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Nonpharmachological Alternatives to the Treatment of Panic in Crises and Emergency Settings

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Abstract

This article provides a brief review of a number of the theories of panic and introduces the reader to several of the nonpharmacologic interventions and assessments for the treatment of panic in crisis and emergency settings. In addition, the mention of pharmacological agents as well as ancillary suggestive techniques are also made in combination with the above.

Introduction: Panic attacks in the (Diagnostic and Statistical Manual for Mental Disorders-lV) (DSM-IV) are diagnosed by the presence of at least four of the following symptoms: (1) shortness of breath or smothering sensation; (2) dizziness, unsteady feelings, or faintness; (3) palpitations or accelerated heart rate; (4) trembling or shaking; (5) sweating; (6) choking; (7) nausea or abdominal distress; (8) depersonalization or derealization--a feeling that the sufferer's body or environment, respectively, is not real; (9) numbness or tingling sensations in one or more parts of the body; (10) hot flashes or chills; (I t) chest pain or discomfort; (12) fear of dying; and (13) fear of going crazy or losing self-control and the inclusion of "unexpected attacks" being required k) make the diagnosis (American Psychiatric Association, 1994).

Panic disorder has continued to rank among the top ten psychiatric disorders found among emergency settings (Wayne & Goldenberg, 1983). It remains one of the most common and disabling psychological disorders encountered in both mental health and general medical settings. In a recent study, investigators estimated that the one-month prevalence of panic disorder among primary care patients .stands at 1.4 percent (Von Korft, Shapiro, Burke, Teitelhaum, Skinner, German, Turner, Klein & Burns, 1987). In addition, statistics derived from a study assessing community-based epidemiological catchment areas estimate that in any given month 0.5 percent of the population will he diagnosed with panic disorder (Regier, Boyd, Burke, Kae, Myers, Kramer, Rohins, George, Karno & Locke, 1988).

Panic is reportedly a common diagnosis among hospital emergency room patients (Rosenmen, 1985; Dunner, 1985). 'This is in part due to the fact that heart palpitations are among the most common symptoms expressed during a panic attack (Ehlers & Breuer, 1992), and panic episodes are quite common particularly in cardiology patients (Beitman, DeRosear, Basha, Flaker & Corcoran, 1987). Outpatient mental health clinics also report a high frequency of complaints of panic, with estimated inci-

dence among the general population of 1.6 to 2.9 percent in women and 0.4 to 1.7 percent in men (Crowe, Noyes, Pauls & Slymen, 1983; Boyd, 1986).

While the literature on crises and crises intervention in general is abundant (e.g., Kocmur & Zavasnik, 1991; Aguilera, 1990; Roberts, 1990), very little has been devoted to specifically addressing crisis intervention for panic disorder (Dattilio & Freeman, 1994; Diez, Gasto & Vallejo, 1989; Aronson & Logue, 1988), especially in the area of nonpharmacological techniques. This situation is surprising, since it is well documented that physicians have experienced difficulty in differentially diagnosing panic disorder from a variety of physiological disorders for the past 120 years (DaCosta, 1871; Westphal, 1871; Prince & Putnam, 1912; Oppenheimer & Rothschild, 1918; Skerrit, 1983). Moreover, since the primary goal of crisis intervention is to reduce the patient's self-reported emotional distress (Kendall & Norton-Ford, 1982, p. 460), it would he expected that panic would appear near the top of the list of disorders targeted in crises intervention. The situation may become even more complex in cases where the panic victim cannot tolerate medication (i.e., pregnancy, certain medical disorders, etc.).

The scarcity of emergency and crises intervention literature regarding panic may be due to the fact that panic disorder or "panic attacks" are often viewed as crises manifestations of some other underlying problem. In the medical setting, for example, a physician will usually focus immediately on any chronic underlying condition suspected of precipitating the panic symptoms, such as temporal lobe epilepsy, coronary artery disease, alcohol or tranquilizer withdrawal, hyperthyroidism, pheochromocytoma, electrolyte abnormalities, or stimulant medications/decongestants. (For a more extensive list, see Katon 11992, p. 621.) In addition, symptoms of panic can be secondary to other mental disorders such as major affective disorders (Breir, Charney & Heninger, 1984), personality disorder (Barlow, 1988; Beck, Freeman & Assoc., 1990) and alcohol withdrawal (Katon, 1992).

