anxiolytic use can interfere with arousal, can be employed as a form of avoidance, and can interfere with both exposure and learning coping skills. A medical condition that accounts for the symptoms of panic disorder should be ruled out in order to rule out medical etiology and to ensure an accurate diagnosis of panic disorder. Individuals were also excluded from the study if they were on disability for a mental health disorder to eliminate possible malingering and other confounds, which could

interfere with the efficacy of the treatment. It was planned, a priori, that patients excluded from the study or refusing to participate, for any reason, would be offered treatment as usual at the clinic of the patients' choice or referred to another treatment facility.

Recruitment at Clinic A

Participants were recruited following their intake at Clinic A. First, as in the case of all new, prospective clients, the individual called Clinic A, where the graduate assistant at the front desk then conducted a telephone screening, including assessment for the presence of panic attacks. The graduate assistant then gave the individual general brief informed consent and advised the individual that he or she could make a decision to participate at their intake session. A clinician then called the individual to schedule a study initiation visit. During the initial study visit, the individual received full informed consent. In addition to the normal intake process with the clinician that screened for inclusion/exclusion criteria, the client was screened with the anxiety/panic section of the SCID–CV.

If all criteria were met, the individual began participation in the study. This included completing the initial baseline measures indicated below. Participants were also asked to obtain medical clearance from a physician to participate in this study to rule out general medical conditions as causes for their panic symptoms.

Recruitment at Clinic B

Participants were recruited following their intake at Clinic B. First, as in the case of all new, prospective clients, the individual called Clinic B, where the administrative assistant at the front desk then conducted a telephone screening, including assessment for

the presence of panic attacks. The administrative assistant then assigned the individual to a doctoral level clinician participating in this study and scheduled a study initiation visit with that clinician during which the individual received full informed consent. After the normal intake process with the clinician, the individual returned to Clinic B to complete measures that screened for inclusion/exclusion criteria, including the anxiety/panic section of the SCID–CV and the initial baseline measures indicated below. Participants were also asked to obtain medical clearance from a physician to participate in this study to rule out general medical conditions as causes for their panic symptoms.

Recruitment at Clinic C

Participants (a) were referred by their current clinician at Clinic C or (b) called the managed care specialists (administrative assistants) at Clinic C, who screened for the presence of panic attacks. The current clinician or managed care specialist gave individuals a general, brief informed consent and informed them that they could make a decision to participate at their study initiation session or later. The current clinician or managed care specialist assigned the individual to a clinician participating in this study and scheduled a visit with the clinician, regardless of the individual's willingness to participate in the study (no discrimination based on willingness). During the intake, the individual received full informed consent from the clinician. In addition to the normal intake process with the clinician who screened for inclusion/exclusion criteria, the individual was screened with the anxiety and panic sections of the SCID–CV and the initial baseline measures indicated below.

If all criteria were met, the individual was offered the chance to begin participation, either then or at a later date. After the individual signed the informed

consent and Health Insurance Portability and Accountability Act Authorization, he or she was able to begin the study. Participants were also asked to obtain medical clearance from a physician to participate in this study to rule out general medical conditions as causes for their panic symptoms.

Measures

The measures used in this study were the BAI, BDI–II, PDSS–SR, ICD, MIA, and SCID–CV. Participants additionally were requested to keep daily self-monitoring logs of information, including the nature of their panic attacks, medication taken, and alcohol and substance use.

Beck Anxiety Inventory.

The BAI (Beck, Epstein, Brown, & Steer, 1988) is a 21-item self-report inventory designed to measure the severity of symptoms of anxiety. Each item is rated on a 4-point scale (0 to 3). The total score is obtained by adding the item scores together, with the possible total ranging from 0 to 63. It has strong established psychometric properties, including a high internal consistency (alpha = .92) and 1-week test-retest reliability of $r(81) = .75$. Additionally, the BAI has good concurrent and discriminant validity. It was able to discriminate anxious groups from nonanxious groups in a previous study. Furthermore, it had a moderate correlation with the revised Hamilton Anxiety Rating Scale, $r(150) = .51$ (Beck et al., 1988).

The current study selected the BAI as an outcome measure for panic-related anxiety because of its strong ability to assess panic symptomology (Cox & Cohen, 1996; Leyfer, Ruberg, & Woodruff-Borden, 2006). Specifically, it also has demonstrated particularly excellent psychometric properties in a panic disorder population. This

included high internal consistency, 5-week test-retest reliability, and convergent and divergent validity (de Beurs, Wilson, Chambless, Goldstein, & Feske, 1997). Additionally, the BAI has been used as an outcome measure in many previous studies of panic disorder treatments (Deacon & Abramowitz, 2006; Kim et al., 2010; Lamplugh, Berle, Milicevic, & Starcevic, 2008).

Beck Depression Inventory.

The BDI–II (Beck et al., 1996) is a 21-item self-report inventory designed to measure the severity of symptoms of depression. Each item is rated on a 4-point scale (0 to 3). The total score is determined by adding the item scores together, with the possible total ranging from 0 to 63. The BDI–II has high internal consistency (alpha = .91) and 1-week test-retest reliability (r = .93; Beck et al., 1996; Dozois, Dobson, & Ahnberg, 1998). Additionally, it has demonstrated a good correlation with the Hamilton Depression Rating Scale (r = .71; Beck et al., 1996). The BDI–II was used as a measure to screen for severe depression, which could confound treatment, and to assess progress. Initial BDI–II scores of 29 or above excluded patients from participation in this study.

Panic Disorder Severity Scale–Self-Report.

The PDSS–SR (Houck, Spiegel, Shear, & Rucci, 2002) is a 7-item self-report rating scale designed to measure the severity of symptoms of panic disorder within the past week. Each item is rated on a 5 point scale (0 to 4). The total score is calculated by adding the item scores together, with the possible total ranging from 0 to 28. The mean score among a psychiatric outpatient population was found to be 9 (Houck et al., 2002). The cutoff for the presence of panic disorder is a score of 8, while 13 is the score used to discriminate between mild and severe panic symptoms (Shear et al., 2001). The PDSS–

SR measures panic frequency, distress during panic attacks, anticipatory anxiety, agoraphobic fear/avoidance, bodily sensation fear/avoidance, and occupational and social functioning impairment (Houck et al., 2002).

