

Dismantling Cognitive—Behavioral Treatment for Panic Disorder Questioning the Utility of Breathing Retraining

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ABSTRACT

Cognitive—behavioral treatment (CBT) protocols for panic disorder (PD) consist of a set of interventions that often includes some form of breathing retraining (BR). A controlled outcome study was designed to assess the necessity of BR in the context of a multicomponent CBT protocol. To accomplish this, patients with PD ($N = 77$) were randomly assigned to receive CBT with or without BR or to a delayed-treatment control. The main study hypothesis was that patients receiving BR would display a less complete recovery relative to the other active-treatment condition given that BR appears to be a more attractive (but less adaptive) option for some patients. Some data suggested that the addition of BR yielded a poorer outcome. However, findings were generally more consistent with treatment equivalence, questioning whether BR produces any incremental benefits in the context of other CBT interventions for PD.

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The evolution in psychological treatments for panic disorder has been rapid and exciting during the past 15 years ([Wolfe & Maser, 1994](#)). Historically, the practice of encouraging patients to repeatedly confront situations that produce intense fear and avoidance has been the hallmark of behavioral treatments for agoraphobia and panic. More recent cognitive models of panic have offered new directions for intervention ([Barlow, 1988](#) ; [Clark, 1986](#)). Within the cognitive framework, panic attacks are conceptualized as the result of catastrophic misinterpretation of benign bodily sensations that are typically involved in the normal anxiety response (e.g., heart palpitations, dizziness, and dyspnea). Cognitive—behavioral therapy (CBT), derived from this cognitive framework, has been found to demonstrate good efficacy in controlled trials using both individually ([Barlow, Craske, Cerny, & Klosko, 1989](#)) and group-administered ([Telch, Lucas, et al., 1993](#)) treatment.

The newer CBT treatment protocols focus on correcting the patient's hypersensitivity to bodily sensations and the misinterpretation of these sensations as signaling immediate threat. Typically, these treatments consist of a set of interventions including (a) education; (b) training in cognitive reappraisal; (c) repeated exposure to bodily sensations connected to the fear response (i.e., interoceptive exposure); (d) repeated exposure to external situations connected to the fear response (i.e., in vivo exposure); and (e) training in breathing-control techniques, such as diaphragmatic breathing.

One of the next steps in the process of developing increasingly effective treatments for panic disorder is determining which components of those included in these multicomponent treatment protocols are most effective and which are extraneous or even maladaptive. Several studies have dismantled various components of CBT for panic disorder, including evaluation of cognitive restructuring alone ([Salkovskis, Clark, & Hackmann, 1991](#)), as well as comparisons of cognitive restructuring versus in vivo exposure ([Margraf, Gobel, & Schneider, 1989](#)).

The breathing retraining (BR) component of CBT protocols has also been evaluated to some extent. One older study found that the combination of BR and in vivo exposure is somewhat better than the individual effects of either intervention ([Bonn, Readhead, & Timmons, 1984](#)). However, more recent studies that include the cognitive and interoceptive components lead to questions about the incremental benefits of BR. Several studies have suggested that relatively little is gained from adding cognitive restructuring and BR to in vivo exposure ([de Ruiter, Ryken, Garssen, & Kraaimaat, 1989](#) ; [Telch, Schmidt, Kamphuis, Jaimez, & Frank, 1993](#)). [Craske, Rowe, Lewin, and Noriega-Dimitri \(1997\)](#) compared the relative efficacy of interoceptive exposure and BR in the context of the other CBT components and found that interoceptive exposure is relatively more potent compared with breathing-control exercises. No studies, however, have directly evaluated the addition of BR to the other main components of CBT (i.e., education, cognitive restructuring, interoceptive exposure, and in vivo exposure).