Two Theories of Panic

There have been several attempts to explain the etiology of panic. The Psychobiologists have contributed a number of hypothesis which include: the septohippocampal theory (Gray, 1982), the locus coeruleus theory (Svensson, 1987; Charney, Heninger & Breier, 1984) and the gamma-aminobutyric acid henzodiazepine hypothesis (Skolnick & Paul, 1983). More recently, cholecystokinin tetrapeptide (cck-4) has been studied as a panicogenic agent (Bradwejn, Koszycki, Payeur, Bourin & Borthwick, 1992).

One of the more popular theories is the Sodium Lactate Theory proposed as a result of the early studies by Pitts and McClure (1967). This study examined the effects of Sodium Lactate infusion which induced panic attacks in subjects who had a history of panic only as opposed to others in the study who had no history of panic. Other studies were repeated yielding similar results (Appleby, Klein, Sachar & Levitt, 1981; Dager, Cowley & Dunner, 1987; Goetz, Klein & Gorman, 1994). on the other hand Ley (1986), in his re-examination of the original data of the 1967 Pitts and McClure study, uncovered that it was actually an increase in a sensitivity to uncomfortable symptoms that panic subjects experienced in the 1967 study. This gives rise to a need to determine whether it was an actual chemical chain reaction that occurred, or catastrophic misinterpretation that caused the individuals in this study

to panic. Even more convincing, is the recent study conducted by Margraf and Ehlers (1989) which investigated into panic disordered individuals' reactions to hyperventilation and compared them to a normal population . The influence of subjects expectancies were tested by manipulating instructions that were presented during an exercise. Two groups (panic and normals) were informed that they were participating in a "biological Panic Attack Test" as opposed to the others who were told that they were involved in a "fast paced breathing task". Apparently, the increase in panic subjects' anxiety and arousal depended on what they were told. The expectation that they were taking a panic test produced physiological reactions and self-reported elevations in both anxiety and arousal. Conversely, the manipulation of the instructions and expectations had no effect on normal subjects' responses. As a consequence of these biologically-based theories, the treatment of choice in medical settings involves a pharmacological regimen of high-potency benzodiazepines, tricycle antidepressants or monomania oxidase inhibitors (Sheehan 1982), coupled with behavior therapy (Brown, Rakel, Well, Downs & Akisdal, 1991).

Although the psychobiological explanation of panic has merit, it retains several drawbacks. For one, there is little evidence to prove a psychobiological etiology for panic disorder exists in every case--that is, there is no sure method to test for chemical imbalances or other difficulties in the brain. While the explosion of research relying on Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PEI) scans and Brain Electrical Area Mapping (BEAM) studies have generated tremendous interest in psychobiological research of panic disorders, the complexity of brain-related behavior continues to stall a comprehensive understanding of brain mechanisms involved in panic and anxiety. Pharmacotherapy has proven to expeditiously alleviate symptoms and, in some cases, reduce the likelihood of phobic avoidance by reducing or ameliorating spontaneous panic attacks. Unfortunately, however, it still fails to serve as a cure. It provides the inflicted patient with little in the way of true coping mechanisms other than the reliance on simply taking the prescribed compound as instructed. In addition, individuals with panic disorder can be among the most difficult groups of patients to treat with medication due to their noted hypersensitivity to side effects (Katon, 1992). Lastly, there are those individuals who cannot benefit from medication as a result of metabolic complications or pregnancy.

While pharmacological compounds have offered the most expedient treatment in crises settings and have long been the treatment of choice for emergency situations involving panic and acute anxiety symptoms (Roy-Byrne, Cowley and Katon, in press), the medical literature still suggests that the first stage of treatment in primary care and emergency settings should be to negotiate explanatory models of the illness with the patients (Katon & Kleinman, 1980) and to elicit patients' beliefs about their illness prior to the actual treatment intervention. Such open-ended questions as "What do you believe is the problem?" and "What reasons would you give for the onset of symptoms at this particular time'!" are recommended (Katon, 1992) Unfortunately, questioning can be limited to gathering some background information about the patient's relevant medical history subsequent to a blood profile and/or electrocardiogram (EKG), and medication is often dispensed without further exploration or explanation.

