BRIEF FOCUSED COGNITIVE THERAPY OF PANIC DISORDER

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"Focused" cognitive therapy was evaluated in treatment of panic disorder. Panic was induced in-session through hyperventilation exercises, which activated catastrophic interpretations of specific physiological sensations. By allowing immediate focus on relevant "hot" cognitions, rapid treatment of the potentially disabling disorder was found to be possible.

While it has been argued that panic disorder is essentially the result of biochemical abnormalities associated with genetic predisposition (Sheehan, 1982), a recent literature review by Rapee (1987) concluded that psychological theories have been better supported by recent empirical research in this area. A central cognitive component of panic disorder is catastrophic misinterpretation of physiological sensations (Rapee, 1987). Consistent with this finding, physiological assessment comparing generalized anxiety disorder (GAD) patients with panic disorders (PD) patients has shown that PD patients have a significantly greater somatic component to their anxiety (Barlow et al., 1984). For example, PD patients were found to have a higher heart rate at each point in assessment compared to GAD patients (Barlow et al., 1984).

Furthermore, a study by Margraf, Ehlers & Roth (1987) has shown that providing false feedback regarding physiological functioning can produce a major spontaneous panic attack. In this study, a patient diagnosed as panic disorder was provided false feedback that her heart rate had suddenly accelerated, and a panic attack resulted. While the hypothesized specific ideational content (catastrophic misinterpretation) was not measured in this study (Margraf et al., 1987), the observed effects of information distortion are consistent with the cognitive model (Beck, Emery, & Greenberg, 1985).

As pointed out by Argyle (1988), given the cognitive model of panic which hypothesizes the central role of catastrophic interpretation of physiological responses, "Pure cognitive therapy might address the process of interpretation itself" (Argyle, 1988). The cognitive model would predict that (1) underlying catastrophic cognitive content can be identified in panic patients by skillful guided discovery in therapy, and (2) elimination of such interpretation will result in elimination of panic.

Subject

The patient was an 18-year-old female who met criteria for Panic Disorder with Mild Agoraphobia (DSM–III–R 300.21). Axis I diagnosis was made using the Structured Clinical Interview for DSM–III–R (SCID) (Spitzer, Williams & Gibbons, 1987). The patient reported that panic attacks had begun eight months earlier, that their average frequency was approximately four per month, and that their intensity had become increasingly more severe over the previous two months. She had first sought treatment for panic three days prior to referral to the Center for Cognitive Therapy, and at that time had rated panic attacks as "4—Extremely Severe" on a 1–5 point scale of severity on the Life History Questionnaire. There was no psychiatric history prior to seeking treatment for panic disorder, and the patient had never taken medications for emotional disorder.

This single-case study is of a patient treated by the first author while he was a postdoctoral Fellow at the Center for Cognitive Therapy of the University of Pennsylvania.

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Procedure

Instruments

The Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown & Steer, 1988) and the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock & Erbaugh, 1961) were administered prior to each therapy session. The Hopelessness Scale (HS) (Beck, Weissman, Lester & Trexler, 1974) was administered prior to the first and last sessions. Psychometric properties of the BDI were recently reviewed by Beck, Steer & Garbin (1988); and, properties of the BAI are described by Beck, Epstein, Brown & Steer (1988).

Symptoms assessed on these measures were judged during clinical evaluation and SCID assessment to be associated with or secondary to panic disorder. The evaluation and assessment was carried out by a postdoctoral clinical psychologist, with consultation by a second such psychologist who likewise met face-to-face with the patient to obtain independent agreement on diagnosis and presenting problems.

Panic attack (PA) frequency was monitored daily during treatment on a "panic log," with frequency from the preceding thirty (30) days being recorded by the patient from recall. The patient reported at least three distinct major attacks in restaurants over the previous thirty days, during each of which she left the restaurants due to panic. Over the same period, she had experienced three additional milder attacks, one on a subway, one in a psychologist's office during testing three days prior to her referral, and one in a theatre the day before treatment began.

Treatment

A total of four consecutive daily individual therapy sessions, approximately 50 minutes each, were conducted. By administering treatment over such a short time span, effects on outcome of extraneous variables which may occur over time, (e.g., maturation effects) were presumably minimized. The first treatment session focused on presentation of the cognitive model of treatment for panic. The therapist discussed with the patient the nature and symptoms of anxiety and panic, pointing out how degree of anxiety is a function of a person's perception of threat in relation to perception of resources available to cope with the threat. This was described as the "risk/resources ratio," and was presented to explain how anxiety is a normal response to the sense of vulnerability. The role of the autonomic nervous system in panic disorder was discussed along with the catastrophic misinterpretation model, with emphasis on the relationship between physiological sensations, misinterpretation, and panic.