The PDSS–SR has demonstrated strong psychometric properties, including a high internal consistency (alpha = .92) and 1-day test-retest reliability (r = .83). Furthermore, it a high established correlation with the clinician interview version of the PDSS (intraclass correlation coefficient of .81). Additionally, the PDSS–SR displayed sensitivity to changes in panic disorder symptoms in response to treatment similar to the PDSS (Houck et al., 2002). The PDSS–SR has been used as an outcome measure in many previous studies of panic disorder treatments (Aaronson et al., 2008; Austin, Sumbundu, Lykke, & Oestrich, 2008; Bitran, Morissette, Spiegel, & Barlow, 2008; Meulenbeek et al., 2010; Nuthall & Townend, 2007; Titov, Andrews, Johnston, Robinson, & Spence, 2010). Therefore, the current study selected the PDSS–SR as an outcome measure for symptoms of panic disorder.

Inventory of Cognitive Distortions.

The ICD (Yurica & DiTomasso, 2001) is a self-report inventory designed to measure the frequency and severity of cognitive distortions. It contains 69 items rated on a 5-point Likert scale (1 to 5). Total scores are calculated by adding together the sum of all item scores, with possible scores ranging from 69 to 345. The ICD demonstrated a high internal consistency (alpha = .98) and a high 5-week test-retest reliability of .998 (n = 28, $p < .0001$). It also had high content validity and high criterion validity in its ability to distinguish a psychiatric outpatient population from a non-patient control group (F = 15.2, df = 169, $p < .0001$). Additionally, the ICD is highly correlated to the BAI and

BDI–II, indicating that cognitive distortions likely underlie anxiety disorders, such as panic disorder (Yurica, 2002).

The psychometrics of the ICD were also validated by Rosenfield (2004), which indicated a high internal consistency (alpha = .97). Additionally, this investigation found a high correlation between ICD scores and number and severity of both axis I and axis II psychological disorders, as measured by the Millon Clinical Multiaxial Inventory–Third Edition (MCMI–III; Rosenfield, 2004). The same high internal consistency (alpha = .97) was found again by Uhl (2007) in a study with an outpatient medical population. Furthermore, the ICD has been used in previous studies to assess changes in cognitive distortions for clients in treatment of mental health conditions (Good, 2010; Tate, 2006).

Mobility Inventory for Agoraphobia.

The MIA (Chambless et al., 1985) is a self-report inventory designed to measure agoraphobic avoidance and frequency of panic attacks. It contains 27 items rated on a 5-point Likert scale (1 to 5). Twenty-six situations are rated for avoidance, both when accompanied by another person and when alone. Scores are calculated for each condition by averaging all item scores within the respective condition, resulting in two total scores: avoidance accompanied and avoidance alone (Chambless et al., 1985).

Based on a meta-analysis of psychometric testing, the MIA demonstrated a high internal consistency (alpha = .93). Test-retest reliabilities over an 8-day span for avoidance accompanied and avoidance alone were high (.86 and .90, respectively). The test-retest reliabilities were slightly lower over a 31-day span (.75 and .89, respectively). Both scales were also found to have good convergent validity when compared to other measures of agoraphobia. The authors of this meta-analysis suggest that, based on the

findings, the MIA could be a useful tool to screen for agoraphobia in research studies (Chambless et al., 2011).

Structured Clinical Interview for the *DSM–IV–TR*.

The SCID–CV (First et al., 1996) is a structured clinical inventory used to assess axis I clinical disorders according to the criteria listed in the *DSM–IV*. The SCID–CV has both good interrater (alpha = .67) and test-retest (alpha = .65) reliability in diagnosing panic disorder (Lobbestael, Leurgans, & Arntz, 2011; Zanarini et al., 2000). Additionally, because the wording of the SCID–CV is designed to match the *DSM–IV*, and it has good consistency with the CIDI in diagnosing anxiety disorders, the SCID–CV is considered to have suitable content and construct validity (Hunsley & Mash, 2008; Kessler et al., 2004).

Panic Attack Record.

The Panic Attack Record (Barlow & Craske, 2007) was used to record each time the client had a panic attack. The client records aspects of the panic attack, including the date, the time it began, triggers, whether it was expected or unexpected, the maximum fear reached on a 0 to 10 scale, with 10 being the most severe, all symptoms present to at least a mild degree, and any associated thoughts and behaviors.

Therapist

The therapist was an advanced doctoral student (5th year). Additionally, this clinician was trained in the techniques that are part of the treatment protocol by the principal investigator, a licensed psychologist, who has previously published on the topic.

Procedure

The procedure for this study began with initial recruitment of potential participants via the intake process at the three aforementioned outpatient clinics. Once potential participants were given full informed consent and identified by the intake clinician as fulfilling all inclusion and exclusion criteria, the participants completed initial baseline measures: the BAI, BDI–II, PDSS–SR, ICD, and MIA. The participants then began daily recordings of frequency and severity of panic attacks using the self-report Panic Attack Record, which continued throughout treatment. Immediately preceding the first treatment session (approximately 1 week after intake), the participants again completed all baseline measures in order to identify any changes that may have occurred during the self-monitoring period. The clinician noted the client's GAF score before the treatment began.

This intervention is largely based on Barlow's protocol (Craske & Barlow, 2007), but accelerated and augmented with relaxation and other cognitive techniques. The first treatment session included psychoeducation, cognitive behavioral functional analysis, breathing retraining, imagery, autogenic training, release-only relaxation, differential relaxation, cognitive restructuring, and the feared fantasy technique. To ensure protocol adherence, a review of the first treatment session transcript was conducted by study collaborator Dr. Wendy Wild as a fidelity check.

Following this session, three more weekly sessions were scheduled with the participant with the purpose of reinforcing the coping skills and concepts learned in the first session, ensuring adherence, and identifying and dealing with obstacles to implementing the protocol and ancillary anxiety. The protocol for these sessions mainly

followed the Barlow protocol (Craske & Barlow, 2007) and was individualized for the participant based on the idiosyncratic difficulties experienced. The only additional CBT intervention used during this period was a discussion and worksheet on emotional reasoning, as it was an obstacle to this participant's use of her newly learned coping skills.

Furthermore, before each session, starting with the second session (approximately 1 week after the first session), all outcome measures were given to the client to complete, for a total of three data collection points during the active treatment phase of the study. Following the completion of treatment, all measures were completed weekly for 4 weeks. Additionally, the daily recordings of frequency and severity of panic attacks continued for 1 month following completion of treatment. Finally, a 3-month posttreatment follow-up of all measures was completed by the participant to determine if treatment effects were maintained.

Results

Data Analysis

Simulation modeling analysis (SMA) and visual analysis were used to evaluate the effectiveness of this treatment protocol.

Simulation modeling analysis.