Psychobiological theories argue that panic is essentially the result of biochemical abnormalities associated with genetic predisposition (Sheen, 1982; Weiss & Uhde, 1990); however, recent literature suggests that psychologically-base(l theories are in some cases better supported by empirical research (Beck, Emery & Greenberg, 1985).

A cognitive-behavioral theory of panic suggests that it is psychological factors rather than solely psychobiological factors that precipitate panic symptoms. While the cognitive- behavioral theories do acknowledge with respect the neurochemical components of autonomic symptoms, more emphasis is placed on the perception of threat or danger whether it involves internal (bodily sensations) or external (environmental) events (Raskin, Peeke, Dickman, Pinsker, 1982; Dattilio, 1986; Ottaviani & Beck, 1987). Most specifically, the misattribution theory introduced in the literature within the last decade has proposed that specific symptoms resulting in hyperventilation elicit panic in individuals who arc predisposed, whether genetically or psychologically, to catastrophic misattribution of internal bodily sensations (Lye, 198~; Clark, 1986). According to this theory the most commonly occurring physical sensations during a panic attack include; dizziness, vertigo, blurred vision, tachycardia, palpitations, numbness, tingling in the hands and fcct, nausea and hrcattllessness (Clark, Salkovskis & Chalkey, 1985; Hihbert, 1984; Kerr, Dalton & Gliebe, 1937). Clark and Associates noticed that these sensations hare similarity to sensations produced by hypervcntilalion. As a result, it was hypolhesized that hyperventilation may play an important role in thc initiation of panic attacks (Clark, et. al., 1985). Tlle theory proports that some individuals increase their rcspiralory ventilalion when under stress. This increase causes carbon dioxide to be expelled from the lungs triggering a decrease in the partial pressure of carbon dioxide (pCo2) in the blood with an increase level of pH. Such changes in the blood's chemistry manifests uncomfortatble body sensations such as the aforementioned in which the individuals respond to with startle and apprehension. This increases apprehension subsequently elicits further augmentation in hyperventilation which spirals into a full-blown panic attack.

Clark and Associates contend that it is either the perception of the feared stimuli itself or the induction of fear already elicited by other stimuli that contributes to the catastrophic reaction during this event that precipitates the se(luence of panic. Hence, the notion of teaching indlividuals how to avoid hyperventilation when under slress via breathing retraining, is the crux of the treatment.

Although panic altacks have oQen been reported as occurring "spontaneously," Beck and colleagues (1985) have found thal some panicular experiences appear to activate a person's "alarm system" involvin~ cognitive-affective and physiological components (p. 112). In addition, the aspect of perceived control has received increasing attention in explaining outcome efrects in the trealment ol panic (Borden, Clum & Salmon, 1991.).

Assessment and Diagnosis

The assessment protocol for diagnosing panic is quite comprehensive and usually involves a structured interview. Such interviews may l~e performed with the use of assessment instruments such as the SCID (Structured Interview Schedule for DSM-IV)(Spitzer, Williams & Gibbon, 1985) or the ADIS-R

(Anxiety Disorders Interview Schedule-Reviscd) (DiNardo, Barlow, Cerny, Vermilyea, Vermilyea, Himadi & Waddell, 1985). Untortunately, such comprehensive assessment usually requires a considerable amount of time, which is not always available during crisis situations Some ahridged versions have been developed in recent studies in order to provide as a more expedient method of assessment in crisis situations (SCID-UP-R) (Spitzer & Williams, 1986; Swinson, Soulios, Cox & Kuch, 1992).

It is recommended that a brief clinical interview be conducted which includes an excerpt from the panic section of the ADIS-R and screening questions that elicit the individual's medical history particularly cardiac or seizure disorders) along with all medication currently in use.

