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Etiology and treatment of panic disorder

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Abstract

Over the past decade, there has been much research related to panic disorder. This is probably due to the high prevalence and incapacitating nature of the disorder. In the United States, anxiety-related disorders are the most prevalent clinical disorder (Michelson & Marchione, 1991), and panic disorder is the most frequently diagnosed of the severe anxiety disorders (Leaman, 1992). Individuals who suffer from panic disorder seek professional help more often than individuals who suffer from any other mental disorder (Margraf & Ehlers, 1991). According to Leaman (1992), 3-4% of Americans either currently have, or will have, panic disorder in their lifetimes. Studies have shown that panic attacks are common in the general population, with 10% of adults occasionally experiencing panic attacks {Margraf & Ehlers, 1991). Craske, Rapee, and Barlow (1992) also found the prevalence of panic to be common, with 6-12% of the population experiencing spontaneous episodes of panic.

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Etiology and Treatment of Panic Disorder

Over the past decade, there has been much research related to panic disorder. This is probably due to the high prevalence and incapacitating nature of the disorder. In the United States, anxiety-related disorders are the most prevalent clinical disorder (Michelson & Marchione, 1991), and panic disorder is the most frequently diagnosed of the severe anxiety disorders (Leaman, 1992). Individuals who suffer from panic disorder seek professional help more often than individuals who suffer from any other mental disorder (Margraf & Ehlers, 1991). According to Leaman (1992), 3-4% of Americans either currently have, or will have, panic disorder in their lifetimes. Studies have shown that panic attacks are common in the general population, with 10% of adults occasionally experiencing panic attacks (Margraf & Ehlers, 1991). Craske, Rapee, and Barlow (1992) also found the prevalence of panic to be common, with 6-12% of the population experiencing spontaneous episodes of panic.

Studies show 65% of all panic disorders first appeared in individuals between the ages of 15-30 (Giesecke, 1987; Kahn, 1990). This makes it highly possible that individuals may be presenting with panic disorder for the first time in a college counseling center. Norton, Dorward, and Cox (1986) found over 35% of a large college student sample reported experiencing panic attacks. Therefore, counselors in university settings should be keenly aware of the symptoms and treatment options of panic disorder.

Misdiagnosis may be possible due to the diversity of symptoms associated with panic disorder (Giesecke, 1987). Katon's (1984) study found 61% of female clients presented over 14 physical complaints, and 33% of male clients had over 12 complaints related to their panic disorders. Ballenger (1987) estimated that 20-35% of Americans had had a panic attack within the previous year and that 77% of the population with panic disorder had not received a correct diagnosis for treatment. According to Giesecke (1987), 73-92% of clients who did not receive appropriate treatment for their panic disorders were experiencing symptoms 20 years after the original diagnosis.

The severity and chronic nature of panic disorder when it remains untreated make early and accurate diagnosis and efficacious treatment of the disorder especially vital. The purpose of this paper is to review the diagnosis, the consequences, the etiology, and the psychological and the pharmacological treatments of panic disorder.

The Diagnosis and Assessment of Panic Disorder

The effective treatment of panic disorder can only proceed from an accurate diagnosis. The DSM-IV (American Psychiatric Association, 1994, p. 402) characterizes panic disorder as

> (a) the presence of recurrent, unexpected Panic Attacks followed by at least 1 month of persistent concern about having another Panic Attack, worry about the possible implications or consequences of the Panic Attacks, or a

> significant behavioral change related to the attacks, (b) absence of Agoraphobia, (c) the Panic Attacks are not due to the direct physiological effects of a substance or a general medical condition, and (d) the Panic Attacks are not better accounted for by another mental disorder such as Social Phobia, Specific Phobia, Obsessive-Compulsive Disorder, Posttraumatic Stress Disorder, or Separation Anxiety Disorder.

A panic attack, as defined in the DSM-IV (American Psychiatric Association, 1994, p. 394), is

> a discrete period of intense fear or discomfort that is accompanied by at least 4 of 13 somatic or cognitive symptoms. The attack has a sudden onset and builds to a peak rapidly (usually in 10 minutes or less) and is often accompanied by a sense of imminent danger or impending doom and an urge to escape. The 13 somatic or cognitive symptoms are palpitations, sweating, trembling or shaking, sensations of shortness of breath or smothering, feeling of choking, chest pain or discomfort, nausea or abdominal distress, dizziness or lightheadedness, derealization or depersonalization, fear of

losing control or "going crazy," fear of dying, paresthesias, and chills or hot flushes. Attacks that meet all other criteria but have fewer than 4 somatic or cognitive symptoms are referred to as limited-symptom attacks.

