Journal of Consulting and Clinical Psychology
1995, Vol. 63, No. 5, 823-830

0022-006X/95/$3.00

Impact of Cognitive–Behavioral Treatment on Quality of Life in Panic Disorder Patients

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Panic disorder (PD) is associated with significant social and health consequences. The present study examined the impact of treatment on PD patients' quality of life. Patients (N = 156) meeting *DSM-III-R* (*Diagnostic and Statistical Manual of Mental Disorders* [3rd ed., rev.]; American Psychiatric Association, 1987) criteria for PD with agoraphobia were randomly assigned to group cognitive-behavioral treatment (CBT) or a delayed-treatment control. An assessment battery measuring the major clinical features of PD as well as quality of life was administered at baseline (Week 0), post-treatment (Week 9) and 6-month follow-up (Week 35). Consistent with previous studies, PD patients displayed significant impairment in quality of life at intake. Compared with delayed-treatment control participants, CBT-treated participants showed significant reductions in impairment that were maintained at follow-up. Consistent with prediction, anxiety and phobic avoidance were significantly associated with quality of life, whereas frequency of panic attacks was not.

Evidence from epidemiological data suggest that panic disorder is associated with significant social and health consequences including perceptions of poor physical and emotional health; alcohol and drug abuse; increased rate of suicide attempts; increased usage of psychoactive medications; and increased marital, social, and financial problems (Markowitz, Weissman, Ouellette, Lish, & Klerman, 1989; Weissman, 1991). Indeed, Markowitz et al. (1989) concluded that panic disorder confers negative social and health consequences of a magnitude equal to or greater than that for major depression.

On the basis of recent advances in the application of cognitive models to panic disorder, a new genre of psychological treatment has emerged that directly targets panic (Margraf, Barlow, Clark, & Telch, 1993). This cognitive-behavioral treatment (CBT) focuses on the patient's hypersensitivity to cues of physical arousal and the misinterpretation of these sensations as signaling immediate threat. Specific treatment components of CBT treatment for panic include (a) education about the nature and physiology of panic and anxiety, (b) cognitive restructuring of patients' faulty threat appraisals (e.g., "I will have a heart attack," "I will lose control"), (c) breathing exercises to control hyperventilation and help patients to cope with anxiety, and (d) graduated exposure to feared somatic cues, such as heart palpitations or dizziness. Evidence from several uncontrolled studies (Clark, Salkovskis, & Chalkley, 1985; Gitlin et al., 1985; Michelson, Marchione, Greenwald, Glantz, Marchione, & Testa, 1990) and seven controlled studies (Barlow, Craske, Cerny, & Klosko, 1989; Beck, Sokol, Clark, Berchick, & Wright, 1992; Clark et al., 1994; Craske, Brown, & Barlow, 1991; Klosko, Barlow, Tassinari, & Cerny, 1990; Margraf et al., 1993; Öst, Westling, & Hellstrom, 1993; Telch et al., 1993) suggest that CBT is effective in the treatment of panic disorder. In their excellent review of treatment outcome research on panic disorder, Michelson and Marchione concluded that "CBT studies evince positive findings. Therapeutic gains appear to be substantive, with elimination of panic attacks and anticipatory anxiety in the vast majority of clients" (1991, p. 105). Despite the encouraging efficacy data of CBT in the treatment of panic, Michelson and Marchione (1991) noted the paucity of data examining the effect of CBT on patients with panic disorder who displayed marked or extensive agoraphobia, as well as the failure to assess the clinical sequalae of CBT-treated patients.

Although the assessment of therapeutic outcome in the published literature has been of high quality, including multiple symptom indices (i.e., panic attacks, anticipatory anxiety, agoraphobic avoidance, depression) and composite measures of endstate functioning, the impact of CBT on panic disorder patients' quality of life (QOL) has yet to be evaluated. The present study examined the impact of CBT on QOL for panic disorder patients. We hypothesized that treatment would have a significant beneficial impact on QOL and that these gains would be maintained at follow-up. Because panic disorder patients typically display heightened levels of anxiety and avoidance associated with their panic attacks, we also examined the relative impact of panic frequency, anticipatory anxiety, and phobic avoidance on patients' QOL. We hypothesized that anxiety and agoraphobic avoidance would be more potent disrupters of patients' QOL than panic attack frequency, because of the persistency with which anxiety and phobic avoidance affect patients'

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Portions of this article were presented at the 27th Annual Convention of the Association for Advancement of Behavior Therapy in Atlanta, Georgia.

This research was supported by National Institute of Mental Health Grant MH74-600-203.

We thank Mike Gottfried for his assistance in data management.

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lives. Similarly, we hypothesized that changes in anxiety and phobic avoidance during treatment would have a greater impact on improving QOL, compared with changes in panic attack frequency.