Some of the briefer diagnostic questionnaires may also help to pinpoint specific symptoms and to support information which has been obtained from the patient verbally. Such quick screening questionnaires include. The Beck Anxiety Inventory (BAI) (Beck, 1987), Body Sensations Questionnaire (BSQ) (Chambless, Caputo, Bright & Gallagher, 1984), Anxiety Sensitivity Index (ASI) (Reiss, Peterson, Gursky & McNally, 1986) and the Zung Anxiety Scale (Zung, 1975) any of which can be completed in a matter of minutes.

Since much of the cognitive-behavioral literature stresses the importance of relating symptoms to the misinterpretation of interoceptive cues and catastrophic cognition (Alford, Beck, Freeman & Wright, 1990; Ottaviani & Beck, 1987; Dattilio, 1986, 1987, 1990, 1992a; 1994), a formal system for linking panic symptoms to thoughts and emotional/behavioral responses is essential. A recently developed assessment technique known as the SAEB system (Symptoms-Automatic Thoughts Emotions-Behavior) hi recommended as an approach for helping panic sufferers recognize the link between their panic symptoms and their catastrophic responses to their initial bodily sensations (Dattilio, 1990, 1992a, 1992b, 1994; Dattilio & Berchick, 1992) .

The unique design of the SAEB system allows the treating clinician to align specific catastrophic thoughts and misinterpretations of symptoms with the onset of subsequent symptoms in a quick, expedient fashion. The system thus allows the panic victim to see the connection between stages of the escalation process setting the stage for the next step which involves the treatment intervention (Figure 1).

This system is applied by having patients identify the beginning symptom of the panicepisode. If the individual has expe rienced more than one attack, it lends more credence to the repetitive sequence of each attack, for example; in Figure 1, a "spontaneous increase in heart rate" was the initial symptom experienced by an individual who experienced a sudden panic attack. This was followed by "difficulty breathing" and subsequently by "hot flashes and sweating" and so on. once the symptoms have been aligned, the automatic thoughts accompanying each symptom are indicated along with the associated emotion and behavior. Vectors are then drawn in order to (demonstrate to the patient in a collaborative fashion how the catastrophic thought content may be in reaction to the autonomic symptoms experienced and how these thoughts contribute ~o the subsequent behavior and possibly to the subsequent escalation of the symptoms (Dattilio, 1990). This technique is demonstrated in detail in a previously published case study (Dallilio and Ber-

chick, 1992) as well as in another recently published article (Dattilio, l994).

This SAEB system sets the stage for the implementation of several cognitive-behavioral treatment interventions that will be explained later in this text. It is recommended as a quick method of assessment for tracking the cognitive, affective, behavioral and physiological sequence of panic. Pinpointing specific triggers of panic symptoms is also another important aspect of assessment that has been emphasized in the literature (i.e.: stress, hot-stuffy climates, excessive exercise, etc. (Beck, Emery & Greenberg, 1985; Dattilio & Berchick, 1992).

Cognitive-Behavioral Intervention Techniques

There has been a great deal of professional literature which has focused on the use of cognitive-behavioral techniques and their effectiveness in treating panic and anxiety disorders (Beck, Emery & Greenberg, 1985; Barlow, 1988; Salas Auvert, 1993). Most specifically, exposure-based treatments have been quite successful in reducing panic (Barlow, 1988), particularly when used in concert with pharmacological interventions (Zitrin, Klein & Woerner, 1981). Cognitive-Behavioral treatment focusing on panic control through education, cognitive restructuring, interoceptive exposure and breathing retraining have been reported in some cases with success amidst a group format (Telch, Lucas, Schmidt, Hanna, Jaimez and Lucas, 1993).