Individuals who have experienced panic attacks describe them as "the worst thing that has ever happened to them" (Leaman, 1992, p. 127). Panic attacks vary greatly from person to person, but are typically unexpected and unprovoked with alarming symptoms. These attacks include both physical symptoms, like sweating, shaking, shortness of breath, racing heart, and the psychological symptoms of dread and terror. Without treatment, this disorder generally progresses. Because of this, Leaman (1992) advises not to postpone treatment until the disorder progresses far enough to have an official label. Early diagnosis and treatment of panic disorder is important since the disorder can be so incapacitating for the individual, and the treatment so successful.

The assessment procedure for diagnosing panic disorder is comprehensive and involves a structured interview (Dattilio & Kendall, 1994). Assessment instruments such as the SCID (Structured Clinical Interview Schedule for DSM-III-R) (Spitzer, Williams, & Gibbon, 1985) or the ADIS-R (Anxiety Disorders Interview Schedule-Revised) (DiNardo, Barlow, Cerny, Vermilyea, Vermilyea, Himadi, & Waddell, 1985) may be used to perform the

structured interview (Dattilio & Kendall, 1994). The panic section of the ADIS-R or an abridged version of the SCID (SCID-UP-R) (Spitzer & Williams, 1986) may be used to expedite the assessment in a crisis situation.

A recently developed assessment is known as the SAEB (Symptoms-Automatic Thoughts-Emotions-Behavior) system (Dattilio & Berchick, 1992). This assessment may be recommended for panic disordered individuals to help them recognize the link between their panic symptoms and their responses to initial physical sensations.

Consequences of Panic Disorder

The consequences of panic disorder can be devastating and the long term prognosis without treatment is unfavorable (Margraf & Ehlers, 1991). Panic disorders tend to increase in severity, frequency, and duration if untreated. Untreated panic disorders can lead to hypochondriasis, chronic anxiety, and phobic avoidance (Kahn, 1990). Phobic avoidance is perhaps one of the most disruptive symptoms in the life of the panic disordered individual.

Individuals with panic disorder often become fearful about the next panic attack and start to avoid situations they believe will trigger panic. The avoidant behaviors that develop as a result of this fear may lead to agoraphobia (American Psychiatric Association, 1994). In this case, panic disorder with agoraphobia (PDA) would be diagnosed. According to Kahn (1990), about 66% of clients diagnosed with panic disorder also have

agoraphobia. Over 95% of people who seek treatment for agoraphobia can also be diagnosed or have a history of panic disorder (American Psychiatric Association, 1994). Agoraphobia is particularly problematic because chronic avoidance of situations may severely impair the individual's ability to carry out his or her responsibilities.

Agoraphobia is just one of the associated risks of panic disorder. Individuals who suffer from panic disorder are also at higher risk for depression; suicide; alcohol/substance abuse; tranquilizer addiction; generalized anxiety disorder; posttraumatic stress disorder; social, simple, and sexual phobias; and impaired social, marital, and vocational functioning (Michelson & Marchione, 1991).

The increased possibility of suicide clearly makes panic disorder a major mental health concern. The suicide rate for individuals diagnosed with panic disorder is reported to be 17 times higher than in the general population (Weissman, Klerman, Markowitz, & Ouellette, 1989). Weissman et al. (1989) also noted that 12-20% of clients diagnosed with panic disorder have attempted suicide in their lifetimes. Researchers have observed that individuals diagnosed with panic disorder attempt suicide more frequently than individuals diagnosed with other psychiatric disorders or those who do not experience panic (Norton, Rockman, Luy, & Marion, 1993; Anthony & Petronis, 1991).

At even greater risk of suicide are individuals with co-existing alcohol abuse disorder (Weissman et al., 1989).