During the initial evaluation, the patient had described phenomenologically her panic experience as being "like when you're playing dodge ball when you're a kid, you know, that panic, like when there's that second when you're not sure whether that ball's going to hit you or not." During the onset of treatment, she appeared aware only of what she called "fear" during the panic attacks. The goal of therapy was first to recover automatic thoughts underlying this affect. A precise description of the patient's typical panic attack was therefore obtained, and the following panic scenario was identified: (1) nausea in stomach, (2) heart racing, (3) shaky, (4) muscle tension, (5) difficulty breathing, (6) leave the situation. When asked, "Before you leave (panic situations), do you notice any images or thoughts?," she replied, "I never really thought about that." However, when asked "Do the symptoms themselves scare you?," she replied emphatically, "Oh, yeah!". The following dialogue demonstrates how subsequent discussion in this area assisted in orienting the patient to the catastrophic misinterpretation model:

PT: I've felt a couple of times like I was going to have a heart attack.

TH: Tell me about that.

PT: It's just like, it starts to beat so hard, and so fast, that, I mean, it *hurts*, and you just think, you're just like sitting there going (breaths rapidly to demonstrate how she hyperventilates).

TH: What was the effect, do you think, of that particular image or idea on the symptoms themselves?

PT: Oh, well, they get worse, as soon as you think . . . (pause).

TH: So you have a bodily sensation here at this point in the spiral of anxiety (pointing to diagram). The next step, thoughts that the sensation is a sign of catastrophe, that you're going to have a heart attack. What do you think happens to your thoughts when your heart rate does in fact go up? Because you said it would increase when you had this fear . . .

PT: Uh-huh.

TH: What would you think then?

PT: I just get really scared, and I have to get out of the situation that I'm in. I mean, I *have* to.

TH: O.K. So you see what's happening there. You perceive a danger, which would lead to more anxiety; the anxiety would make the symptoms worse, would make the heart rate even faster; and, you interpret that, then, as "I really *am* going to have a heart attack." Right?

PT: Yeah, but only fleetingly, you know, and then I go "I'm not going to have a heart attack, but I've got to get out of here!"

TH: O.K.
PT: But, I mean, *fleetingly*, I've also thought all kinds of, you know, I'm sure . . .

A second, but related, goal of therapy was treatment of secondary hopelessness and depression associated with the patient's panic disorder. Because the patient could see objectively (when not experiencing panic) that her behavior was unrealistic, she had additional automatic thoughts that her panic response meant she must be a "weak and pathetic" person. To counteract this process, reattribution was used as a strategy to challenge these negative views of self. Reattribution focused on the explanation that misinterpretation of physiological sensations and the subsequent vicious cycle could better account for her panic experiences. To further enhance self-confidence, strategies of distraction and "rational response" were introduced as techniques she could learn in order to interrupt the catastrophic interpretation, and, therefore, the panic itself.

During the second and third therapy sessions, in-office "panic inductions" were used to create immediate experience of panic sensations. (The patient had received a recent complete physical examination, and had no medical condition which would be adversely affected by such a procedure.) The patient was instructed to hyperventilate, and this produced the following report of sensations: (1) feelings of unreality, (2) heart racing, (3) shakiness, (4) heart pounding, (5) tension, (6) tight muscles, and (7) breathlessness. In comparing these sensations to those associated with a typical panic attack, the patient found them to be highly similar. This demonstrated that normal physiological and psychological response to hyperventilation could create sensations like those experienced during spontaneous panic attacks. From this point in treatment, the patient was led to evaluate further the meaning of this discovery. Consistent with cognitive theory regarding panic disorder etiology, in-office panic induction was followed by guided discovery to facilitate decatastrophizing of specific physiological sensations misinterpreted by the patient as dangerous. Collaborative exploration of sensations and thoughts following panic induction found the core cognitive component of this patient's panic disorder to be interpretation of rapid, pounding heartbeat as signaling possible "heart attack."