The theoretical basis for scrutinizing BR goes to the core of our treatment approach to panic disorder. In line with cognitive models of panic and panic disorder, it is assumed that this disorder results from fully correctable processes involving faulty learning and misperception ([Barlow, 1988](#) ; [Clark, 1986](#)). This treatment approach emphasizes the "complete" resolution of these problems, not merely learning to cope with the symptoms of anxiety and panic. Accordingly, panic disorder is described as a "fear of fear," and recovery is defined as no longer fearing panic and anxiety symptoms. Consistent with this idea, behaviors that are designed to avoid or minimize panic symptoms are deemed maladaptive. These behaviors are termed *false safety aids* because they attempt to keep the person "safe" from a false threat (i.e., panic and high anxiety; [Kamphuis & Telch, 1998](#)). For example, leaving the room or using cognitive distraction techniques when anxiety rises is a false safety aid used to prevent the experience of panic.

This treatment orientation emphasizes the need to eliminate all false safety aids because their use may contribute to or maintain the anxiety problem by preventing corrective learning experiences. Consistent with emotional processing views of fear reduction ([Foa & Kozak, 1986](#)), one potential reason that patients fail to show recovery despite multiple exposures to feared situations is that they are engaging in safety aids that disallow corrective learning experiences (i.e., coping may minimize the experience of anxiety).

Teaching breathing-control techniques in the context of the false safety aid model is somewhat antithetical because this process involves instructing patients in the use of a new coping skill or "safety aid." In treatment groups, patients often recognize that this intervention is a safety aid. When this occurs, the therapist's response is to confirm that breathing-control techniques are, indeed, safety aids. It has been suggested, however, that safety aids fall along a continuum of maladaptation. Thus, patients are told that some false safety aids are less adaptive (e.g., phobic avoidance of feared situations) than others (e.g., diaphragmatic breathing). The therapist explains that part of the process of recovery is to replace more maladaptive safety aids with less maladaptive ones, although preferably patients would eventually engage in no safety aids.

A controlled outcome study was designed to assess the necessity of BR in the context of a multicomponent CBT protocol. To accomplish this, we randomly assigned patients with panic disorder to receive CBT (i.e., education, cognitive restructuring, and interoceptive and in vivo exposure) with or without BR relative to a delayed-treatment control (WL). The main study hypothesis was that patients receiving BR would be more likely to display a less complete recovery. Consistent with speculation by [Craske et al. \(1997\)](#), who suggested that BR may become a countertherapeutic false safety aid, this hypothesis is premised on the belief that even though both groups have the option of using any or all of the other treatment components, BR appears to be a more attractive (but less adaptive) option for some patients. More specifically, we hypothesized that patients receiving BR would show improvement relative to those in WL but would be more symptomatic relative to those patients in the other active-treatment condition. During follow-up, we expected that patients receiving BR would continue to show greater symptoms and would be more prone to relapse relative to the other active-treatment group.

Method

Participants

The sample consisted of 77 patients meeting the following criteria: (a) principal *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM—IV*; [American Psychiatric Association, 1994](#)) Axis I diagnosis of panic disorder with or without agoraphobia; (b) no change in medication type or dose during the 12 weeks prior to treatment; (c) no evidence of serious suicidal intent; (d) no evidence of current substance abuse; and (e) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder. Sixty-nine percent of the participants were female, with an average age of 37.8 years ($SD = 11.1$). A majority of the patients were Caucasian (83%), married (60%), and employed full-time (77%). Fifty-two percent of the patients received at least one current co-occurring Axis I diagnosis, with 32% reporting another anxiety disorder diagnosis, 14% reporting a mood disorder diagnosis, and 6% reporting additional anxiety and mood disorder diagnoses. Fifty-four percent of the patients were taking psychotropic medications (23% benzodiazepines, 15% antidepressants, and 3% beta-blockers), with 13% of the sample taking both benzodiazepines and antidepressants. Patients on medications met stability requirements such that initial administration of the medication and dose had been maintained for at least 3 months prior to treatment and was maintained until posttreatment.