According to Kahn (1990), panic disorder is estimated to be prevalent in 15% of adult male alcoholics and was present before they started drinking. Norton et al. (1992) found suicide attempts to be more common among clients who were comorbid with another disorder, particularly female alcoholics who suffer from panic attacks. Working with panic clients who abuse alcohol must include procedures for reducing suicidal concerns (Norton et al., 1992).

According to Cox, Norton, Swinson, and Endler (1990), data suggest that alcohol and anxiety problems frequently co-exist and the majority of alcoholic clients with panic disorder and agoraphobia believe self-medication to be an effective coping strategy. Males, in particular, report more alcohol use than females and may be more prone than females to using alcohol in coping with their anxiety (Cox, Swinson, Shulman, Kuch, & Reichman, 1993).

Epidemiological studies have shown that panic disorder has a high correlation with major depressive disorder (Lesser, 1988), with comorbidity rates ranging from 64-75% between panic and depression (Laberge, Gauthier, Cote, Plamondon, & Cormier, 1993). Klerman (1988) found the percentage of panic disordered clients suffering from major depression ranged from 23-53%. Clum and Pendry (1987) reported that panic disorder is typically more severe and incapacitating when it co-exists with depression.

When there is co-existing panic disorder and depression, Laberge et al. (1993) found individuals to be more socially

anxious, fearful of criticism, unassertive, and highly agitated. These individuals also reported less symptom free periods and a higher frequency of panic attacks. The complexity of this co-occurring syndrome leaves the person more disabled and may require a different method of treatment than those clients who suffer only from panic disorder (Laberge et al., 1993).

Etiology of Panic Disorder

Although researchers agree that the etiology of panic disorder is unclear, there are several etiological theories and there are clear characteristics that separate individuals who suffer from panic disorder from those who do not. Margraf and Ehlers (1991) suggested that there seems to be an interaction between psychological and physiological processes. The question remains whether the individual's tendency to associate bodily sensations with threat leads to physiological responses or whether physiological dysfunctions such as hyperventilation lead to panic (Margraf & Ehlers, 1991).

In most cases, there is no distinct precipitant of an individual's first panic attack. Kahn (1990) estimated that a significant life event may precede the first attack in 20-30% of cases and a physiologic event such as substance abuse may precede another 20% of cases. In 81% of panic disordered clients, the site of their first panic attack is a public place rather than at home (Kahn, 1990).

As early as 1962, Klein and Fink reported that childhood separation anxiety is a possible precursor to the development of

panic anxiety and agoraphobia. Kahn (1990) agreed that childhood separation anxiety apparently plays a role in the history of adult panic clients in as many as 20-50% of cases. In retrospective studies, Zitrin and Ross (1988) found an increased incidence of childhood separation anxiety reported by agoraphobics as compared to "normals," and Stewart, Knize, and Pihl (1992) found clinical reports and some experimental data to suggest agoraphobics were overprotected as children. A study by Stewart et al. (1992) contradicts these findings, so it appears it may be inappropriate to draw conclusions about the relationship between early childhood separation anxiety and adult panic disorder.

Individuals who suffer from panic attacks appear to differ in several ways from those who do not report feelings of panic. Panic disordered clients and "normals" have a similar number and intensity of stressors in their lives, but individuals who panic report more negative impact as a result of their stress and they react more intensely to stress (Rapee, Litwin, & Barlow, 1990). Panic disordered clients, as compared to "normals," are more highly aware of somatic cues and tuned into internal stimuli (Last, 1991). These individuals have a pattern of thought during panic attacks that are characterized by fears of immediate, drastic outcomes such as death, insanity, or loss of control (Rapee, 1991).

Etiological Models

According to Acierno, Hersen, and Van Hasselt (1993), etiological theories of panic can be divided into four main models: biological, hyperventilation, classical conditioning, and cognitive.

Biological model. Early etiological theories of panic were biologically based. It was believed that panic attacks were spontaneous and had no connection to environmental stressors (Acierno et al., 1993). In the biological model, panic attacks arise from cellular dysfunction, rather than psychological conflict (Kahn, 1990).

Evidence of biological causes came from familial history and the effectiveness of pharmacological interventions (Acierno et al., 1993; Leaman, 1992). Genetic studies suggest that panic is a disorder of localized brain chemistry to which there is genetic vulnerability (Kahn, 1990). Liebowitz (1986) found genetic evidence included higher rates of panic disorder in first degree relatives of individuals with panic disorder and monozygotic twin concordance exceeding that for dizygotic twins. A study by Crowe, Noyes, Pauls and Slymen (1983) reported the relatives of panic disordered clients had a lifetime morbidity risk of 24.7%.