Method

Participants

The sample consisted of 156 patients participating in clinical trials investigating the efficacy of a group-administered CBT (Telch et al., 1993). Entry criteria for these studies consisted of the following: (a) principal *DSM-III-R* (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed., rev.; American Psychiatric Association, 1987) Axis I diagnosis of panic disorder with agoraphobia, (b) at least one panic attack during the past 4 weeks, (c) no change in medication type or dose during the 8 weeks before treatment, (d) willingness to keep medication status stable during the 8-week treatment phase, (e) no evidence of serious suicide intent, (f) no evidence of current substance abuse, (g) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder, and (h) willingness to undergo randomization. Participant demographics are presented in Table 1.

Table 1
Demographic and Clinical Variables Describing the Patient Sample

Variable	Panic inoculation (n = 126)	Delayed treatment (n = 30)	Total (N = 156)
Age (years)			
M	34.6	36.8	34.8
SD	9.2	11.4	9.3
Sex (% female)	67.5	73.7	68.3
Ethnicity (%)			
White	81.2	88.8	82.2
Hispanic	10.3	0.0	8.9
Black	6.8	5.6	6.7
Asian	1.7	5.6	2.2
Marital status (%)			
Never married	26.1	27.8	26.3
Married	56.3	38.9	54.0
Divorced–separated	17.6	33.3	19.7
Education (%)			
Less than high school	17.3	27.8	18.7
High school	13.8	11.1	13.4
Part college	40.5	27.8	38.8
Graduated college or beyond	28.4	33.3	29.1
Employment status (%)			
Employed	63.9	50.0	62.0
Unemployed	29.4	44.4	31.4
Student	6.7	5.6	6.6
Panic severity (%)			
Mild	14.2	11.1	13.8
Moderate	55.0	55.6	55.1
Severe	30.8	33.3	31.2
Agoraphobia severity (%)			
Mild	42.4	27.7	40.6
Moderate	50.8	66.7	52.9
Severe	6.8	5.6	6.5
Chronicity (years)			
M	7.8	11.9	8.4
SD	8.9	11.2	9.3
Panic frequency (past month)			
M	2.3	2.1	2.3
SD	2.6	3.5	2.8
Anxiety (SPRAS)			
M	56.3	54.5	56.0
SD	26.5	25.5	26.2
Agoraphobia (FQ-Ago)			
M	13.6	11.2	13.0
SD	9.2	8.4	9.0
Secondary diagnoses (%)			
Major depression	24.3	28.1	25.4
Simple phobia	21.8	21.8	21.8
Social phobia	17.9	15.6	17.3
GAD	11.5	9.3	10.9
Dysthymia	7.7	6.2	7.3

Note. There were no baseline differences between treated and delayed-treatment patients. SPRAS = Sheehan Patient-Rated Anxiety Scale; FQ-Ago = Agorphobia scale of the Fear Questionnaire; GAD = generalized anxiety disorder.

Procedure

Patients were selected from a pool of 442 applicants who had undergone evaluation for panic disorder as part of a treatment outcome study sponsored by the National Institute of Mental Health (NIMH). 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-III-R*, Nonpatient Edition (SCID–NP; Spitzer, Williams, Gibbon, & First, 1990).

Twenty-eight videotaped interviews were selected at random and assessed by an independent rater for reliability. Kappa coefficients were very high for panic disorder diagnoses (κ = 1.00) and for all Axis I diagnoses (κ = .86).

Treatment

Participants were randomly assigned to either group administered CBT or a delayed-treatment control. Treatment consisted of 12 sessions over an 8-week period. The treatment protocol consisted of four major treatment components: (a) education and corrective information concerning the etiology and maintenance of panic disorder, (b) cognitive therapy, (c) training in diaphragmatic breathing techniques, and (d) interceptive exposure to feared bodily cues. A 65-page treatment manual (Telch & Schmidt, 1990) describes the specific goals and strategies for each session. Two licensed psychologists with at least 5 years of experience in CBT for anxiety disorders served as therapists. One therapist conducted a majority (80%) of the treatment groups.

Treatment Integrity

Treatment integrity was maintained by using a structured and manualized treatment protocol (Telch & Schmidt, 1990). Treatment integrity was rated by independent evaluators on a random sample of videotaped sessions. Compliance with the treatment manual was high (i.e., 177 of 180 exercises rated as completed, 177 of 177 exercises rated as consistent with manual description) across all sessions assessed (n = 36).

Assessment

An assessment battery tapping QOL issues as well as the major clinical dimensions of the disorder (i.e., panic attacks, anxiety, phobic avoidance, anxiety sensitivity) was administered at baseline (Week 0), posttreatment (Week 9) and 6-month follow-up (Week 35). Participants in the delayed-treatment control received treatment after the posttreatment assessment and were not assessed further.

Assessment Battery

The assessment battery consisted of two QOL measures: the Social Adjustment Scale—Self-Report (SAS) and the Sheehan Disability Scale

(SDS). Three facets of panic disorder (i.e., panic attacks, anxiety, and panic-related avoidance) were assessed with the following measures: (a) the Texas Panic Attack Record Form, (b) the Sheehan Patient-Rated Anxiety Scale (SPRAS), and (c) the Agoraphobia scale of the Marks and Mathews Fear Questionnaire (FQ–Ago).