Most recently, brief treatments of panic attacks in emergency situations have used exposure instruction with relative effectiveness (Swinson, et al., I992). In this particular study, thiny-three (33) patients with panic attacks were seen in two emergency room settings. Forty percent (40%) of the patients, who had been diagnosed with the Structured Clinical Interview Schedule (SCID), as meeting the criteria for panic disorder with agoraphobia. patients were randomly assigned to groups receiving either reassurance or exposure instruction. outcome measures demonstrated significant improvement over a 6 month period for individuals of the exposure group. Individuals of the reassurance group showed no improvement on any measure, and in fact, reported increased symptomatology (Swinson, et.al., 1992). The specific treatment involved informing patients that the most effective way to reduce their fear was to confront the situation in which the attack occurred. Patients were advised to return to the situation as soon as possible after the interview and to wait until thei anxiety decreased (p. 945). While this approach improves upon the use of pharmacological intervention, it still falls short of providing the individud with any specific set of coping techniqucs, especially for dealing with future pdnic episodes. It also relies on extinction procedures and appears to require more time for reducing symptoms and may not always be practical depending on the circumstances in which the individual experienced the attack.

There are a number of additional cognitive-behavioral techniques that may prove to be more satisfactory alternatives in the treatment of panic in crises settings by providing the individual with specific coping mechanisms to apply during future episodes or attacks.

Controlled Breathing

Very early studies on anxiety refer to the use of progressive muscle relaxation and controlled breathing as well as carbon dioxide inhalation (Wolpe, 1958).

These techniques are based on the premise that a state of relaxation and a state of anxiety cannot co-exist and are viewed by some as being all that is necessary to put a stop to recurrent panic attacks (Lum, 1981; Clark, Salkovskis & Chalkley, 1985).

The concept of controlled breathing is an offshoot of the hyperventilation hypothesis mentioned previously which contends that individuals commonly hyperventilate prior to panicking (Hibbert, 1984). Individuals who hyperventilate tend to breathe through their mouths, taking short shallow breaths of air or sighing frequently. Diaphragmatic breathing is one form of breathing retraining for coulnteracting hypcrvcnlilation. In it, individuals are instructed to breathe through their nose normally and to count the number of breaths while at rest, keeping the frequency to 9-16 times per minute. They are also told to place both hands over the abdomen while breathing, noticing the movement of the diaphragm. The goal is to slow the respiration rate to 9-16 times per minute. Individuals are instructed to practice the exercise during both panic and non-panic periods (Clum, 1990).

If during a severe panic attack, the diaphragmatic method does not enable the individual to obtain a full breath, which is often the case, then the alternative of breathing into a paper bag or cupped hands may be used in order to increase the level of carbon dioxide -(Danilio, 1990). An alternative is to exhale through the mouth as much as possible and then slowly inhale through the nose, repeating the process several times. While practicing these techniques, distraction may be used to divert one's attention from the panic symptoms to enhance the positive effects of the breathing exercise

Symptom Induction and De-escalation

The cognitive-behavioral model of panic contends that very often, individuals' misinterpretations of bodily sensations play an integral role in the escalation of panic symptoms. Consequently, such misinterpretations can he responsible for maintaining the vicious panic cycle (Argyle, 1988; Beck, et al., 1985; Dattilio, 1987; Ottaviani & Beck, 1987). During this period of vulnerability, individuals tend to overestimate perceived danger and to underestimate their capacity for coping (Dattilio, 1987, 199O; Greenberg, 1989).

In symptom induction, the client is presented with a therapeutic exercise whereby he or she is instructed to follow the therapist in taking short successive breaths of air, inhaling and exhaling, for approximately 2 to 3 minutes. This procedure serves to reproduce the symptoms of panic by activating the autonomic nervous system and disrupting the balance of oxygen and carbon dioxide levels, sometimes causing hyperventilation as well (Dattilio, 1990). Symptom induction allows the therapist to obtain a direct report of the client's thought processes as the attack develops and to assist the client first-hand in controlling the attack through progressive breathing and thought restructuring. The goal here is to reproduce the type of situation that may precipitate an attack and then show the client that he or she can "turn on" as well as "turn of" the attacks.

Once the symptoms have been induced, the therapist records the sequence of events that have occurred, paying particular attention to the specific symptoms experienced, the automatic thoughts that occurred, and the emotional reaction that was experienced as a result. Figure I provides an example of how to track the client's panic sequence

during an attack. In response to the initial symptom, spontaneous increase in heart rate, the automatic thought is overactive in the sense that it is assumed that "something is wrong" or that the client "could faint".