Wolpe and Rowan (1988) criticized the biological model and suggested looking at pharmacological effectiveness as evidence that biological etiology may be an erroneous inference. They point out that an 86% relapse rate after pharmacological

interventions only demonstrates a symptomatic relief and not a significant affect on the underlying pathology of panic disorder. Dattilio and Kendall (1994) stated there is little evidence to prove a biological etiology for panic disorder, since there are no sure methods for testing chemical imbalances in the brain.

Hyperventilation model. Hyperventilation is common among individuals with panic disorder and proponents of this theory implicate inappropriately increased ventilation in panic attacks. Dyspnea, the sensation of suffocation, is the most frequently found symptom of panic sufferers and also central to hyperventilation (Acierno et al., 1993). Wolpe and Rowan (1988) reported the voluntary induction of hyperventilation in a controlled setting frequently caused subjects to report the beginning of a panic attack.

Hyperventilatory dyspnea appears to be directly associated with the intense symptoms of panic. However, Acierno et al. (1993) suggested that the frequent nature of attacks in panic disordered individuals versus the infrequent nature of spontaneous hyperventilation in the general public may indicate that causal factors other than hyperventilation exist. Studies by Margraf and Ehlers (1991) showed the relationship between hyperventilation and panic attacks cannot be explained by a simple physiological mechanism. They found it was possible to induce anxiety and bodily arousal in panic clients by manipulating their cognitions (Margraf & Ehlers, 1991).

Classical conditioning model. Classical conditioning theory purports that intense hyperventilation produces symptoms which cause the majority of initial panic attacks, and that physiological sensations become conditioned elicitors of future panic attacks (Acierno et al., 1993). In the classical conditioning model, the panic attack is the unconditioned response elicited by an unspecified, unconditioned stimulus (Ley, 1987). Thoughts or experiences connected with the individual's earlier panic episodes are the conditioned stimulus that elicit fear.

Wolpe and Rowan (1988) proposed that initial panic attacks could be attributed to the physiological effects of hyperventilation which was the result of intense, prolonged anxiety. They concluded that panic attacks were the climax of severe and prolonged anxiety and that panic disorder was a product of classical conditioning. According to Kahn (1990), this is an incomplete theory because it does not specify the initial unconditioned stimulus and, therefore, prevents the development of effective interventions.

Cognitive model. Panic disordered individuals differ from "normals" on subjective levels of reported fear (Acierno et al., 1993). This points to a cognitive origin of the panic attack, rather than biological causes. According to cognitive models of panic, panic attacks are a result of catastrophic misinterpretations of bodily sensations (Borden, Lowenbraun, Wolff, & Jones, 1993). A vicious cycle begins with the

catastrophic misattribution of somatic cues. The individual interprets unfamiliar physiological sensations as evidence of imminent danger of physical or mental breakdown. This interpretation leads to increased anxiety, which in turn, exacerbates the bodily sensations. With this increase, the individual reaffirms the initial disastrous misattribution. The cycle concludes with a panic attack (Acierno et al., 1993).

Conversely, Wolpe and Rowan (1988) found that catastrophic thoughts follow, rather than precede, the initial panic attack. Acierno et al. (1993) warned that it is important to remember that just because an individual with panic disorder produces catastrophic cognitions in response to his or her bodily cues does not prove these cognitions caused the attack. The importance of catastrophic cognitions should not be underestimated though, because these thoughts can exacerbate existing panic or become conditioned elicitors of future panic attacks (Acierno et al., 1993).

Etiological Models Summary

It becomes evident there are problems with each of these theories. These problems blur the etiological picture. It is highly unlikely that any one theory accounts for all panic attacks. Furthermore, the tenets of each of these theories are not mutually exclusive, and interactions among them are probable. Pharmacological-, conditioning-, and cognitive-based interventions have all proven successful in reducing panic, with a combination of the three producing the greatest success

(Acierno et al., 1993). This implies that multiple etiological factors exist in panic disorder. Evidence seems to support the theory that hyperventilation or some other highly somatic event occurs during a stressful period and leads to an individual's initial panic attack. The internal physiological cues associated with hyperventilation then become conditioned elicitors of panic in the future. This situation is exacerbated by the person's catastrophic misinterpretations of somatic cues, which increase arousal and fear and ultimately leads once again to panic.