Procedure

Patients were consecutive applicants presenting for evaluation at an academic research center specializing in the assessment and treatment of anxiety disorders who met the study criteria. Diagnostic assessment was based on an initial phone screening interview followed by a face-to-face structured clinical interview using the Structured Clinical Interview for *DSM—IV*—Patient Edition ([First, Spitzer, Gibbon, & Williams, 1994](#)). Randomly selected videotaped interviews from this sample ($n = 16$) have demonstrated perfect interrater agreement for the panic disorder diagnosis ($\kappa = 1.0$).

After 5 to 7 treatment-eligible patients had completed the baseline assessment, this "group" was assigned to one of three conditions: CBT with BR (CBT + BR), CBT without BR (CBT), or WL. Five groups ($n = 32$) were assigned to the CBT condition and received a group-administered cognitive—behavioral treatment that consisted of 12 sessions over a 12-week period (cf. [Schmidt, Staab, Trakowski, & Sammons, 1997](#)). Each session lasted approximately 120 min. The treatment protocol includes four major components: (a) education and corrective information regarding the etiology and maintenance of panic disorder, (b) cognitive therapy techniques aimed at helping the patient to identify and alter faulty appraisals of threat that contribute to panic occurrence, (c) interoceptive exposure exercises that are designed to reduce patients' fear of somatic cues through the repeated exposure to feared bodily sensations, and (d) instruction for conducting in vivo exposure exercises designed to reduce patients' fears of external situations through the repeated exposure to a fear hierarchy. Patients assigned to the treatment conditions were reassessed immediately following treatment, 6 months after treatment, and 12 months after treatment.

Four groups ($n = 21$) were assigned to the CBT + BR condition, which was identical to the CBT condition except for the addition of instruction in BR exercises (i.e., diaphragmatic breathing) plus related practice homework. This intervention was added to Sessions 4 and 5 (see [Telch, Lucas, et al., 1993](#)). In this condition, these sessions ran approximately 30 min longer per session to control for the relative time allotted to each of the other interventions across the active-treatment groups. Thus, the BR was provided in addition to all the other interventions. Four groups ($n = 24$) were assigned to the WL condition. Patients in this condition were reassessed after 12 weeks and then received treatment but were not assessed further.

In the active-treatment groups, treatment integrity was maintained by using a structured and manualized treatment protocol ([Schmidt, 1994](#)) that describes the specific goals and strategies for each session. In our lab, an independent rater's assessment of adherence ([Young, Beck, & Budenz, 1983](#)) to the treatment protocol has yielded extremely high rates of adherence (see [Schmidt & Woolaway-Bickel, 2000](#)). In the present study, an independent rater viewing 20 hr of randomly selected sessions indicated no adherence differences across the two active-treatment groups. Norman B. Schmidt administered the treatment to all the groups. He is a licensed clinical psychologist with approximately 10 years of experience with cognitive—behavioral treatment of anxiety disorders. In each group, there was also a graduate fellow in clinical psychology, a psychiatry resident, or a psychiatrist acting as cofacilitator.

Measures

A multimodal assessment battery tapping the major clinical dimensions of panic disorder was administered to all the participants at baseline and posttreatment. The same battery was administered to those in the active-treatment groups at follow-up. Clinician ratings were made by raters unaware of treatment condition.

Multicenter Panic Anxiety Scale (MC—PAS).

The MC—PAS (formerly the CY—PAS) is a semistructured interview rating scale for panic disorder ([Shear et al., 1997](#)) that includes ratings of panic frequency and intensity, anticipatory anxiety, avoidance of sensations and situations, and impairment in work and social functioning. Each of these symptoms is rated on a scale ranging from 0 (*none*) to 4 (*extreme*). The MC—PAS has good psychometric properties ([Shear et al., 1997](#)). In the present sample, two clinicians in a dual interview making MC—PAS ratings for a subsample ($n = 25$) were found to have consistently high reliability ($r = .71—.94$).