SAS. The SAS (Weissman, Prusoff, Thompson, Harding, & Myers, 1978) is a 54-item self-report instrument that has been used extensively in epidemiological studies to index QOL. The SAS consists of nine role areas: (a) work outside the home, (b) work at home, (c) work as a student, (d) social and leisure, (e) extended family, (f) marital, (g) parental, (h) family unit, and (i) economic. Scored items within each role area are averaged. Although Weissman et al. (1978) use only work outside home in computing the overall adjustment score, we present data from each of the three work area scales (i.e., work outside home, work at home, and work as a student) in an attempt to capture differential effects in the separate work areas. For example, phobic avoidance is likely to affect work outside the home to a greater extent than work inside the home. An overall adjustment score is computed from the average of all scored items. The SAS has demonstrated adequate psychometric properties and has been shown to be sensitive to treatment effects (Weissman, Paykel, & Prusoff, 1990; Weissman, Paykel, Siegel, & Klerman, 1971).

SDS. The SDS is a four-item self-report measure of global impairment created by the presenting problem. The SDS was used in the Upjohn Cross-National Collaborative Panic Study (Ballenger et al., 1988). Three items assess impairment: (a) work activities, (b) social life and leisure activities, and (c) family life and home responsibilities. Each item is rated on an 11-point Likert scale (0 = not at all, 1–3 = mild, 4–6 = moderate, 7–9 = marked, 10 = severe). One item assesses overall (i.e., global) work and social disability and is scored on a 5-point scale.

Texas Panic Attack Record Form. Panic attacks were assessed using a prospective self-monitoring approach similar to that used in the Ballenger et al. (1988) study that has been found to reduce overreporting bias (Margraf et al., 1987). Participants were provided with daily panic diary forms. For each panic episode, participants were instructed to record the (a) date, (b) time, (c) duration, (d) severity, (e) symptoms experienced, and (f) setting parameters (e.g., place, activity, presence of others). Panic attacks with three or fewer symptoms (i.e., limited symptom attacks) were not included in the panic attack count. The importance of immediate recording was emphasized to increase the accuracy of participants' recollection of the panic attack.

SPRAS. The SPRAS (Sheehan, 1983) is a 35-item self-report scale for assessing the intensity of anxiety symptoms. Each of the 35 symptoms (e.g., shaking or trembling) is rated on a 5-point scale ranging from 0 (*not at all distressing*) to 4 (*extremely distressing*). The instructions were modified so that symptom ratings were based on a 1-week time frame.

FQ–Ago. The FQ (Marks & Mathews, 1979) was used to assess level of phobic avoidance. The FQ consists of 15 items representing three separate phobia types (agoraphobia, blood–injury phobia, and social phobia). For each item, the participant rates the degree of avoidance to the object or situation. The five-item agoraphobia subscale (FQ–Ago) has demonstrated adequate psychometric properties and is the most widely used self-report measure for assessing agoraphobia in treatment outcome research (Jacobson, Wilson, & Tupper, 1988).

Results

Analytic Overview

We examined baseline differences between CBT-treated patients and delayed-treatment control participants on demographic, QOL, and clinical variables using independent *t* tests for continuous variables and chi-square tests for categorical variables. Between-group differences on posttreatment QOL were examined using analyses of covariances (ANCOVAs), with treatment group (CBT vs. Delayed-treatment) as the grouping factor and the baseline score on each domain serving as the covariate. Multivariate analyses of variance (MANOVAs) were not conducted because of incomplete data on various SAS subscales (e.g., only married patients completed the Marital Functioning subscale). Repeated measures ANOVAs were conducted to assess within-group changes in QOL from pretreatment to posttreatment and from posttreatment to follow-up. Bonferroni correction was used to control for family-wise error at each assessment period. The adjusted level of significance was .004.

Differences at Baseline

Participants in the two groups did not differ significantly on any of the demographic or clinical variables at baseline (see Table 1). Means and standard deviations of the QOL measures at each assessment period are presented in Tables 2 and 3. The two groups did not significantly differ on the SAS or SDS at baseline ($ps > .05$).

Examination of overall SDS scores at baseline revealed that 69% of all participants rated the panic disorder symptoms as creating moderate-to-severe impairment in work functioning, 80% rated the symptoms as creating moderate-to-severe impairment in social functioning, and 62% rated the symptoms as creating moderate-to-severe impairment in family and home responsibilities. Global impairment ratings indicated that 26% of the participants reported that the panic disorder symptoms prevented or radically changed their normal activities. An additional 36% of the participants rated their overall impairment as marked. Only 12% of the participants rated their symptoms as not interfering with their activities. These disability scores are comparable with those reported in the Upjohn Cross-National Collaborative Panic Study (Ballenger et al., 1988).