Treatment Interventions

There are several psychological and pharmacological interventions currently being used in the treatment of panic disorder. Pharmacological agents such as benzodiazepines, tricyclic antidepressants, and MOA inhibitors have been found effective for some individuals. Recently, different combinations of cognitive and behavioral therapy have been attracting attention due to their high efficacy rates in treating panic disorder.

Pharmacotherapy

Several classes of medications have demonstrated panic-blocking effects (Telch, 1988), including tricyclic antidepressants (Imipramine), MAO inhibitors (Phenelzine), and high-potency benzodiazepines (Alprazolam). These medications have proven helpful for many panic sufferers, but have troublesome side effects including high client attrition rates

and relapse upon withdrawal of medication (Telch, Lucas, Schmidt, Hanna, Jaimez, & Lucas, 1993).

Benzodiazepines such as alprazolam (Xanax) and clonazepam (Klonopin) have been the drugs of first choice for clients with panic disorder. Their advantage is immediate action (Alexander, 1991) and the fact they are anti-anxiety as well as anti-panic medications (Kahn, 1990). Ballinger (1990) reported a reduction in panic attacks for approximately 60% of clients receiving benzodiazepine treatment. Results of the same study showed that of those who discontinued the treatment, 30% suffered rebound panic attacks worse than the original attacks. According to Ballinger (1990), 90% of those clients treated with benzodiazepine experienced a relapse following medication withdrawal. Benzodiazepines appear to be beneficial in reducing panic, but there are several concerns related to their use. The possibility of physical or psychological dependence, side effects such as sedation and psychomotor impairment, low efficacy for treating PDA, and high relapse rates are all major drawbacks to the use of benzodiazepines (Alexander, 1991; Michelson & Marchione, 1991).

Tricyclic antidepressants (Imipramine) are the drugs of first choice when clients have a history of alcohol abuse or if they fear addiction (Alexander, 1991; Kahn, 1990). Imipramine is less expensive than alprazolam and does not cause dependence (Gelder, 1992). Dattilio and Kendall (1994) stated that imipramine has been found to be more effective than other

medications in the long run for treating panic, particularly when panic is accompanied by depression.

A major drawback to imipramine treatment is that many clients drop out in the early weeks because of the drug's side effects, which include increased anxiety and poor sleep (Gelder, 1992). According to Alexander (1991), antidepressants take up to eight weeks to become optimally effective, so clients often find this delay another disadvantage. Antidepressants are toxic when individuals overdose. Since 20% of panic disordered individuals have a history of suicide attempts, these high risk clients may be better treated with benzodiazepines (Alexander, 1991).

The clinical course for clients on antidepressants alone appears less optimistic than when combined with cognitive-behavioral treatments. A relapse rate of 35-50% is the norm rather than the exception when treating panic with antidepressants (Michelson & Marchione, 1991).

MOA inhibitors have been found to be effective in treating agoraphobia, but there is little information about their effects in treating panic disorder (Gelder, 1992). MOA inhibitors are usually prescribed only if trials with other pharmacological treatments prove ineffective due to the long list of dietary restrictions and potential dangers (Kahn, 1990). MOA inhibitors are potentially toxic and can cause serious medical problems such as convulsions, hypertensive crisis, and dangerous interactions with other medications and common foods (Michelson & Marchione, 1991). According to Alexander (1991), the dietary restrictions

can be overcome by reliable, well-motivated clients, but the anxiety that is often raised about the potential dangers can be a major deterrent for the individual.

According to Katon (1992), individuals who experience panic attacks are often hypersensitive to bodily sensations. Since all medications have side effects, this hypersensitivity can make the panic disordered individual difficult to treat with medications (Dattilio & Kendall, 1994). Michelson and Marchione (1991) found that 20% of panic clients refused to even take medications. Another 35-40% prematurely terminated pharmacotherapy because of side effects. One-fourth to one-half of the clients who did improve, relapsed on medication withdrawal (Michelson & Marchione, 1991). Although pharmacotherapy can quickly alleviate panic symptoms, it fails to serve as a cure and does not provide coping mechanisms for the individual. Because of these concerns regarding pharmacotherapy, many professionals are turning to psychological interventions, the most popular being cognitive and behavioral therapies.