SMA is a statistical program that accounts for autocorrelation in both parametric and nonparametric data. Autocorrelation occurs in data when each subsequent data point is at least partly influenced by the data point(s) that come(s) before it. Using SMA with autocorrelated data reduces the risk of Type I errors (Borckardt, Shaw, O'Neil, Nash, Murphy, & Moore, 2008). SMA is also applicable to data with relatively few observation points, but "requires a minimum of 10 –16 total observations in the data stream (i.e., 5– 8 per phase)" (Borckardt et al., 2008, p. 87).

SMA tests for significant level change (change in means) and slope change between two phases by processing 10,000 simulations of a data stream to determine the likelihood of the changes occurring randomly. Slope change is determined by a comparison to five different vector templates to determine best fit, as determined by the lowest *p*-value below the chosen significance level. SMA lends more statistical power to the findings of this study. Only the frequency and severity of panic attacks were intended to be evaluated via SMA. As SMA only allows the comparison of two consecutive phases, two analyses were completed: baseline vs. intervention and intervention vs. follow-up (Borckardt et al., 2008).

Visual analysis.

Any clinically significant treatment effects should be evident with visual analysis, supporting the argument that visual analysis could be used as the sole means of data interpretation (Baer, 1977; Michael, 1974; Parsonson & Baer 1978, 1986). The advantages of visual analysis include identifying conservative treatment effects and low error rates (Baer, 1977; Huitema, 1986).

The visual analyses include the change in means, the level of performance, and the trend in performance. The change in means compared the difference between the initial baseline mean and the intervention mean, as well as the initial baseline mean and the posttreatment mean. The level of performance compared the difference between the last data point in the initial baseline phase and the first data point in the intervention phase. The trend in performance compared the difference between the mean of the first half of the intervention data points and the mean of the second first half of the intervention data points. Each dependent variable was evaluated via visual analysis.

Protocol fidelity check.

A fidelity check of the first treatment session's transcript by an independent evaluator, Dr. Wendy Wild, used a 130-point checklist. According to this fidelity check, the therapist followed the protocol 100%.

Beck Anxiety Inventory.

The BAI was administered on a weekly basis throughout the course of the study. The weekly scores for the baseline, intervention, and posttreatment phases are indicated in Figure 1.

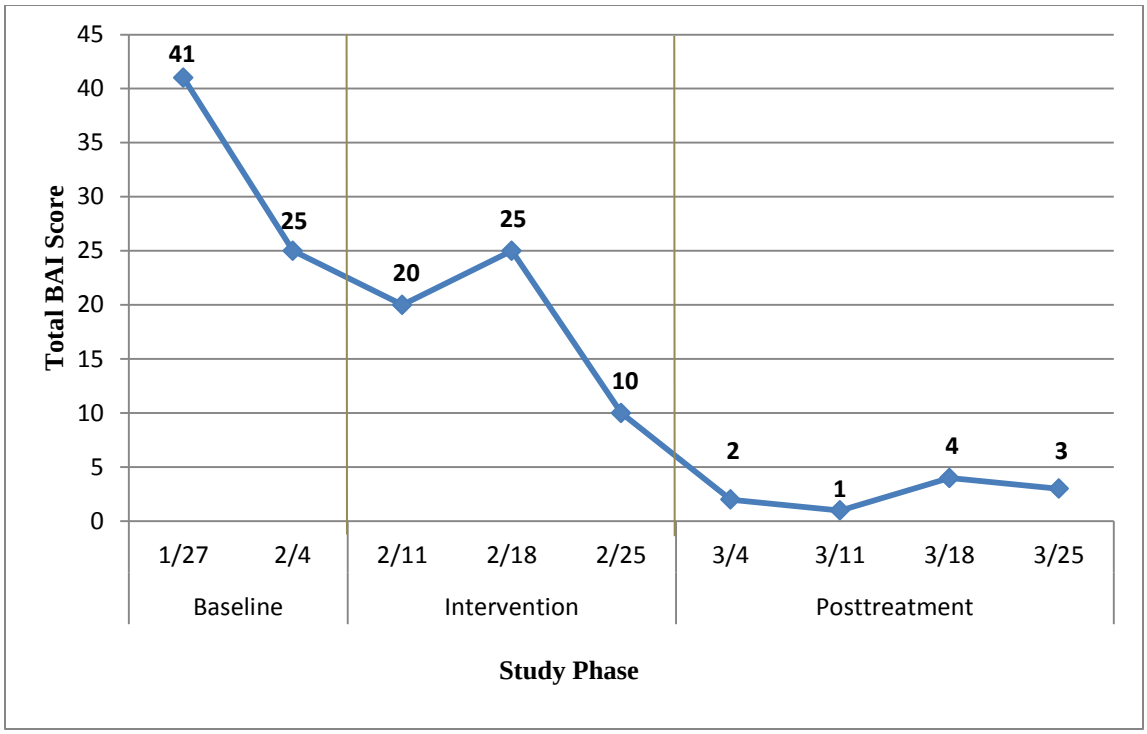

Figure 1. Changes in BAI scores.

As can be seen in the figure above, the subject's BAI score changed from severe (41) during intake to minimal (3) 1 month after the final treatment session. This 92.7% reduction in BAI scores indicates a significant reduction in anxiety symptom severity. A more conservative analysis using mean scores for each phase still found a 92.4% reduction from 33.0 in the baseline phase to 2.5 in the posttreatment phase. This included a decrease of 44.5% from the baseline phase mean of 33.0 to the intervention phase mean of 18.3. The 86.3% reduction from the intervention mean of 18.3 to the posttreatment mean of 2.5 indicates that symptoms continued to improve after treatment ended as the subject consolidated and practiced her learned coping skills.

The level of performance compared the difference between the last data point in the initial baseline phase and the first data point in the intervention phase. The last BAI score in the baseline phase was 25. When compared to the score of 20 at the beginning

of the intervention phase, there is a decrease of 20.0%. The largest percentage decrease in BAI scores (80.0%) occurred between the final intervention data point (10) and the first posttreatment data point (2). The largest decrease in BAI raw scores was 16 points between the first data point at intake (41) and the second data point (25), just before the start of the first treatment session.

The trend in performance compares the difference between the mean of the first half of the intervention data points (22.5) and the mean of the second half of the intervention data points (17.5). The 22.2% reduction between these two means suggests that the intervention was responsible for the reduction in anxiety symptom severity.

Finally, while the subject's BAI score started at 41, well above the cutoff to determine severe anxiety symptoms (scores above 25), it decreased into the moderate range by the first treatment session (25) and remained there until the final treatment session, when it decreased to the mild range (10). More importantly, her scores decreased to below the critical cutoff used to discriminate for the presence of anxiety symptoms (scores below 8) by the first posttreatment data point and remained there through the rest of the posttreatment phase, with a 3-month follow-up score of 4 on the BAI (Beck et al., 1988).