Effect of Treatment on QOL

CBT-treated participants displayed significantly less impairment on most QOL indices, compared with the posttreatment levels of the untreated participants (see Tables 2 and 3). Treated participants showed less impairment on the SAS scales measuring working outside the home, $F(1, 89) = 4.03, p < .05$; working inside the home, $F(1, 38) = 4.12, p < .05$; social and leisure activities, $F(1, 123) = 6.08, p < .05$; extended family relationships, $F(1, 121) = 4.27, p < .05$; marital relationships, $F(1, 82) = 4.38, p < .05$; and overall functioning, $F(1, 124) = 7.23, p < .05$. Treated participants showed less disability on the SDS subscales measuring family functioning, $F(1, 125) = 9.54, p < .05$; social functioning, $F(1, 132) = 21.84, p < .05$; work functioning, $F(1, 132) = 23.12, p < .05$; and global functioning, $F(1, 132) = 20.33, p < .05$.

Compared with baseline levels, participants receiving CBT displayed improved scores on the three SDS individual scales—family, $F(1, 111) = 104.33, p < .004$; social, $F(1, 111) = 131.84, p < .004$; work, $F(1, 110) = 86.91, p < .004$—as well as the SDS global disability index, $F(1, 111) = 134.73, p < .004$. On the SAS, CBT-treated participants showed significant improvement on the overall SAS score, $F(1, 111) = 32.45, p < .004$, in addition to improvement in the following areas: work outside the home, $F(1, 75) =$

Table 2
Means and Standard Deviations for the Social Adjustment Scale at Each Assessment Period

Measure	Treatment			Delayed-treatment	
	Pre (n = 126)	Post (n = 112)	FU (n = 91)	Pre (n = 30)	Post (n = 28)
Work outside home					
M	1.9_a	1.6_b	1.5_b	2.0	1.9[a]
SD	0.5	0.6	0.4	0.4	0.4
Work inside home					
M	2.3_a	1.5_b	1.9_b	1.9	1.9[a]
SD	0.7	0.4	0.7	0.5	0.6
Work as student					
M	1.9	1.4	1.9	1.8	1.8
SD	0.5	0.4	1.2	0.6	0.6
Social–leisure					
M	2.4_a	2.0_b	1.8_b	2.4	2.3[a]
SD	0.5	0.5	0.5	0.6	0.5
Extended family					
M	1.9_a	1.6_b	1.6_b	1.9	1.8[a]
SD	0.5	0.5	0.5	0.5	0.5
Marital					
M	2.1_a	1.7_b	1.9_b	2.1	2.1[a]
SD	0.6	0.6	0.6	0.5	0.5
Parental					
M	1.7	1.5	1.6	1.8	1.8
SD	0.6	0.6	0.6	0.8	0.9
Family unit					
M	2.2_a	1.8	1.8_b	2.3	2.0
SD	0.8	0.8	0.7	0.8	0.7
Economic					
M	1.8	1.7	1.7	2.2	2.1
SD	1.1	1.1	1.1	1.2	1.2
Overall					
M	2.1_a	1.7_b	1.7_b	2.1	2.0[a]
SD	0.4	0.4	0.5	0.4	0.6

Note. Means with different subscripts indicate significant within-group differences ($p < .004$). Pre = preassessment; post = postassessment; FU = follow-up.
[a] Significant treatment versus delayed-treatment group difference at posttreatment ($p < .05$).

11.32, $p < .004$; work inside the home, $F(1, 32) = 13.30$, $p < .004$; social and leisure activities, $F(1, 111) = 75.30$, $p < .004$; extended family relationships, $F(1, 111) = 40.17$, $p < .004$; and marital relationships, $F(1, 73) = 24.13$, $p < .004$.

Findings at the 6-month follow-up are similar to those seen at the posttreatment assessment. Treated participants made significant improvements on the majority of QOL indices. Compared with baseline levels, CBT-treated participants displayed improvement on the three SDS individual scales—family, $F(1, 90) = 80.63$, $p < .004$; social, $F(1, 90) = 153.00$, $p < .004$; and work, $F(1, 89) = 86.10$, $p < .004$—as well as on the SDS Global Disability Index, $F(1, 90) = 147.00$, $p < .004$. Participants reported significant improvement on six of nine SAS role areas: work outside the home, $F(1, 47) = 10.74$, $p < .004$; work inside the home, $F(1, 17) = 13.31$, $p < .004$; social and leisure activities, $F(1, 90) = 76.59$, $p < .004$; family functioning, $F(1, 67) = 23.66$, $p < .004$; marital functioning, $F(1, 46) = 9.98$, $p < .004$; and functioning in the extended family, $F(1, 59) = 20.42$, $p < .004$; as well as the overall SAS score, $F(1, 90) = 24.23$, $p < .004$. Similar to the posttreatment assessment, participants' level of improvement failed to reach statistical significance in the areas of work as a student, parental functioning, and economic functioning (see Table 2).

To assess whether gains at posttreatment were maintained, we examined posttreatment to follow-up scores on each QOL measure. As can be seen in Tables 2 and 3, QOL improvements were maintained from posttreatment to follow-up. There were no significant differences in post-follow-up scores on the SDS or SAS overall scales or any of the SDS or SAS subscales ($ps > .004$).