Behavioral Therapies

The foundation of behavioral treatment and theory is in learning theory, which proposes symptoms such as anxiety and panic are learned behaviors and can therefore be unlearned (Kahn, 1990). The most common panic-related disorder presented for treatment is panic disorder with agoraphobia (PDA). The phobic behaviors that are associated with PDA are typically treated with

behavior therapy or with a combination behavioral therapy and pharmacotherapy (Laraia, Stewart, & Best, 1989).

The dominant treatment technique, and one of the most effective for PDA, is exposure therapy (Laraia et al., 1989). Findings from diverse studies indicate that therapist-assisted, prolonged, graduated in vivo exposure appears to be the most effective behavioral treatment. Michelson and Marchione (1991), reported that exposure enhanced the rate and level of improvement in 50-65% of panic clients. Swinson, Soulios, Cox, and Kuch (1992) also reported that using in vivo exposure therapy in the brief treatment of panic during crisis situations has been found to be relatively effective.

Exposure brings the individual into contact with the feared object or situation using imagery or in vivo experiences. In vivo exposure requires the client to contact fear-eliciting stimuli in real life situations. The rate of exposure can be varied with gradual steps called graded exposure or more rapidly called flooding or implosion therapy (Wright & Borden, 1991). Flooding involves presenting high-intensity, aversive anxiety-producing stimuli, either in imagery or in vivo, until the anxiety is extinguished (Laraia, 1989). Implosive therapy presents highly anxiety-producing imagery to the client in a dramatic, vivid manner and maximizes the anxiety producing reaction to the phobic stimulus (Laraia, 1989).

Systematic desensitization is another behavioral technique which is an imagery-based, three-part approach to treating phobic

avoidance. The three steps are (a) muscular relaxation training, (b) anxiety hierarchy construction, and (c) gradual presentation of scenes from the hierarchy in order of increasing difficulty. In vivo systematic desensitization is similar, but imagery is replaced with exposure to the client's fears in real life (Laraia, 1989). Another technique, progressive relaxation, reduces the level of fear producing thoughts, anxiety, muscle tension, and autonomic arousal (Wright & Borden, 1991).

According to Laraia et al. (1989), behavioral treatments of the phobic disorders is generally considered to be effective for 75% of the clients who complete treatment. But up to 49% of all individuals who seek treatment drop out (Laraia et al., 1989), leaving an overall efficacy rate for in vivo exposure of 51%. Attrition rates for exposure modalities average 15-20% (Clum, 1989), and approximately 30-40% of PDA clients who receive exposure therapy exhibit only moderate benefits (Michelson & Marchione, 1991).

Although research shows behavioral techniques, particularly exposure, to be efficacious in alleviating the phobic avoidance behaviors often associated with panic disorder, the effectiveness of behavioral treatments in reducing panic attacks remains questionable (Kahn, 1990; Laraia et al., 1989). Behavioral strategies are beneficial, but it appears they too have their limitations.

Cognitive Therapies

Cognitive therapy was originally developed as a short-term therapy for depression, but has been found to be successful in the treatment of anxiety disorders (Wright & Borden, 1991). According to Beck, Sokol, Clark, Berchick, and Wright (1992), individuals who experience panic attacks are particularly sensitive to cognitive and cognitive-behavioral interventions. In individuals who experience panic, negative thoughts about normal but unpleasant bodily sensations may lead to exaggerated and inappropriate anxiety, which then progresses to panic. Cognitive and behavioral techniques have been combined to counteract the negative thoughts common in anxiety and the behaviors that result from negative thinking.

Beck et al. (1992) stated that research shows benzodiazepines and imipramine can alleviate panic symptoms, but cautioned that many clients are resistant to relying on medication. These authors also had concerns about pharmacological interventions regarding side effects, withdrawal symptoms, and relapse rates as high as 71-95% (Beck et al., 1992). For these reasons, they believe that psychosocial interventions provide many advantages over pharmacological methods and suggested non-pharmacological options are worth exploring.