It is essential that all clients who are candidates for this technique receive medical clearance prior to the exercise in order to ensure that the technique is not contraindicated by an existing medical condition. The therapist can then begin to intervene with the de- escalation techniques by collaboratively focusing with the client on the initial symptoms.

In the case presented in Figure 1, a spontaneous increase in heart rate followed by the thought, "Something is wrong" or "I'm going to faint," translates into fear. By identifying the early onset of symptoms in the panic cycle, the therapist can aid clients in the de-escalation of symptoms. This is done by having patients downplay the severity of the symptoms by altering their misinterpretations. For example, the individual in Figure I had developed a pattern of responding to increased heart rate by perceiving it as dangerous and a sure sign that "Something is wrong." In having clients restructure their thoughts, they are asked to consider an alternative response which may involve a less catastrophic implication. For instance, "Just because I have an increase in heart rate doesn't mean that this is necessarily dangerous or that something is wrong. It is probably just benign autonomic activity which will last for a limited time." This cognitive response is then supported by having the client log each attack and review the log for reassurance that since nothing dangerous has occurred in the past, it is unlikely to occur in the future. Patients are then taught controlled breathing in order to regulate their oxygen intake level and reduce autonomic activity.

The purpose of this type of restructuring is to lessen the likelihood that the individual's automatic thoughts are fueling the subsequent increase in symptoms and emotional reaction and to persuade them that their fear ("I might faint") is unsubstantiated. This point can be reaffirmed with cognitive correction via tactual information (e.g., in order to faint, one must experience a decrease in blood pressure; blood pressure increases with increased heart rate and anxiety). In addition, this serves to improve the patient's perceived sense of bodily control which reduces the intens ty of threat and danger.

This type of thought correction is followed throughout the entire panic cycle and then reinforced by virtue of re-exposure to symptoms through the use of the panic induction exercise. It is the combination of the artificial induction of symptoms (e.g., purposely increasing heart rate), as well as the reinterpretation of these symptoms, (e.g., it won't hurt me), de-escalation of the catastrophic thoughts, (e.g., this won't last forever), and eventual reduction of symptom severity that makes the technique effective. In addition, follow- through on having the client expose him/herself to real-life situations is also an important component to treatment so that the ability to generalize the techniques to a variety of situations can develop.

This technique is usually well received by panic sufferers, particularly after they have overcome their initial apprehension about raising their autonomic activity level. With those clients who sometimes do not benefit from the intervention (e.g., become too overwhelmed or are unable to increase their autonomic activity level), it is recommended that the same technique of cognitive restruc-

turing be used without the symptom induction exercise.

Paradoxical Intention

Paradoxical intention, originally developed by Frankl (1984), is much like symptom induction in that it involves a behavioral prescription for clients to perform responses that seem incompatible with the goal for which they are seeking help. The specific difference, however, is that in paradoxical intention patients are asked to exaggerate their anticipation rather than behaviorally induce the symptoms by deliberately hyperventilating. For example, individuals who experience panic attacks and tear that they may die suddenly or become "overwhelmed" would he instructed to "go ahead and let themselves die" or do whatever they tear they might do (Dattilio, 1987) After several attempts, they often discover that they are unable to achieve the feared response, and their anxiety then diminishes. At this point, many patients are able to perceive the ridiculous or irrational aspect of their apprehensions, which is strongly encouraged by the therapist. They are then instructed to repeat this same procedure in selected settings at graded levels of panic-evoking situations until they experience few or no symptoms. This technique also differs from symptom induction and de- escalation in that there is no de-escalation of symptoms and no instruction in the use of controlled breathing as an anxiety-reducing agent. In fact, it poses the opposite approach to the patient with the reliance on the paradoxical focus itself as the trigger in reducing anxiety (Dattilio, 1987; 1994).