Panic Disorder Severity Scale–Self-Report.

The PDSS–SR was administered on a weekly basis throughout the course of the study. The weekly scores and the means for the baseline, intervention, and posttreatment phases are indicated in Figure 2.

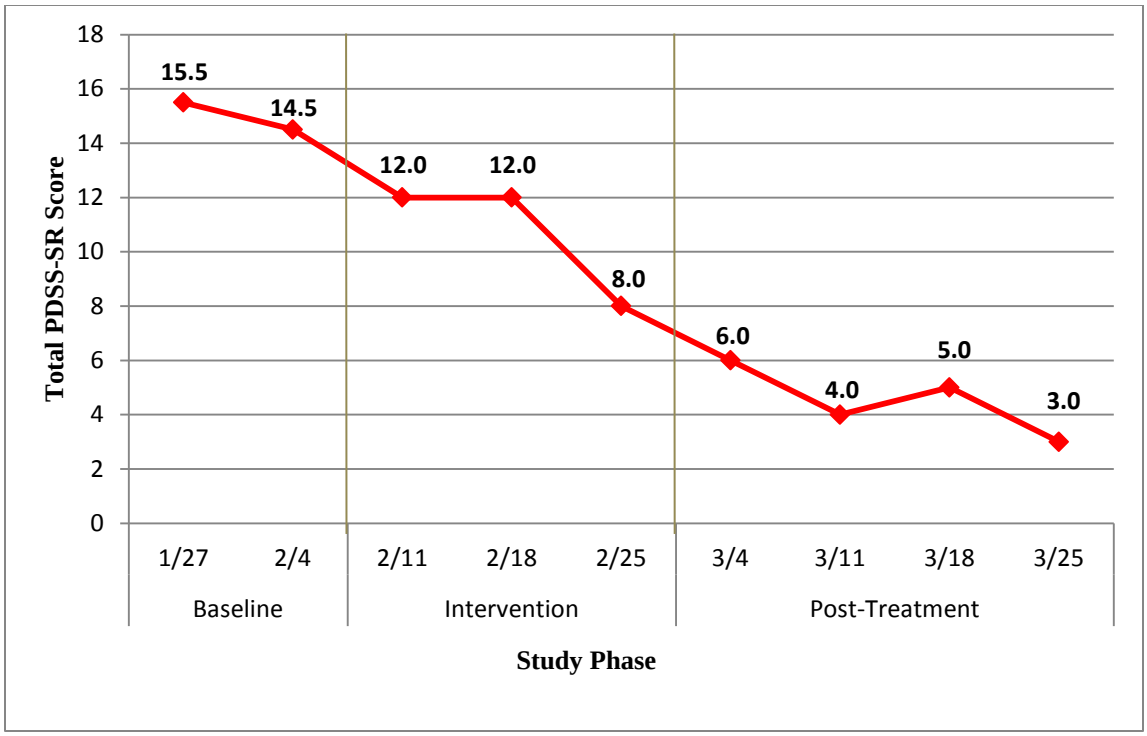

Figure 2. Changes in PDSS–SR scores.

The study responsible for the development of the PDSS–SR defined treatment response as a reduction of at least 40.0% from baseline levels on both the PDSS and PDSS–SR (Aaronson et al., 2008). In the current study, the PDSS–SR identified a decrease in the severity of panic symptoms from an initial baseline score of 15.5 to 3.0 one month after the final treatment session, a reduction of 80.6%. A more conservative estimate, using the mean scores in each phase, still identified a 70.0% reduction from 15.0 in the baseline phase to 4.5 in the posttreatment phase. This included a decrease of 29.0% from the baseline phase mean of 15.0 to the intervention phase mean of 10.7. The 57.9% reduction from the intervention mean of 10.7 to the posttreatment mean of 4.5 indicates that symptoms continued to improve after treatment concluded as the subject consolidated and practiced her learned coping skills.

The level of performance compared the difference between the last data point in the initial baseline phase and the first data point in the intervention phase. The last PDSS–SR score in the baseline phase was 14.5. When compared to the score of 12.0 at the beginning of the intervention phase, there is a decrease of 17.2%. The largest decrease in PDSS–SR raw scores (4.0) and percentage (33.3%) occurred between the final two intervention data points (12 and 8, respectively).

The trend in performance compares the difference between the mean of the first half of the intervention data points (12) and the mean of the second half of the intervention data points (10). The 16.7% reduction between these two means suggests that the intervention was responsible for the reduction in panic symptom severity.

Finally, while the subject's initial PDSS–SR score was 15.5, above the cutoff to determine severe panic symptoms (score of 13.0), it decreased into the mild range by the first intervention data point. More importantly, her scores decreased to the critical cutoff used to discriminate for the presence of panic disorder (score of 8.0) by the final session. Her scores then decreased and remained below this critical cutoff for the entirety of the posttreatment phase, with a 3-month follow-up score of 1.0 on the PDSS–SR (Shear et al., 2001).

Inventory of Cognitive Distortions.

The ICD was also administered on a weekly basis throughout the course of the study. The weekly scores and the means for the baseline, intervention, and posttreatment phases are indicated in Figure 3.