According to the cognitive model of panic, the essential factor in panic attacks is the individual's inability to reason rationally about the problem, whether it is physical or

interpersonal (Beck, Emery, & Greenberg, 1985). Wright and Borden (1991) stated that the goal of cognitive therapy is to teach individuals to evaluate, control, and modify excessive and maladaptive levels of anxiety by targeting unrealistic fears and avoidance behaviors.

The effects of cognitive treatments are confounded by behavioral exposure exercises, although pure cognitive interventions also appear to be effective in the short-run. There are several widely used cognitive-behavioral treatments which directly target panic. The focus of treatment is on recognizing the client's emotional hypersensitivity to bodily cues and reinterpreting the client's belief that these cues signal impending catastrophe (Telch et al., 1993). Cognitive-behavioral treatments include (a) education about the nature and physiology of anxiety and panic, (b) cognitive restructuring of the client's faulty threat appraisals, (c) breathing exercises to control hyperventilation, and (d) interoceptive exposure which is purposeful and repeated induction of feared somatic cues (Telch et al., 1993).

Cognitive restructuring of self-statement training consists of three steps which include (a) identifying negative self-statements; (b) recognizing how negative self-statements influence self-concept, behavior, and mood; and (c) replacing negative self-statements with positive statements that will help the client cope with high levels of anxiety (Laraia et al., 1989). The purpose of this type of restructuring is to lessen

the likelihood that client's thoughts are increasing their symptoms and emotional reactions and to persuade them that their fear is unsubstantiated (Dattilio & Kendall, 1994). By restructuring their thoughts, clients are asked to consider an alternative response that may involve a less catastrophic implication.

Some therapists believe that progressive muscle relaxation and controlled breathing are all that is needed to stop recurring panic attacks (Dattilio & Kendall, 1994). These techniques are based on the premise that a state of relaxation and a state of panic cannot coexist (Clark, Salkovskis, & Chalkley, 1985). In controlled breathing, individuals are asked to breathe through their noses normally, counting the number of breaths, usually six to nine times per minute (Dattilio & Kendall, 1994). The goal of this exercise is to slow the respiration rate.

Another cognitive technique, symptom induction, involves instructing the client to intentionally bring on a panic attack. The rationale behind this technique is that phobics struggle against anxiety. In stopping the struggle by intentionally initiating the feared symptoms, the individual gains power and control over anxiety. This should lead to a decrease in the individual's anxiety level (Laraia et al., 1989). The goal of symptom induction is to reproduce the type of situation that may precipitate a panic attack and then demonstrate to the client that he or she has the power to "turn on" as well as "turn off" the attacks.

Therapists who use paradoxical intention in treating panic attacks ask clients to exaggerate their anxiety. An individual who fears he or she is dying may be instructed to "let yourself die" (Dattilio, 1987, p. 57). Therapists encourage the client to look at the irrational aspect of his or her fear (Dattilio & Kendall, 1994). The individual usually discovers he or she is unable to achieve the feared response and anxiety decreases.

Telch et al. (1993) found that cognitive-behavioral therapy can be effective in both the long and short-term using the cost-effective group format. Group-administered cognitive-behavioral treatment resolved panic attacks in over 85% of treated clients (Telch et al., 1993). In one study, Craske (1991) reported that cognitive-behavioral group therapy evidenced the strongest pattern of improvement on measures of panic.

Efficacy data on cognitive-behavioral therapy indicate that approximately 80-90% of treated clients were symptom-free by the end of treatment (Telch et al., 1993). An important element of cognitive-behavioral therapy is the extent to which improvement is maintained following treatment completion. In contrast to the substantial relapse observed in drug treatment, cognitive-behavioral therapy showed lasting improvement for periods up to two years (Craske, 1991; Telch et al., 1993). According to Chambless and Gillis (1993), clients in a number of studies continued to improve during the period following cognitive-behavioral therapy, which has not typically been the case when using strictly behavioral methods.