Sheehan Patient-Rated Anxiety Scale (SPRAS).

The SPRAS ([Sheehan, 1983](#)) is a widely used self-report scale for assessing the intensity of anxiety symptoms. The SPRAS has demonstrated adequate test—retest reliability ($r = .67$) and is highly associated with other measures of anxiety and overall impairment in panic disorder samples ([Schmidt, Staab, et al., 1997](#)).

Mobility Inventory (MI).

The MI was used to assess phobic avoidance ([Chambless, Caputo, Jasin, Gracely, & Williams, 1985](#)). The MI includes two subscales for determining level of phobic avoidance: When Alone and When Accompanied. The subscales are scored separately and have been found to possess good psychometric properties in clinical samples ([Chambless et al., 1985](#)).

Sheehan Disability Scale (SDS).

The SDS is a four-item self-report measure of impairment created by the presenting problem ([Ballenger et al., 1988](#)). One representative item from this scale tapping overall work and social impairment was used in the present report. This item is associated with clinical global ratings of impairment and quality of life in panic disorder samples ([Schmidt & Telch, 1997](#)).

Beck Depression Inventory (BDI).

Level of depressive symptoms was assessed by the BDI. The BDI is a reliable and well-validated measure of depressive symptomatology ([Beck & Steer, 1993](#)).

Composite Measure of End-State Functioning

In addition to self-rated disability and clinician-rated impairment, a composite measure of clinically significant change was computed for evaluation of end-state functioning at posttreatment. This composite measure provides an estimation of clinical significance (see [Kendall, Marrs-Garcia, Nath, & Sheldrick, 1999](#)). Specifically, a measure of high end-state functioning addresses questions regarding the degree to which patients are distinguishable from nonclinical controls. In this particular case, a patient was classified as having high end-state functioning when scores on each of three central symptom dimensions of panic disorder fell within the normal range of functioning (i.e., panic frequency = 0, anxiety [the SPRAS] <30, and phobic avoidance [the MI When Alone and When Accompanied subscales] <1.5; [Telch, Lucas, et al., 1993](#)). Note that the time frame for assessing panic frequency differed at posttreatment (i.e., during previous week) and follow-up (i.e., during previous 6 months).

Results

Analytic Method

We evaluated pretreatment between-groups differences and differential treatment effects using random-effects regression models ([Bryk & Raudenbush, 1987](#) ; [Laird & Ware, 1982](#) ; [Nich & Carroll, 1997](#)). A random-effects regression approach models changes over time at the population, as well as the individual, level. As a first step, person-specific random effects are included in a regression equation with the fixed effects of the independent variables to predict values for the dependent variable over time and to provide an intercept and slope for each participant. As a second step, between-groups differences are measured with analysis of variance—type models on the intraindividual values calculated during Step 1. A random-effects regression approach offers a number of advantages over other statistical methods in dealing with some of the problems often associated with longitudinal data, including missing data, irregularly spaced measurement intervals, and person-specific deviations from the group average response trend ([Gibbons et al., 1993](#)).

In the present study, the primary hypothesis involved the differential effectiveness of CBT, versus CBT + BR, compared with WL. The a priori contrasts in this analysis tested the null hypothesis of (a) no differences between the two active-treatment groups (CBT vs. CBT + BR), (b) no differences between CBT and WL, and (c) no differences between CBT + BR and WL. We conducted random-effects regression models for an ever-followed sample who provided posttreatment data ($n = 67$), as well as for the intention-to-treat sample ($n = 72$). For all the analyses, time and the intercept were treated as random without autocorrelated errors.

Preliminary Analyses

Preliminary analyses for the association between demographic variables and treatment group assignment indicated no significant differences. Similarly, there were no differences in regard to psychiatric comorbidity or percentage of medicated participants across groups. Session 1 ratings of treatment credibility following introduction of the treatment components (i.e., rating beliefs regarding the degree to which the treatment is perceived to be potentially helpful, logical, and reasonable and the degree of confidence in recommending the treatment to others) did not differ across the active-treatment groups.