Relationship Between the Clinical Features of Panic Disorder and QOL at Baseline

We investigated the relationship between each of the major clinical features of panic disorder (i.e., panic attacks, anxiety, and panic-related avoidance) and QOL at baseline (see Table 4). The panic attack frequency data was highly skewed and was normalized using logarithmic transformation. As would be expected, the three symptom variables were moderately associated with each other. Anxiety and avoidance were moderately correlated (pr [partial correlation] = .27). Panic attack frequency was weakly associated with phobic avoidance (pr = .09) and more strongly associated with anxiety (pr = .31). The level of association among these symptoms is consistent with the idea

Table 3
Means and Standard Deviations for the Sheehan Disability Scale at Each Assessment Period

	Treatment			Delayed treatment	
Measure	Pre (n = 126)	Post (n = 112)	FU (n = 91)	Pre (n = 30)	Post (n = 28)
Family					
M	3.7_a	1.2_b	1.5_b	3.5	2.5[a]
SD	2.3	1.8	2.0	2.5	2.4
Social					
M	5.5_a	2.2_b	2.0_b	4.9	4.2[a]
SD	2.9	2.3	2.2	2.6	2.5
Work					
M	4.4_a	1.7_b	1.7_b	4.5	4.1[a]
SD	2.8	2.2	2.3	3.3	2.9
Global					
M	3.7_a	2.3_b	2.3_b	4.0	3.5[a]
SD	1.0	1.0	1.0	0.9	1.0

Note. Means with different subscripts indicate significant within-group differences ($p < .004$). Pre = preassessment; post = postassessment; FU = follow-up.
[a] Significant treatment versus delayed-treatment group difference at posttreatment ($p < .05$).

of separate but related clinical features that constitute an overarching syndrome.

Consistent with prediction, anxiety was significantly associated with both pretreatment QOL measures, whereas phobic avoidance was significantly associated with pretreatment SDS scores. In contrast, panic attack frequency showed no significant relationship to either of the QOL measures. Partial correlations simultaneously controlling for the influence of each clinical variable revealed that anxiety as measured by the SPRAS was the only significant predictor of SAS (pr =.43, $p < .05$). Both the SPRAS (pr = .23, $p < .05$) and the FQ–Ago (pr = .29, $p < .05$) were significant predictors of SDS.

The relationship between QOL and pretreatment levels of panic, agoraphobia, and anxiety is shown in Figure 1. The severity of panic attacks and agoraphobia severity were based on DSM-III-R criteria as determined by the SCID-NP. Anxiety severity was derived from a tertian split of the SPRAS. As can be seen in Figure 1, panic severity shows no relation to level of impairment on the SDS work, social, and family indices. However, increasing severity of phobic avoidance and anxiety are each associated with increasing levels of impairment in all SDS scales.

Does the Severity of Panic Disorder Symptoms at Baseline Predict Changes in QOL?

Because baseline severity of anxiety and agoraphobic avoidance was related to pretreatment QOL, we examined whether the severity of panic-related symptoms at baseline was predictive of changes in QOL at posttreatment and follow-up. Multiple regression analyses were used to examine the relationship between baseline severity of panic disorder and changes in QOL. Each respective pretreatment QOL measure (i.e., overall SAS, global SDS) was forced into the model and served as its own covariate. Next, the baseline scores for the three clinical variables were simultaneously entered. These analyses revealed no relationship between intake severity of panic disorder symptoms and changes in QOL at posttreatment or follow-up ($ps > .05$).

Do Changes in the Clinical Features During Treatment Predict Changes in QOL at Posttreatment and Follow-Up?

We performed stepwise regression analyses to assess whether changes in panic disorder symptoms predicted changes in QOL

Table 4
Correlations Between Overall SAS, Global SDS, and the Major Clinical Features of Panic Disorder

	Overall SAS			Global SDS		
Feature	Pre (n = 156)	Post[a] (n = 130)	FU[a] (n = 85)	Pre (n = 156)	Post[a] (n = 117)	FU[a] (n = 84)
Panic frequency						
r	−.04	.15	.14	.08	.34*	.24*
Partial *r*	−.20*	−.09	.11	−.07	.13	.17
Anxiety (SPRAS)						
r	.40*	.47*	.36*	.30*	.59*	.42*
Partial *r*	.43*	.36*	.18	.23*	.49*	.35*
Phobic Avoidance (FQ)						
r	.16	.34*	.26*	.36*	.44*	.43*
Partial *r*	.08	.11	−.01	.29*	.17	.22*

Note. Panic frequency underwent log transformation. Partial *r*s at baseline control for each clinical feature. Partial *r*s at postassessment and follow-up control for baseline quality of life scores as well as each clinical feature. SAS = Social Adjustment Scale—Self-Report; SDS = Sheehan Disability Scale; Pre = preassessment; Post = postassessment; FU = follow-up; SPRAS = Sheehan Patient-Rated Anxiety Scale; FQ = Fear Questionnaire.
[a] Residualized change scores were computed at postassessment and follow-up for each clinical variable. Positive correlations indicate the relationship between degree of improvement in each domain and positive changes in quality of life.
* $p < .05$.