Treatment Summary

Alprazolam, imipramine, and cognitive-behavioral therapy appear at this time to be the treatments of choice for panic disorder. According to Gelder (1992), no other treatments have been shown convincingly to be as effective. Clum, Clum, and Surls (1993), Craske (1991), Dattilio and Kendall (1994), Margraf and Ehlers (1991), and Michelson and Marchione (1991) suggested that when all criteria were considered, psychological treatments were found to be superior to pharmacological treatments. Michelson and Marchione (1991) reported that efficacy studies found cognitive-behavioral therapy to be superior to beta-blockers, benzodiazepines, and anti-depressants. A study by Klosko, Barlow, Tassinari, and Cerny (1990) compared cognitive-behavioral therapy to alprazolam. They found 87% of the clients treated with cognitive-behavioral therapy were panic-free at posttreatment, compared to 50% in the alprazolam group. Cognitive-behavioral therapy may be the first choice if the resources necessary for conducting this intervention are available. The short-term results appear equivalent to those of drugs, there are no side effects, and there is some evidence that the benefits persist over time (Gelder, 1992).

Conclusion

Panic disorder is one of the most common and disabling psychological disorders both in mental health and general medical settings. The course of panic disorder is typically one of relapses and remissions over the decades, the attacks worsening

in frequency, intensity, or duration. If untreated, panic attacks can contribute to hypochondriasis, chronic anxiety, depression, agoraphobia, and often suicide. If possible, treatment should begin in the early stages, before the disorder becomes incapacitating for the individual. Therefore, it is critical that therapists be able to accurately assess and diagnose the diverse symptoms of panic disorder.

Clients may differ in the etiology of their panic, so a comprehensive assessment of etiology will help direct treatment toward areas relevant to each individual client. For example, a client may report no catastrophic cognitions during a panic attack, but clearly experience hyperventilation. Perhaps an intervention which emphasizes breathing retraining over cognitive restructuring would be most productive with that client. Acierno et al. (1993) reported that no studies have been done on matching etiological models to treatment interventions for panic. Until the time that such studies occur, a multicomponent approach addressing several etiological factors may be the most efficacious treatment for panic disorder.

Interventions currently being used in the treatment of panic disorder appear to be consistent with the therapist's chosen etiological framework. Biological interventions include benzodiazepines, tricyclic antidepressants, and MOA inhibitors. Psychological interventions include cognitive-behavioral-based cognitive restructuring, breathing retraining, symptom induction,

and paradoxical intention. Behavioral-based interventions include exposure, systematic desensitization, and relaxation techniques.

There are many concerns about pharmacological interventions regarding their side effects, withdrawal symptoms, and relapse rates as high as 71-95%. These problems make non-pharmacological interventions worth exploring, and cognitive-behavioral therapy offers a promising alternative to medication in the treatment of panic disorder. Cognitive strategies, combined with exposure and anxiety management techniques, provide a comprehensive intervention that addresses all possible etiological origins of panic. Medication may serve as an adjunct to cognitive-behavioral therapy, rather than the reverse. Whenever possible, cognitive-behavioral techniques should be considered prior to the use of pharmacological agents.

It is clear that no single model of etiology or treatment has proven sufficient to explain or successfully treat panic disorder in all individuals. It is likely that panic disorder is not a single etiological entity. Conceptualizing this complex disorder using a biopsychosocial framework may be more useful. Unconscious conflicts, conditioning, cognitive misattributions, and biological phenomena may be working individually or in combination to produce and maintain the symptoms of panic disorder. The therapist's diagnostic and treatment skills are extremely important in determining the treatment of choice for each individual client. A comprehensive diagnostic and treatment approach using a combination of cognitive, behavioral, and

pharmacological interventions may be most efficacious in the treatment of panic disorder.

There are some limitations in the current research on panic disorder. Despite the positive results achieved with cognitive-behavioral and pharmacological treatments for panic disorder, many of the clients in these controlled studies have met strict inclusion criteria. Efficacy rates could be reduced when multiaxis disorders are superimposed on panic disorder. Another area of concern is that several populations have been underrepresented in this research including minorities, children and adolescents, geriatric subjects, and clients with accompanying psychiatric dysfunctions. Continued research is needed in these areas.

Future investigations should pay attention to the long-term effects of treatment for panic disorders. Do certain types of cognitive therapies have more lasting effects than others--more explicitly metacognitive versus distraction or self-statement training? Finally, other psychotherapies, perhaps psychodynamic therapies, should be explored as potential complementary or alternative approaches to treating panic disorder.

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