Parameter estimates indicated no significant group differences on initial severity (i.e., no significant intercept differences) of pretreatment self-report and clinician ratings, suggesting that randomization was successful in minimizing symptomatic differences across groups.

Treatment Outcome at Posttreatment

[Table 1](#) displays parameter estimates, standard errors, and test statistics for all the outcome measures from pretreatment through posttreatment. Rate of improvement is represented in the slopes for each condition, with significant slope estimates indicating significant change (improvement) over time. The three a priori slope contrasts are also provided. These contrasts estimate differential change in improvement over time.

Consistent with previous reports demonstrating the efficacy of CBT for panic disorder, the pattern of findings at posttreatment attests to the benefits of CBT relative to WL. Both of the active-treatment groups showed significant rates of improvement over time except in one instance (i.e., the CBT + BR group for the MI

When Accompanied subscale). The WL group also showed significant improvement over time on three measures, suggesting some regression to the mean/spontaneous recovery effects.

Planned contrasts indicated that the treated groups generally showed a differentially greater rate of improvement relative to the WL group. The CBT group did not differentiate itself from the WL group on 1 outcome variable (the MI When Alone subscale), and the CBT + BR group did not differentiate itself from the WL group on 4 out of 12 outcome variables. This pattern of findings is consistent with the hypothesis that the CBT + BR patients did not fare as well as the CBT group, but contrasts between active-treatment groups did not reach significance.

Evaluation of the composite recovery measure indicated that 38% of the CBT group met the recovery criteria for high end-state functioning compared with 21% of the CBT + BR group and 0% of the WL group. We also computed random-effects regression models for the composite recovery variable using the MIXOR program ([Hedeker & Gibbons, 1996](#)), which handles dichotomous dependent variables. Random effects, however, could not be reliably estimated using this method. Logistic regression analyses indicated significant group differences for both active-treatment groups relative to the WL group: for the CBT group, $\chi^2(1, N = 53) = 15.60, p < .0001$; for the CBT + BR group, $\chi^2(1, N = 43) = 7.06, p < .01$. The difference between the active-treatment groups did not reach significance, $\chi^2(1, N = 48) = 1.57, p > .10$.

Follow-Up Differences Between the Active-Treatment Groups

[Table 2](#) shows rates of change (slopes) and slope contrasts for the active-treatment groups from pretreatment through the 12-month follow-up period. These analyses included available data for pretreatment, posttreatment, and 6- and 12-month assessment periods. Slope estimates indicated significant changes (improvement) for both groups over time on all the outcome measures. As can be seen by these estimates, the CBT group showed a somewhat higher rate of improvement on all the measures except one. Slope contrasts, however, did not reach significance, although there were trends for the CBT group to show differentially better improvement on panic frequency (the MC—PAS Panic Frequency subscale), anticipatory anxiety (the MC—Anticipatory Anxiety subscale), avoidance when accompanied (the MI When Accompanied subscale), and overall disability (the SDS). By the 12-month follow-up, evaluation of the composite recovery variable indicated that 57% of the CBT group met the recovery criteria compared with 37% of the CBT + BR group, $\chi^2(1, N = 42) = 1.59, p > .10$.