Paradoxical intention has at times been rather loosely defined in the literature, particularly since it has been utilized by therapists of varying theoretical orientations who conceptualize it quite differently (Dell, 1981; Efran & Caputo, 1984; Ascher, 1984; Dowd & Trutt, 1988; Sexton, Montgomery, Goff & Nugent, 1993). More specifically, the debate appears to center around whether or not the "intention" referred to is the patient's or the therapist's. This issue is fully discussed by Dowd and Trutt (1988) and the intention was clearly defined earlier by Frankl (1975) as being the patient's. Thus, the technique of paradoxical intention may fall more clearly within the bounds of a cognitive intervention, since it first forces a behavioral change which is followed by a restructuring of cognition upon reflection on the implication of the behavioral change. This point has been challenged, however, by others (Bandura, 1977) arguing that the behavioral change precedes the restructured cognition.

Paradoxical intention appears antithetical to the other cognitive-behavioral treatments for panic, such as symptom induction, de-escalation and the relaxation-based techniques, mainly because it seems to provide patients with few coping techniques for anxiety. However, at times certain individuals may prove to benefit more from paradoxical treatments than others because of the extinction based philosophy. It is particularly recommended for individuals who may experience relaxation-induced anxiety (Heide & Borkovec 1983; Cohen, Barlow & Blanchard, 1985; Lazarus & Mayne, 1990), in which many of the traditional anxiety-reducing techniques are ineffective. Such side effects as tingling, numbness, dizziness, paradoxical increases in tension, increased heart rate and other untoward reactions have been reported with relaxation-based treatment (Borkovec & Grayson, 1980; Edinger & Jacobsen, 1982). Patients have often reportedly lost interest in progressive muscle relaxation or have repeatedly fallen asleep. Relaxation techniques may at

times even evoke seizure activity or traumatic memories, which may undermine the intention of the treatment (Kiselica & Baker, 1992).

Paradoxical intention would also be recommended in patients who appear resistant to techniques that involve actual symptom induction, as well as patients with a history of cardiovascular disorders. Even though paradoxical intention encourages the symptoms to worsen, there is no direct induction of symptoms (e.g., over breathing); thus, the likelihood of cardiovascular stress is reduced. It is therefore suggested as an alternative treatment when induction is contraindicated and when an expedient intervention is required such as with crisis situations.

Symptom induction, de-escalation, breathing retraining and paradoxical intention are all non-pharmacological techniques which may be applied for rapid amelioration of panic symptoms in emergency and crises situations. In combination with exposure and/or pharmacological interventions, these techniques may prove to be the most efficacious (Brown, Rakel, Wells, Downs & Akiskal, 1991).

Pharmacotherapy

Since medication is so widely used in many crisis settings, it is essential that it be addressed in this chapter. As mentioned earlier, pharmacotherapy has been the medical treatment of choice in most crises or emergency settings involving acute anxiety or panic.

This is particularly so due to the quick- acting effects of many of the pharmacological agents, particularly the benzodiazepines, which act more rapidly than any other antipanic agents (Liebowitz, Fyer & Goman, 1986). Alprazolam is probably the most widely used benzodiazepine (Ballenger, Burrows & Dupont, 1988). A variety of other benzodiazepines, including clonazepam, lorazepam and diazepam, also have antipanic effects when administered in significant doses (Brown, Rakel, et al., 1991).

Tricyclic antidepressants, most notably imipramine hydrochloride, have been studied in depth as antianxiety agents (Brown, Rakel, et. al., 1991). Imipramine has been found to be more effective in the long run in treating panic, particularly when it is accompanied by depression. other tricyclic antidepressants such as trimipramine, amitriptyline, doxepin, nortriptyline and maprotiline have also been documented as effective in the treatment of panic (Lydiard & Ballenger, 1987). More recently, the use of low dose selective serotonin uptake inhibitors (SSRT) (fluoxeline) has demonstrated effectiveness in low doses (5 mg/day) (Schneier, F. R., Liebowitz, M. R., Davies, S. O., Fairbanks, J., Hollander, E., Campeas, R., and Klein, D. F. (1990).

As an alternative to the antidepressants, the monomania oxidase inhibitors (MAOI) have also been successful with individuals who suffer from panic associated with atypical depressive features. MAOIs, while effective, carry a number of serious side effects and require a strict dietary restrictions that exclude foods containing tyramine. MAOIs, like the tricyclic antidepressants require an onset period before any therapeutic effects may be gained.