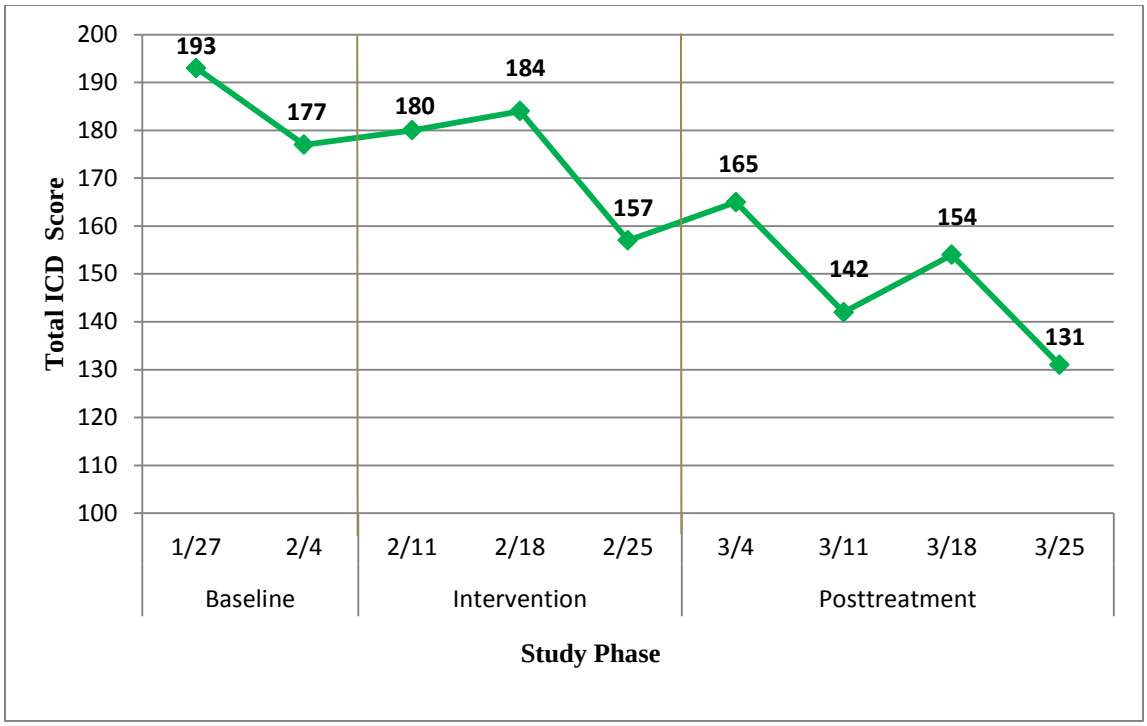

Figure 3. Changes in ICD scores.

ICD scores decreased from an initial intake score of 193 to a final posttreatment data point score of 131. This 32.1% reduction in ICD scores indicates a reduction in the frequency and severity of cognitive distortions. A more conservative analysis using mean scores for each phase identified a 20.0% reduction from 185.0 in the baseline phase to 148.0 in the posttreatment phase. This included a decrease of 6.1% from the baseline phase mean of 185.0 to the intervention phase mean of 173.7. The 14.8% reduction from the intervention mean of 173.7 to the posttreatment mean of 148.0 indicates that the frequency and severity of cognitive distortions continued to improve after the conclusion of treatment.

The level of performance compared the difference between the last data point in the initial baseline phase and the first data point in the intervention phase. The last ICD score in the baseline phase (177.0) was actually lower than the score taken at the

beginning of the intervention phase (180.0). However, this 1.7% increase in ICD scores is only temporary, as the scores decreased to 157 by the end of the intervention phase. The largest percentage decrease in ICD scores (14.9%) occurred between the final two posttreatment data points (154 to 131). The largest decrease in ICD raw scores was 27 points between the final two intervention data points (184 to 157).

The trend in performance compares the difference between the mean of the first half of the intervention data points (182.0) and the mean of the second half of the intervention data points (170.5). The 6.3% reduction between these two means suggests that the intervention was responsible for the reduction in frequency and severity of cognitive distortions.

Finally, while there was a 32.1% reduction from the intake to 1 month posttreatment, the ICD score decreased 51.1% from the last posttreatment data point of 131.0 to the 3-month follow-up score of 64. This represents a 67.0% reduction in ICD scores from the initial intake score of 194.

Frequency of panic attacks.

Daily measurements of the frequency of panic attacks were completed by the subject starting from the intake session (1 week preceding the first treatment session), indicated in Figure 4. During this week, the subject recorded eight panic attacks. The 3 weeks of measurements during the intervention phase indicated three, one, and one panic attack(s), respectively. All 4 weeks of measurement during the posttreatment phase indicated a complete absence of panic attacks.

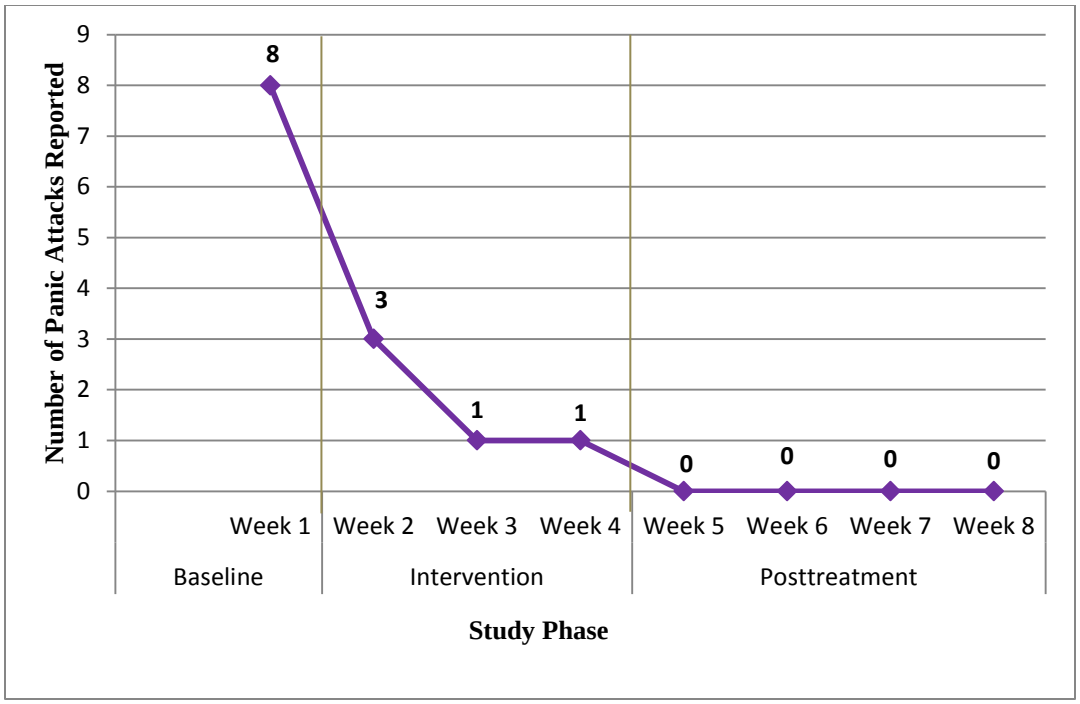

Figure 4. Weekly number of panic attacks.

SMA was used for two comparisons of the daily recordings of panic attack frequency: baseline vs. intervention and intervention vs. posttreatment. There was no baseline vs. posttreatment comparison because SMA requires contiguous observation points. The daily recordings of the frequency of panic attacks for the baseline and intervention phases, with means score for each phase, are indicated in Figure 5.