Attrition and Intent-to-Treat Analyses

There were 5 dropouts at posttreatment. Two (10%) patients dropped out of the CBT + BR group, 3 (10%) dropped out of the CBT group, and no patients dropped out of the WL group. In three cases, attrition was the result of a logistic problem (e.g., moving or taking a new job during treatment). The other two cases were undetermined. Attrition was not statistically significant across groups, $\chi^2(2, N = 77) = 3.82, p > .10$. Attrition at follow-up was defined as either (a) lost contact or the follow-up not completed because the clinical trial ended prior to the follow-up period or (b) patient reported a relapse, was provided additional treatment, and was excluded from follow-up. We were unable to contact or schedule a follow-up assessment for 11 patients (6 CBT, 5 CBT + BR) during the 12-month follow-up period. This was not a significant group difference, $\chi^2(1, N = 41) = 0.65, p > .10$. Five patients (1 CBT, 4 CBT + BR) who contacted the project during the follow-up period reporting some increase in symptomatology were retreated and not followed. This gauge of relapse approached significance, $\chi^2(1, N = 48) = 3.82, p = .05$, and suggested that patients in the CBT + BR group were somewhat more prone to seek additional treatment

during the follow-up period. Note that relapse is defined in a manner that is distinct from end-state functioning. Our measure of end-state functioning captures the percentage of patients meeting the criteria for "normal" levels of functioning, whereas our measure of relapse captures the percentage of patients who showed the worst possible outcome (i.e., pretreatment or worse levels of symptoms suggesting a need for additional treatment).

We computed intent-to-treat analyses using random-effects regression models for the pretreatment—posttreatment and pretreatment—follow-up data. In the case of missing data, the last available value provided the basis for all additional time points. The pattern of findings is highly similar to those reported for the completer analyses, with only two exceptions. Two significant pretreatment—posttreatment contrasts were reduced to trends. The CBT + BR versus WL contrast for anticipatory anxiety was no longer significant (estimate = -0.60 , $SE = 0.40$, $z = -1.52$, $p = .13$), and the CBT versus WL contrast for disability was no longer significant (estimate = -0.63 , $SE = 0.36$, $Z = -1.75$, $p = .08$).

Moderator Analyses: Medication Status and Psychiatric Comorbidity

We also conducted tests of moderator effects for two potential moderators (medication status and psychiatric comorbidity) using a regression approach described by [Baron and Kenny \(1986\)](#). A significant percentage of patients were medicated (for the BR group, 45%; for the CBT + BR group, 60%; and for the WL group, 50%). Because medication use is a prominent safety aid, a penchant toward medication usage may interact with overuse of BR. We evaluated the interactive effects of medication status during the treatment (yes or no) and treatment condition on outcome. Regression analyses examined the singular effects of condition and medication status, as well as their interaction, on residualized change at posttreatment and follow-up. These analyses revealed no significant main effects for medication use and no significant interactions.

A significant percentage of patients were found to meet the diagnostic criteria for additional Axis I conditions (for the BR group, 50%; for the CBT + BR group, 56%; and for the WL group, 50%). Because psychiatric comorbidity has been found to predict poorer outcome for patients with panic disorder in the context of CBT ([Brown, Antony, & Barlow, 1995](#)), we evaluated the interactive effects of comorbidity (yes or no) and treatment condition on outcome. Regression analyses examined the singular effects of condition and comorbidity, as well as their interaction, on residualized change at posttreatment and follow-up. Consistent with previous work, these analyses revealed several significant main effects (at posttreatment and follow-up), suggesting that comorbid patients had relatively poorer outcomes, but there were no significant Comorbidity \times Condition interactions.

Discussion

The present study was designed to assess whether BR contributes to a battery of interventions that are more directly focused on correcting key problem areas (i.e., cognitive misappraisal and interoceptive conditioning) that, at least according to cognitive behavioral models, are believed to contribute to panic disorder. This study suggests that the addition of a coping skill—specifically, diaphragmatic breathing—does not yield any clear benefits to a treatment package consisting of education, cognitive restructuring, and exposure-based techniques (i.e., both in vivo and interoceptive). Moreover, some data suggest that introduction of BR may adversely affect patients, leading to less complete recovery and greater risk for relapse.