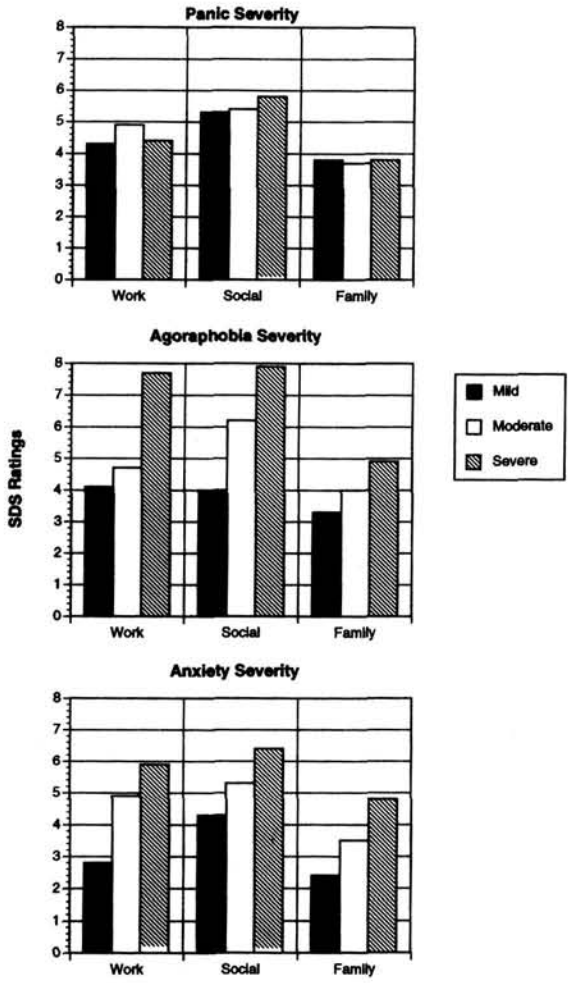

Figure 1. The relationship between pretreatment severity of panic, agoraphobia, and anxiety and Sheehan Disability Scale (SDS) ratings of impairment in work, social, and family life.

at posttreatment. Pretreatment QOL levels (i.e., overall SAS, global SDS) were entered first and locked into the model. Pre- to posttreatment residualized change scores were computed for each of the three symptom measures. Residualized changes in anxiety entered first in predicting both the SAS, $R^2 = .43$, $F(1, 127) = 27.38$, $p < .0001$; and the SDS, $R^2 = .50$, $F(1, 121) = 35.76$, $p < .0001$. There were no other significant predictors of change in QOL at posttreatment.

A similar stepwise regression procedure was used for predicting QOL at follow-up. Pretreatment QOL levels (i.e., overall SAS, global SDS) were entered first and locked into the model. Pre- to posttreatment residualized change scores for each of the three clinical features were then allowed to enter the model. Similar to findings at posttreatment, anxiety was first to enter equations predicting both the SAS, $R^2 = .40$, $F(1, 83) = 23.61$, $p < .0001$; and the SDS, $R^2 = .42$, $F(1, 82) = 26.36$, $p < .0001$. No other variables predicted follow-up QOL. Once again, changes in anxiety during treatment was the most potent predictor of changes in QOL.

Discussion

The present study examined the effect of CBT on QOL in panic disorder patients. Consistent with other reports, our findings suggest that panic disorder patients show QOL impairment across a variety of domains (Maisson, Warshaw, & Keller, 1993; Markowitz et al., 1989; Weissman, 1991). Patients showed relatively greater QOL impairment in the areas of work inside the home, social and leisure activities, and functioning as a family unit. The impairment ratings for work inside the home and family functioning exemplify the pervasive debilitation associated with panic disorder. Not only does panic disorder affect patients when they leave the home, its effects appear to pervade what is typically considered to be their safest haven, the home.

The use of standardized indices such as the SAS and SDS allows for QOL comparisons across different samples and different populations. QOL impairment for our sample is comparable with the level of disability reported for panic disorder patients in the Upjohn Cross-National Collaborative Panic Disorder Study (Ballenger et al., 1988). In addition, baseline QOL scores for our panic disorder sample are similar to or greater than scores reported for patients with alcoholism, schizophrenia, and personality disorders, but somewhat less than those reported for patients with acute depression (Weissman et al., 1978; Winston et al., 1991). Impairment in our panic disorder sample is also greater than that reported for medical populations such as patients undergoing a mastectomy (Jones & Reznikoff, 1989) or patients completing elective sterilization (Cooper, Gath, Rose, & Fieldsend, 1982). In summary, QOL impairment in panic disorder patients appears to be comparable with or greater than the disability reported in other psychiatric and medical populations.

Significant improvement in QOL indices were observed for patients receiving CBT but not for delayed-treatment control participants. These findings increase our confidence that the observed improvements were a result of CBT and not extraneous factors such as the passage of time. Preliminary data from the 6-month follow-up assessment suggest that the improvements in QOL evidenced by CBT-treated patients are maintained after treatment termination. These findings are consistent with data from our center and others showing relatively low rates of relapse among patients with panic disorder treated with CBT (Margraf et al., 1993).