Hyperventilation was not used again to induce panic during session four (the final session). Rather, session four focused on review of the cognitive model of panic, and rehearsal of realistic cognitive response to automatic thoughts associated with sensations previously misinterpreted in the manner delineated above. Another focus of session four was discussion of success the patient had experienced the day before (following session three) in returning to eat in a restaurant where she had previously experienced severe panic, a place which she had previously been avoiding. While she identified previous cognitive response to pounding heartbeat in that restaurant as "I'm going to die," she reported that, this time, "Every time my heart rate increased I'd say, it's O.K., it's no big deal, I'm just sitting here in the restaurant, it's O.K." When asked, "How strongly did you believe the rational response?," she replied, categorically, "I believed it." It was clear that the course of brief focused cognitive therapy administered to this particular patient had been instrumental in modifying the meaning she attributed to rapid pounding heartbeat associated with anxiety. Consequently, she reported an anxiety level in that situation of only 2 on a scale of intensity from 0 (none) to 10 (most severe). By interrupting the "vicious cycle" through realistic cognitive response, anxiety level did not escalate to the point of panic in this previously feared situation.

Treatment Integrity

Treatment integrity essentially refers to whether a specific treatment is administered, and received, as intended (Vermilyea, Barlow & O'Brien, 1984; Yeaton & Sechrest, 1981). To evaluate treatment administered in the present study, the Cognitive Therapy Scale (Vallis, Shaw & Dobson, 1986) was used by an experienced cognitive therapist (the Director of Training of the Center for Cognitive Therapy) to evaluate videotapes of therapy sessions for specific process and content.

Successful treatment was further evidenced in that the patient demonstrated her understanding of the cognitive model of panic through her responses during treatment sessions, and she reported using specific cognitive techniques, taught during therapy, in panic situations outside the therapist's office. For example, she reported at the beginning of the second session that she "tried rational responding last night, whenever I had a negative automatic thought." She spontaneously reported during the third session that she had used distraction when panic sensations began while she was on a bus, stating she had focused her attention on a man's shoes to distract herself from symptoms in

that situation. During the fourth and final daily session, as was noted above, the patient reported utilizing what she had learned in cognitive therapy to prevent onset of panic in the restaurant where she had previously experienced her most severe panic attacks prior to treatment.

Results

As treatment progressed over the four daily sessions, the patient reported a clinically significant decrease in symptoms of anxiety, depression, and hopelessness judged during previous clinical evaluation to be secondary to panic disorder (see Table 1).

There was one panic attack reported the day prior to the first session, but none on days following the first, second, third, or fourth sessions. Additionally, the patient reported that she was not avoiding situations where she had previously experienced panic, but rather was using cognitive skills learned in treatment to manage panic symptoms in these situations. Furthermore, during one-month and five-month follow-up interviews, the patient stated that she had experienced no panic attacks since treatment, and that she continued to approach situations which had previously been associated with panic attacks.

Discussion

Upon identification of physiological sensations and negative automatic thoughts associated with the sensations, the patient was able to rapidly obtain some distance from her fearful thoughts. Results of the present case study appear consistent with observations made recently (Baumbacher, 1989) regarding the role of "signal anxiety" in etiology of panic disorder. Baumbacher conceptualizes signal anxiety as "a subjective experience that may be misperceived or not perceived for multiple reasons" (Baumbacher, 1989). He goes on to elaborate the manner in which this misperception, or lack of perception, may lead to onset of panic disorder. Cognitive therapy of panic disorder is designed to enhance sensitivity to (and realistic interpretation of) normal physiological responses or sensations associated with anxiety. If misperceived, such responses can escalate via catastrophic misinterpretation (Argyle, 1988; Beck, Laude & Bohnert, 1974; Hibbert, 1984; Margraf et al., 1987) in a "vicious cycle" (Beck & Emery, 1979; Beck et al., 1985; Rapee, Mattich & Murrell, 1986) leading to panic. For example, in the present case study, pounding heartbeat was misinterpreted as impending "heart attack," which resulted in increased fear and, therefore, further increased heart rate.

Likewise, if the relevant physiological sensations and associated cognitions (referred to by Baumbacher (1989) as "signal anxiety") are not consciously perceived, symptoms may escalate to the point of panic. While further development in clinical cognitive assessment may assist in addressing this issue (Parks & Hollon, 1988), given the often idiosyncratic nature of distortions in interpretation of physiological sensations, there may be no substitute for clinical skills focused precisely on uncovering the cognitive components responsible for activation of panic. As shown in this study, the collaborative empirical nature of cognitive therapy is specifically designed to identify and directly address this process of interpretation, allowing more rapid treatment of this potentially disabling disorder.

TABLE 1. Assessment of Anxiety, Depression, Hopelessness, and Panic Attack Frequency throughout Therapy

Measure	Session 1	Session 2	Session 3	Session 4
BAI[a]	23	7	11	5
BDI[b]	19	12	13	8
HS[c]	12	—	—	6
PA[d]	1	0	0	0

[a]Beck Anxiety Inventory.
[b]Beck Depression Inventory.
[c]Hopelessness Scale.
[d]Panic Attacks.

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