Additional compounds, such as B-adrenergic blockers and have also been used to treat panic with increasingly effective results.

The combination of cognitive-behavioral techniques and pharmacological agents have been most successful in treating panic disorder. When pharmacological agents are used in conjunction with nonpharmacologic techniques, it is recommended that a multicomponent treatment package be used such as the one described by Craske & Barlow (1990). This program consists of four major components: (1) education and corrective information concerning the nature, etiology and maintenance of panic; (2) cognitive therapy techniques aimed at helping the patient identify, monitor an alter faulty appraisals of threat that contribute to panic occurrence; (3) framing in methods of slow diaphragmatic breathing as a way of reducing or eliminating physical symptoms that often trigger panic attacks and (4) interceptive exposure exercises designed to reduce patients' fear of somatic sensations through repeated exposure to feared bodily sensations. It is suggested thah it might also apply to emergency settings as well.

Bibliotherapy

One of the adjunct techniques that has proven very helpful with anxiety disordered individuals is the use of bibliotherapy (Gould, Clum, & Shapiro, 1993).

As a behavioral technique, bibliotherapy serves as a continuous reinforcement of many of the principles and concepts promoted in therapy regarding coping skills. Literature designed for clients may also serve to help individuals who are suffering from panic feel less of a sense of isolation and become aware that others experience these symptoms and struggle with the same reactions.

There are a number of excellent self-help books available on the market for panic sufferers, among them are "Don't Panic" by Reid Wilson (1986) and "Coping with Panic" by George Clum (1990). Both of these books have been written by professionals skilled in the cognitive-behavior therapies. As a result, they serve as fine supplemental reading and supportive aids to many of the aforementioned techniques described in this chapter. Individuals should be directed to read these books as they are receiving treatment and may even benefit from them subsequent to the termination of therapy.

Homework

Homework is a very important aspect of cognitive-behavioral treatment of panic. Many of the coping skills that are taught, require practice in order to become part of the individual's work repertoire of skills. Such homework assignments as the practicing of breathing exercises and cognitive coping skills are necessary to learn how to respond effectively when a spontaneous panic attack occurs.

Recording information on such forms as the "Weekly Panic Log" which was developed by the Center for Cognitive Therapy in Philadelphia (reprinted in Dattilio & Berchick, 1992) is also vital in allowing both the client and the therapist to track the occurrence and progress with the clients' panic occurrences.

Lastly, homework is also a prelude to the eventual coping skills that will be used in relapse prevention.

Relapse Prevention

Panic relapse after treatment occurs in the majority of cases as a result of the discontinuation of skills practice and exposure as well as poor follow-up in therapy. In fact, often times treatment is ended abruptly by the patient due to their

symptom remission.

It is essential that clients contract with the therapist to complete their treatment and include all of the follow-up visits. The follow-up visits should focus on a skills check and anticipation of the use of techniques in the event of a spontaneous reoccurrence of panic symptoms. other issues that should be addressed are psychosocial and internal stressors that may serve to trigger panic.

Finally, it is also recommended that patients be instructed not to delay in contacting the therapist for booster sessions when they are experiencing difficulty coping on their own. It is often the extreme delay that facilitates the return of the panic cycle in full force.

Conclusion: The treatment of panic in crises settings is ripe for the application of cognitive- behavioral interventions that build upon coping skills. The techniques proposed in this chapter are suggested as alternatives to the sole reliance on pharmacotherapy in treating panic in crises settings. Cognitive-behavioral techniques may prove to be most efficacious when used in conjunction with pharmacotherapy, especially alprazolam (Wardle, 1990; Sanderson& Wetzler, 1993).

It is recommended that the cognitive-behavioral techniques described in this article be considered prior to the use of pharmacological agents whenever possible (Solkol, Beck, Greenberg, Wright & Berchick, 1989). Medication may serve as an adjunct to cognitive-behavior therapy rather than the reverse. Continued research in this area is certainly warranted.

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