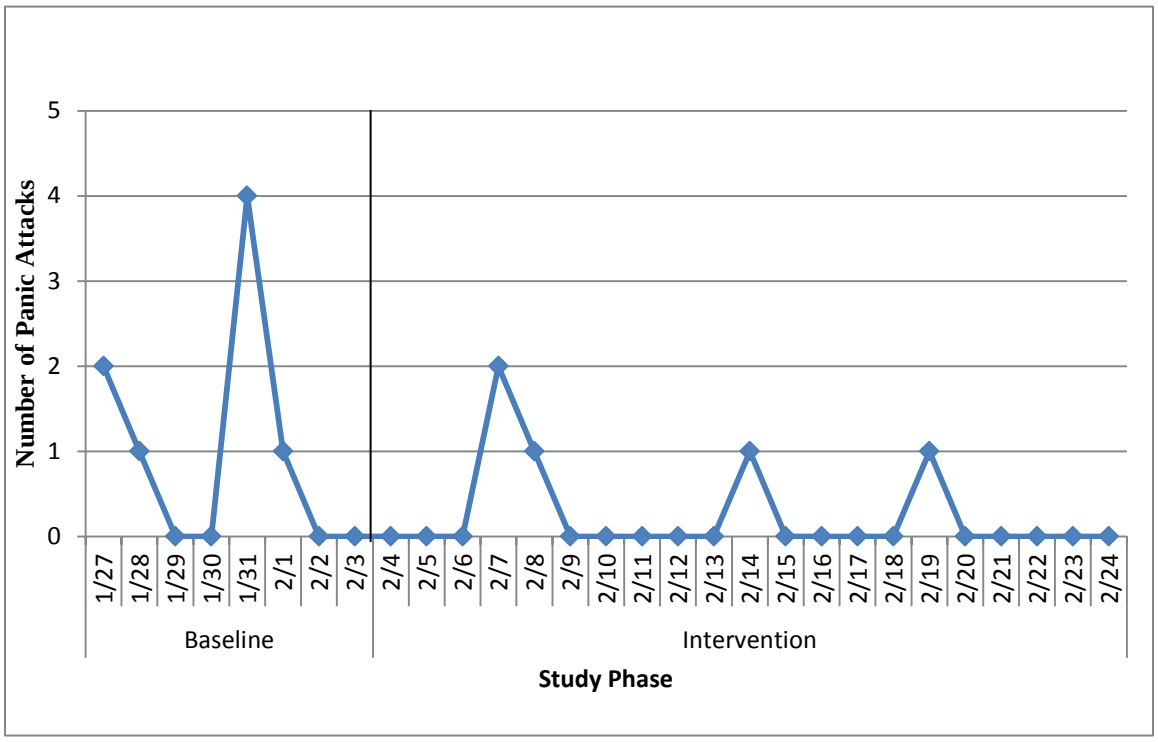

Figure 5. Daily number of panic attacks: baseline vs. intervention.

The test for level change did not produce any significant differences in the means between the baseline and intervention phase (R = -0.381, p = 0.0690). Despite the nonsignificant findings, this change does approach significance, indicating that the trend is in the expected direction (positive treatment outcome). Testing for slope change indicated only vector 3 met significance (R = -0.426, p = 0.0341). This significant negative correlation indicates that the frequency of panic attacks was decreasing during baseline and relatively flat during the intervention phase.

The daily recordings of the frequency of panic attacks for the intervention and posttreatment phases, with means score for each phase, are indicated in Figure 6.

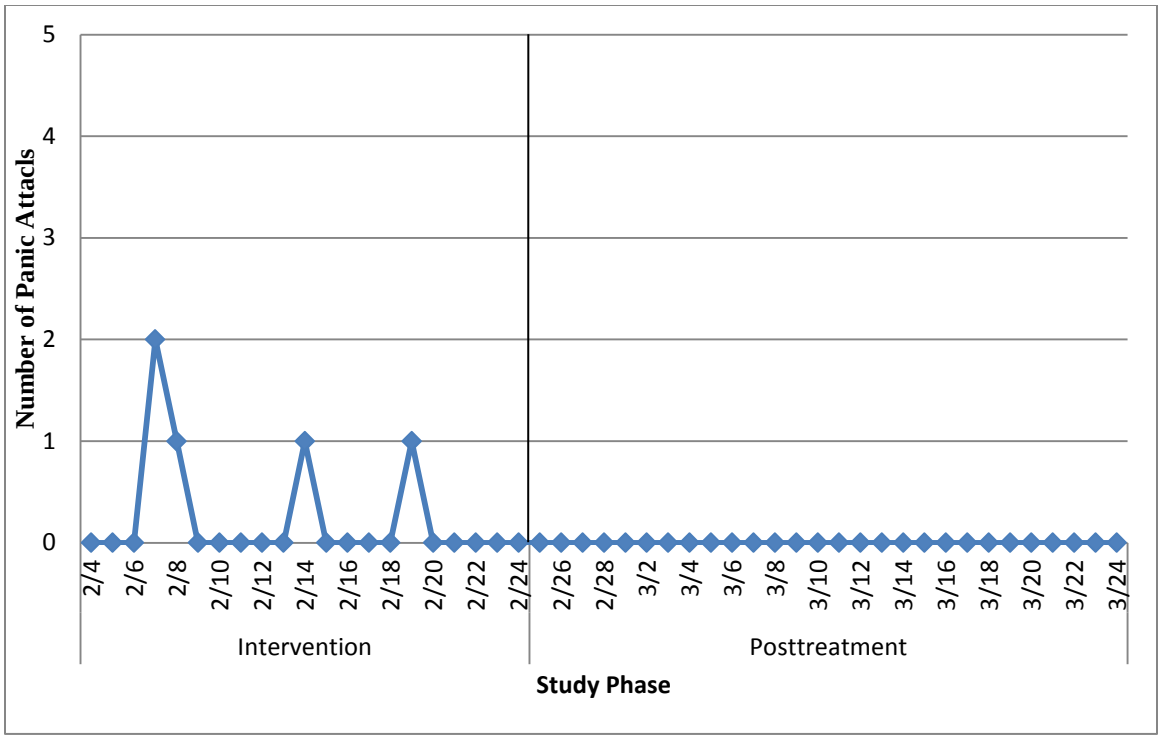

Figure 6. Daily number of panic attacks: intervention vs. posttreatment.

The test for level change in this second analysis also did not identify any significant differences in the means between the intervention and posttreatment phase (R = -0.324, p = 0.0743). However, in this case, this lack of any significant difference would be expected if the treatment gains were maintained during the posttreatment phase. Testing for slope change indicated that again only vector 3 was significant (R = -0.387, p = 0.0257). This significant negative correlation indicates that the frequency of panic was decreasing during the intervention phase and relatively flat during the posttreatment phase.