Were the changes in QOL clinically meaningful? One commonly used strategy for assessing the clinical significance of treatment gains is to examine the extent to which treated patients' scores move into the range of control participants' scores. At posttreatment and 6-month follow-up, the overall SAS QOL index for our treated patients ($M = 1.7$, $SD = 0.4$) closely approximated those reported by Weissman et al. for a community sample of control participants ($M = 1.6$, $SD = 0.3$). Normative comparisons based on meta-analytic procedures can be expressed as effect sizes (Kendall & Grove, 1988). The SAS comparisons between our sample and the community control participants at posttreatment and follow-up are described by relatively small effect sizes (i.e., .31 at posttreatment and .30 at follow-up). indicating that a patient at the mean of the treated group would fall at approximately the 62nd percentile of the community sample. In comparison, normative comparisons at

posttreatment with the waiting-list controls yield an effect size of 1.25, indicating that a patient at the mean of the untreated group would fall at approximately the 89th percentile of the community sample. These SAS score comparisons across samples suggest that the changes in QOL observed for our CBT-treated panic disorder sample are not only statistically significant but are also clinically meaningful.

Our results help shed light on which symptom features of panic disorder affect patients' QOL. As predicted, panic frequency at intake was not associated with QOL indices. Similarly, pre- to posttreatment changes in panic frequency did not predict QOL indices at posttreatment or at follow-up when controlling for panic-related anxiety and avoidance. In contrast, anxiety and phobic avoidance predicted QOL at baseline. Moreover, pre- to posttreatment change in anxiety was the most potent predictor of QOL at posttreatment and follow-up. These findings suggest that panic attacks have a less direct impact on QOL than do anxiety and avoidance. Perhaps the infrequency and transient nature of panic leads to less impairment than the more chronic and pervasive symptoms of anxiety and agoraphobic avoidance.

These data do not suggest that panic attacks are unimportant in the psychopathogenicity of panic disorder. On the contrary, evidence from diverse sources points to the centrality of panic attacks in mediating patients' chronic anticipatory anxiety and phobic avoidance. However, our findings do suggest that once panic disorder is present, the anxiety and avoidance symptoms of the disorder produce the greatest negative impact on patients' QOL.

The treatment implications of our findings deserve comment. First, our data point to the importance of evaluating treatment efficacy across multiple domains rather than relying solely on panic frequency or the commonly reported index of the percentage of panicfree patients. free. Second, our results suggest that treatments that correct anticipatory anxiety and phobic avoidance are likely to yield the greatest benefit to panic patients. Although our findings provide encouraging evidence that CBT results in clinically significant improvement in patients' QOL, it would be premature to conclude that CBT is uniquely effective in this regard. The efficacy of alternative treatments (e.g., pharmacotherapy and combined pharmacological and psychological treatments) in enhancing patients' QOL have yet to be reported. Our findings suggest that alternative treatments should lead to enhanced QOL to the extent that they produce meaningful improvements in patients' anxiety and phobic avoidance.

Despite the overall gains made in QOL, our sample failed to improve in several role areas assessed by the SAS. A floor effect is the most likely explanation because patient scores in those role areas were close to or in the normal range at baseline. Also, the present QOL battery did not assess several other relevant QOL domains such as health care use, alcohol and substance abuse, or suicide attempts. Studies that evaluate multiple treatments across a diverse range of QOL indicators will further the understanding of the impact of treatment on the QOL of patients with panic disorder.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

Ballenger, J. C., Burrows, G. D., DuPont, R. L., Jr., Lesser, I. M., Noyes, R., Jr., Pecknold, J. C., Rifkin, A., & Swinson, R. P. (1988). Alprazolam in panic disorder and agoraphobia: Results from a multicenter trial. *Archives of General Psychiatry, 45,* 413–422.

Barlow, D. H. (1988). *Anxiety and its disorders: The nature and treatment of anxiety and panic.* New York: Guilford Press.

Barlow, D. H., Craske, M. G., Cerny, J. A., & Klosko, J. S. (1989). Behavioral treatment of panic disorder. *Behavior Therapy, 20,* 261–282.

Beck, A. T., Sokol, L., Clark, D. A., Berchick, R., & Wright, F. (1992). A crossover study of focused cognitive therapy for panic disorder. *American Journal of Psychiatry, 149,* 778–83.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24,* 461–470.

Clark, D. M., Salkovskis, P. M., & Chalkley, A. J. (1985). Respiratory control as a treatment for panic attacks. *Journal of Behavior Therapy and Experimental Psychiatry, 16,* 23–30.

Clark, D. M., Salkovskis, P. M., Hackman, A., Middleton, H., Anastasiades, P., & Gelder, M. (1994). A comparison of cognitive therapy, applied relaxation and imipramine in the treatment of panic disorder. *British Journal of Psychiatry, 164,* 759–769.

Cooper, P., Gath, D., Rose, N., & Fieldsend, R. (1982). Psychological sequelae to elective sterilization: A prospective study. *British Medical Journal, 284,* 461–464.