There is little question that BR has been shown to provide some level of symptomatic relief for patients with

anxiety disorders ([Clark, Salkovskis, & Chalkley, 1985](#)). This may be particularly true when patients are reporting salient respiratory symptoms, even though there are few data to suggest that these patients are acutely hyperventilating in the context of panic attacks ([Garssen, Buikhuisen, & van Dyke, 1996](#) ; [Hibbert & Pilsbury, 1988](#)). The present study suggests, however, that BR may add little to other components of the CBT intervention package. Further work is needed to determine whether these findings also apply to individually administered CBT.

Variation in therapy contact time strengthens this point. Controlling for contact time across conditions may favor the abbreviated intervention program because a less complex treatment program may create differentially greater patient expertise with treatment-related skills. Varying time across conditions to allow for sufficient coverage of all skills controls for this potential problem, although it may give patients in the lengthier condition an advantage because they have more therapy time. In the present study, there was no apparent benefit for those in the CBT + BR group despite increased contact time.

A related question is whether BR is actually contraindicated for patients with panic disorder receiving multimodal CBT. Data from the present study are less clear with regard to this question because the active-treatment groups were statistically similar on the outcome measures. A number of findings suggest, however, that patients receiving BR exhibited trends toward poorer end-state functioning on both self- and clinician-rated measures. Typically, it is difficult to show statistically significant differences between active treatments because of relatively low power. The present study is no exception; nevertheless, despite low power, some nonsignificant trends emerged in the hypothesized direction. If one assumes that active treatments produce moderate to large effect sizes in predicting outcomes ($r^2 = .13$ — $.20$), a cell size range of 20 to 38 would be needed to adequately power a small effect size ($r^2 = .01$ — $.05$) increment for an interaction between treatments (i.e., testing differential treatment effects). It would be expected that larger trials would confirm these differences.

The age of managed care has made it apparent that cost-effectiveness considerations, including limiting treatment length, are critical in the pragmatics of designing treatment plans. The present data, as well as [Craske et al.'s \(1997\)](#) data, would suggest that, given a choice, it is preferable to use interoceptive exposure versus BR. If there are a limited number of sessions, therapists would likely produce better outcomes with the use of cognitive and interoceptive techniques versus teaching a coping skill such as diaphragmatic breathing. It remains to be tested, however, whether this follows for other related types of techniques, such as progressive muscle relaxation.

Teaching patients various coping techniques is a common component to many different types of psychotherapy. In light of cognitive conceptualizations of panic disorder, it is useful to highlight the idea that teaching such techniques is theoretically antithetical to the goals of current cognitive models. These models clearly specify that the disorder is fully correctable. Often therapists are tempted to provide a coping skill believing that it will provide the most immediate relief to the patient. Although there certainly may be situations when some immediate coping skill (or medication referral) is needed to reduce distress to a manageable level in patients with panic disorder, we suggest that clinicians use careful judgment about the introduction of such strategies. The early introduction of coping strategies may run an increased risk of "safety aid substitution," perhaps making patients prone to relying on coping skills at the expense of corrective interventions.

We have suggested that therapists refrain from the use of respiratory-control techniques as a means for coping with or managing anxiety. On the other hand, respiratory interventions may still have some role in the

treatment of panic disorder. There are at least two types of interventions pertaining to respiration that are likely to be helpful to patients with panic disorder that would not directly lead to coping strategies. These techniques include educating patients about the natural role of hypocapnia in fear responses and manipulating respiration rate as a corrective intervention during interoceptive exposure. To debunk unrealistic ideation surrounding anxiety reactions, it is useful to educate patients about respiratory physiology, particularly the types of bodily cues that are created by hyperventilation. Patients may also benefit from directly experiencing the relationship between respiration rate, respiratory alkalosis, and anxiety. Interoceptive exposure often involves repeated intentional hyperventilation. The goal of these exercises is for patients to learn that they do not need to fear bodily perturbations produced by hypocapnia.