Because these slope vectors are all relative, the intervention phase can appear flat compared to the baseline phase, but decreasing compared to the posttreatment phase. Additionally, given the rather small magnitude of the correlations, it is possible that the aforementioned slope patterns are due to natural variability in the data.

Severity of panic attacks.

The quick reduction and elimination of panic attacks seen in this study do not allow for proper analysis via SMA, as there was not a sufficient number of observation points. However, 2 critical weeks in the study, before and after the first treatment session, have multiple panic attacks allowing for a comparison of means. The week immediately preceding the first treatment session had eight panic attacks, with a mean severity of 4.6 on a 1 to 10 interval scale. In the week following the first treatment session, three panic attacks with a mean severity of 2.8 were recorded. This 39.1% reduction was followed by 2 weeks with one panic attack each (severity of 7.0 and 3.0, respectively), before a posttreatment period of 4 consecutive weeks without a single panic attack.

Global Assessment of Functioning.

The subject's baseline GAF score was 51 at intake. This assessment was made due to the functional impairment caused by the panic, which kept the subject from engaging in the social and recreational activities that she used to enjoy. The more than occasional panic attacks provided rationale for a lower GAF score, but the client did not meet the criteria for severe impairment implied by a score of 50 or below (such as few friends and conflict with peers). At the end of the posttreatment period, the subject reported a complete absence of panic attacks for over 1 month and a return to engaging in activities that she previously avoided or endured with great distress. Based on this information, the GAF score was 80 at the end of the posttreatment period. However, this is a conservative score because there is evidence suggesting an even higher score (such as improved functioning in many areas and involvement in a wide range of activities).

Discussion

This study investigated the effectiveness of a four-session treatment protocol for panic disorder, which intended to greatly reduce the frequency and severity of panic attacks after the first session and to reduce residual panic-related anxiety with three additional booster sessions. This briefer treatment is designed to reduce attrition by providing significant relief after the first session so that it may also be more appealing to clients who are terrified of their panic symptoms, avoidant, hesitant to utilize protracted mental health care, or are worried about missing time from other obligations at work and home. Follow-ups were completed posttreatment to establish the long-term effectiveness of the treatment protocol at 3 months.

The first hypothesis stated that the frequency and severity of panic attacks would be significantly reduced after the first treatment session of the proposed treatment protocol (reduction of 30.0% or more). In the week before the first treatment session, the subject reported the presence of eight panic attacks. In the week after this session, there were only three panic attacks reported. This change represents a 62.5% reduction, more than double the 30.0% expected. The mean severity of these panic attacks (on a 1 to 10 interval scale) decreased from 4.6 before the first treatment session to 2.8 in the week following this first treatment session. This 39.1% reduction also exceeds the 30.0% expected for this hypothesis. Taken together, these results provide more than sufficient evidence needed to support this hypothesis.

The second hypothesis stated that the additional cognitive, behavioral, and physiological symptoms of panic-related anxiety would be reduced at posttreatment and at 3-month follow-up, as measured by the BAI and the PDSS–SR (with significance

operationally defined as a reduction of 30.0% or more). Symptoms of anxiety and panic as measured by the BAI and PDSS–SR declined throughout the study. In fact, the subject's BAI and PDSS–SR scores both decreased considerably from the first to last observation points. The subject's BAI scores plummeted 92.7% from severe (41) during intake to minimal (3.0) 1 month after the final treatment session. PDSS–SR scores decreased 80.6% from severe at intake (15.5) to a score indicating a lack of panic disorder (3.0) 1 month after the final treatment session, more than doubling the 40.0% reduction needed to determine treatment response on this measure (Aaronson et al., 2008). Even a more conservative estimate of change using phase means indicated drastic improvements in panic-related anxiety symptoms, as evidenced by 92.4% and 70.0% reductions in on the BAI and PDSS–SR, respectively. These results provide overwhelming evidence to support this hypothesis of significant reductions in cognitive, behavioral, and physiological symptoms of panic-related anxiety.

The fact that the largest decrease in BAI raw scores was between the first data point at intake (41) and the second data point (25) taken just before the start of the first treatment session may be due to both expectancy to treat and self-monitoring effects, as the subject had been instructed to begin tracking the daily frequency and severity of her panic attacks during this time. The 86.3% decrease in BAI scores and 57.9% reduction in PDSS–SR scores from the intervention to posttreatment phase suggests continued improvement in panic and anxiety symptoms after treatment ended as the subject consolidated and practiced her learned coping skills.

The third hypothesis stated that GAF scores would be significantly improved at posttreatment and 3-month follow-up compared to baseline GAF (operationally defined

as an increase of 10 or more; Eisen, Ranganathan, Seal, & Spiro, 2007). GAF score changed from 51 at intake to 80 at the end of the posttreatment phase. The initial GAF score of 51 was because the participant experienced more than occasional panic attacks, had few friends with whom she interacted, thought she could not manage the symptoms of panic, feared having more panic attacks, and avoided activities because of this fear. The final GAF score of 80 was because she no longer experienced any panic attacks, was able to engage in many more activities she previously avoided, knew that she could manage her panic symptoms, knew that the symptoms would not lead to her losing control, still experienced anxiety in response to life stressors, and felt competent in continuing to improve. Mainly as evidenced by the elimination of panic attacks and the return to activities that she previously avoided or endured with great distress, the client's global functioning had improved significantly. This improvement from a GAF score of 51 at intake to 80 one month after treatment concluded represents an improvement of 29 points on the GAF scale. This increase is almost three times greater than the minimal improvement needed to qualify as significant, operationally defined as an increase of 10 or more by Eisen et al. (2007).

The fourth hypothesis stated that the frequency of cognitive distortions would be significantly decreased, as measured by the ICD, at posttreatment and 3-month follow-up compared to ICD scores at baseline (operationally defined as a reduction of 30.0% or more). As measured by comparing the first and last data points (1 month posttreatment), this hypothesis was confirmed by a 32.1% reduction, and substantiated by a 67.0% reduction in the 3-month follow-up score. However, using a more conservative estimate of phase means, there was only a 20.0% reduction from 185 in the baseline phase to 148

in the posttreatment phase. It is important to note that ICD scores continued to decrease throughout the course of the study, from the baseline phase mean of 185 to the intervention phase mean of 173.7 to the posttreatment mean of 148, and to a score of 64 at the 3-month follow-up, suggesting that improvements in cognitive distortions occurred continuously. However, because of the conservative estimate of a 20.0% reduction, these results lack conclusive evidence to support the hypothesis that the frequency of cognitive distortions would be significantly decreased following this intervention. However, it appears that the change in behavior, in this case, cessation of panic attacks, seems to have an enduring therapeutic effect over time, a continued reduction in cognitive distortions that is sustained long beyond the intervention phase. This is consistent with Beck's model and likely occurs as daily events challenge maladaptive cognition.

The fifth hypothesis stated that the reduction in panic attack frequency and severity would be maintained at 3-month follow-up. As hypothesized, there was a complete absence of panic attacks at the 3-month follow-up. Based on the complete absence of panic attacks, a measure of panic attack severity was not possible.

Clinical Significance

Jacobson, Follette, and Revenstorf (1984) defined clinically significant change as a change in an individual's scores on a particular measure from the dysfunctional to the functional range. On the BAI, a measure of the severity of symptoms of anxiety, mean scores decreased from the severe range during baseline (mean score of 33) to the moderate range during intervention (mean score of 18.3), and then to the minimal range during the posttreatment phase (mean score of 2.5). On the PDSS–SR, a measure of panic disorder symptom severity, the subject began in the severe range during the

baseline phase (mean score of 15), decreased below the cutoff score of 13 to the mild range for the intervention phase (mean score of 10.7), then ended well below the cutoff for the presence of panic disorder (cutoff of 8) for the posttreatment phase (mean score of 4.5). While the ICD does not have predetermined cutoff scores indicating different ranges of functioning or disability, mean scores decreased 20.0%. This suggests a reduction in the frequency and severity of cognitive distortions that underlie panic disorder (Yurica, 2002). Furthermore, a 20.0% reduction in ICD scores may be sufficient for clinically significant initial change.

The frequency and severity of panic attacks also speaks greatly to the clinically significant improvements seen in the study. Following the initial treatment session, there was a 39.1% reduction in panic attack severity (comparing the panic attacks in the week before and after the first treatment session). Because the subject's panic frequency was already improving during the baseline phase, SMA is unable to statistically demonstrate that the intervention was responsible for the reduction in panic frequency. However, the results of SMA indicated no return of panic attacks to a statistically significant degree. Furthermore, a zero panic attack frequency was maintained for 4 consecutive weeks to conclude the posttreatment follow-up period, and the cessation of all panic attacks was maintained at 3-month follow up, indicating significant clinical results by any estimation. Finally, the 29-point improvement in GAF scores from 51 at baseline to 80 after completing treatment indicates a significant improvement in functioning, operationally defined as an increase in GAF of 10 or more points and important behavioral measures of functionality (Eisen et al., 2007).

Jakobsen, Gluud, Winkel, Lange, and Wetterslev (2014) suggest that clinical significance can be defined through agreement with the subject before beginning treatment. For this participant, we sought to reduce panic attacks to reduce her significant distress and so that she would be able to function in the areas of her life that had been impaired by the panic. Even before finishing treatment, the subject reported that she had been able to re-engage in the important areas of her life that had previously been inhibited by her fear and panic, suggesting that this definition of clinical significance had been met.

Based on the scores from the BAI, PDSS–SR, and the self-reported frequency of panic attacks, it appears that the subject made clinically significant improvements and would no longer meet criteria for a diagnosis of panic disorder. The relatively extreme improvements measured by the BAI and PDSS–SR, but not the ICD, suggest that significant changes in cognitions may be helpful, but not necessary to create clinically significant improvements in functioning and panic reduction. Conversely, it may be that more modest reductions in cognitive distortions may be sufficient to produce clinically significant results.

Limitations

There are many limitations of this study. First, the intervention was conducted with only a single participant, restricting statistical power and calling into question generalizability of results. The small *N* was solely due to difficulty in recruitment and a surprising dearth of patients meeting criteria for panic disorder and other inclusion criteria. Second, the follow-up period was relatively short, 3 months, restricting the ability to determine long-term implications of this treatment. Third, the study was

conducted in a geographically small area, concentrating in southeastern Pennsylvania, potentially further limiting external validity. Fourth, the treatment was administered by only one clinician, a predoctoral intern. The differences in experience between this intern and veteran clinicians may alter the findings.

Furthermore, this study did not await stabilization of measurements during the baseline period. Because the measurements were already been trending in the positive direction, it is difficult to conclude that the intervention was responsible for the improvements. However, due to the distressing nature of panic, it would not have been ethical to make the subject wait and suffer before receiving treatment. Additionally, according to Borckardt et al., "statistically speaking, reasonable sensitivity and selectivity can be achieved with as few as 7–10 baseline observations" (2008, p. 87). A related limitation is that this study did not exclude individuals whose baseline data was already trending towards the positive, which may mean that the subject could have already been experiencing spontaneous improvement without treatment. Conversely, the early positive trend during baseline may have been due to the expectation of treatment and the self-monitoring effect, in which the act of self-monitoring alone can produce symptom reduction (de Jong & Bouman, 1995).

Implications and Future Research

These results indicate that this treatment provides faster and more economical treatment for panic disorder and faster relief for patients from a terrifying and often debilitating disorder than previously validated treatments (Barlow et al., 2000; Klosko et al., 1990). Faster relief can hopefully lower attrition rates, which is a common difficulty for both panic research and treatment (Barlow et al., 2000; Klosko et al., 1990). This

successful intervention may help clinicians treat panic disorder and improve their efficiency and cost effectiveness by reducing the time and expense needed to significantly decrease panic attacks.

This study may encourage researchers to explore brief treatments for other disorders. In terms of advocacy, this validation of a brief treatment could lower costs and increase utilization of mental healthcare service for individuals with panic disorder. The lower cost could also increase accessibility for individuals with lower socioeconomic status.

Future research should attempt to study this intervention with a much larger population (at least 40 participants) in order to use a randomized control trial design. This replication can also address other limitations by extending the follow-up period, varying the geographic diversity, varying the cultural and ethnic diversity of the participants, using a representative community sample, including individuals with high levels of agoraphobia, and administering the treatment with a mix of doctoral and master's level clinicians. A component analysis can also be conducted in which particular components are removed from the treatment in order to determine which interventions are necessary and sufficient to create positive change and which components may be worth discarding from the treatment protocol. This may be complicated because behavioral experiments can be an effective cognitive treatment by challenging assumptions and underlying beliefs. It is also possible that this protocol could benefit from adding an intervention for safety behaviors, which could interfere with overall treatment success.

A further fruitful area of inquiry would be to further explore the role of cognitive distortions in the perpetuation and amelioration of this and other troubling disorders. A determination could be made as to the magnitude in ICD score reduction required for clinically significant change. Additionally, there may be particular cognitive distortions specific to the maintenance of panic disorder. Finally, an assessment of anxiety sensitivity can be conducted throughout treatment to determine if there is any significant change as a result of this intervention.

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