The present study did not assess patients' specific use of each of the skills introduced during treatment. This is an obvious limitation because we can presume only that patients exposed to the BR intervention may have relied on this intervention and that differential reliance on this technique accounted for differences across groups. Alternatively, the presentation of respiratory-control coping skills in the group may have given patients an implicit message that the general use of coping skills for managing or avoiding panic is acceptable such that these patients were more likely to use other safety aids (e.g., meditation or cognitive distraction). Assuming that use of specific interventions can be reliably assessed, evaluation of the degree to which each patient used diaphragmatic breathing techniques would be one method for providing a more fine-grained analysis of the hypothesis that these coping skills techniques are less productive than other CBT interventions. Future studies should also consider identifying the use of protocol and nonprotocol interventions (e.g., relaxation procedures) to conduct a more detailed analysis of the impact of specific interventions on outcome.

In summary, there is little doubt that current multicomponent cognitive—behavioral treatments for panic disorder represent first-line treatments of choice. More recent reports, including the present study, suggest that it will be useful to begin to dismantle these highly effective treatment packages. Ultimately, we are likely to find that specific components of these protocols, such as respiratory-control techniques, are extraneous and that our therapies should concentrate on more potent interventions, such as interoceptive exposure and cognitive restructuring.

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Table 1. Parameter Estimates, Standard Errors, and Test Statistics: Pretreatment Through Posttreatment

Table 2. Parameter Estimates, Standard Errors, and Test Statistics: Pretreatment Through 12-Month Follow-Up

Table 3
Annual Through, Landing, Stock, and Release Information
Product: US West Kallin Gy

| Season | Stock | | Release |
|---|-------------|-------------|-------------|
| | CBQ | CBQ + 50 | |
| Chromosome 1000 to 10000 | | | |
| 1998-1999 | 1000 | 1000 | 1000 |
| 1999 | 1000 | 1000 | 1000 |
| 2000 | 1000 | 1000 | 1000 |
| Chromosome 10000 to 100000 | | | |
| 1998-1999 | 10000 | 10000 | 10000 |
| 1999 | 10000 | 10000 | 10000 |
| 2000 | 10000 | 10000 | 10000 |
| Chromosome 100000 to 1000000 | | | |
| 1998-1999 | 100000 | 100000 | 100000 |
| 1999 | 100000 | 100000 | 100000 |
| 2000 | 100000 | 100000 | 100000 |
| Chromosome 1000000 to 10000000 | | | |
| 1998-1999 | 1000000 | 1000000 | 1000000 |
| 1999 | 1000000 | 1000000 | 1000000 |
| 2000 | 1000000 | 1000000 | 1000000 |
| Chromosome 10000000 to 100000000 | | | |
| 1998-1999 | 10000000 | 10000000 | 10000000 |
| 1999 | 10000000 | 10000000 | 10000000 |
| 2000 | 10000000 | 10000000 | 10000000 |
| Chromosome 100000000 to 1000000000 | | | |
| 1998-1999 | 100000000 | 100000000 | 100000000 |
| 1999 | 100000000 | 100000000 | 100000000 |
| 2000 | 100000000 | 100000000 | 100000000 |
| Chromosome 1000000000 to 10000000000 | | | |
| 1998-1999 | 1000000000 | 1000000000 | 1000000000 |
| 1999 | 1000000000 | 1000000000 | 1000000000 |
| 2000 | 1000000000 | 1000000000 | 1000000000 |
| Chromosome 10000000000 to 100000000000 | | | |
| 1998-1999 | 10000000000 | 10000000000 | 10000000000 |
| 1999 | 10000000000 | 10000000000 | 10000000000 |
| 2000 | 10000000000 | 10000000000 | 10000000000 |

Note: CBQ = Chromosome Banding Quality; CBQ + 50 = Chromosome Banding Quality + 50; Release = Chromosome Banding Quality + 50; Stock = Chromosome Banding Quality + 50; 1998-1999 = Chromosome Banding Quality + 50; 1999 = Chromosome Banding Quality + 50; 2000 = Chromosome Banding Quality + 50.