Craske, M. G., Brown, T. A., & Barlow, D. H. (1991). Behavioral treatment of panic disorder: A two-year follow-up. *Behavior Therapy, 22,* 289–304.

Gitlin, B., Martin, J., Shear, M. K., Frances, A., Ball, G., & Josephson, S. (1985). Behavior therapy for panic disorder. *Journal of Nervous and Mental Diseases, 173,* 742–743.

Jacobson, N. S., Wilson, L., & Tupper, C. (1988). The clinical significance of treatment gains resulting from exposure-based interventions for agoraphobia: A reanalysis of outcome data. *Behavior Therapy, 19(4),* 539–554.

Jones, D. N., & Reznikoff, M. (1989). Psychosocial adjustment to a mastectomy. *Journal of Nervous and Mental Disease, 177,* 624–631.

Kendall, P. C., & Grove, W. M. (1988). Normative comparisons in therapy outcome. *Behavioral Assessment, 10,* 147–158.

Klosko, J. S., Barlow, D. H., Tassinari, R., & Cerny, J. A. (1990). A comparison of alprazolam and behavior therapy in treatment of panic disorder. *Journal of Consulting and Clinical Psychology, 58,* 77–84.

Maisson, A. O., Warshaw, M., G., & Keller, M. B. (1993). Quality of life and psychiatric morbidity in panic disorder and generalized anxiety disorder. *American Journal of Psychiatry, 150,* 600–607.

Margraf, J., Barlow, D. H., Clark, D. M., & Telch, M. J. (1993). Psychological treatment of panic: Work in progress on outcome, active ingredients, and follow-up. *Behaviour Research and Therapy, 31,* 1–8.

Margraf, J., Taylor, C. B., Ehlers, A., Roth, W. T., & Agras, W. S. (1987). Panic attacks in the natural environment. Special issue: Mental disorders in their natural settings: The application of time allocation and experience-sampling techniques in psychiatry. *Journal of Nervous and Mental Disease, 175,* 558–565.

Markowitz, J. S., Weissman, M. M., Ouellette, R., Lish, J. D., & Klerman, G. L. (1989). Quality of life in panic disorder. *Archives of General Psychiatry, 46,* 984–92.

Marks, I. M., & Mathews, A. M. (1979). Brief standard self-rating for phobic patients. *Behaviour Research and Therapy, 17,* 263–267.

Michelson, L. K., & Marchione, K. (1991). Behavioral, cognitive, and pharmacological treatments of panic disorder with agoraphobia: Critique and synthesis. *Journal of Consulting and Clinical Psychology, 59,* 100–114.

Michelson, L. K., Marchione, K., Greenwald, M., Giantz, L., Marchione, N., & Testa, S. (1990). Panic disorder: Cognitive-behavioral treatment. *Behavior Research and Therapy, 28,* 141–151.

Öst, L. G., Westling, B. E., & Hellstrom, K. (1993). Applied relaxation, exposure in vivo and cognitive methods in the treatment of panic disorder with agoraphobia. *Behaviour Research and Therapy, 31,* 383–94.

Sheehan, D. V. (1983). *The anxiety disease.* New York: Scribners.

Spitzer, R. L., Williams, J. B., Gibbon, M., & First, M. B. (1990). *Structured Clinical Interview for DSM–III–R, nonpatient edition* (SCID–NP, Version 1. 0). Washington, DC: American Psychiatric Press.

Telch, M. J., Lucas, J. A., Schmidt, N. B., Hanna, H. H., Jaimez, T. L., & Lucas, R. A. (1993). Group cognitive-behavioral treatment of panic disorder. *Behaviour Research and Therapy, 31,* 279–287.

Telch, M. J., & Schmidt, N. B. (1990). *Cognitive-behavioral treatment for panic disorder and agoraphobia: Panic inoculation treatment manual.* Unpublished manuscript.

Weissman, M. M. (1991). Panic disorder: Impact on quality of life. *Journal of Clinical Psychiatry, 52,* 6–9.

Weissman, M. M., Paykel, E. S., & Prusoff, B. A. (1990). *Social adjustment scale handbook: Rationale, reliability, validity, scoring, and training guide.* Unpublished handbook.

Weissman, M. M., Paykel, E. S., Siegel, R., & Klerman, G. L. (1971). The social role performance of depressed women: Comparisons with a normal group. *American Journal of Orthopsychiatry, 41,* 390–405.

Weissman, M. M., Prusoff, B. A., Thompson, W. D., Harding, P. S., & Myers, J. K. (1978). Social adjustment by self-report in a community sample and in psychiatric outpatients. *Journal of Nervous and Mental Disease, 166,* 317–326.

Winston, A., Pollack, J., McCullough, L., Flegenheimer, W., Kestenbaum, R., & Trujillo, M. (1991). Brief psychotherapy of personality disorders. *Journal of Nervous and Mental Disease, 179,* 188–193.

Received February 24, 1994
Revision received October 24, 1994
Accepted December 19, 1994 ■

Correction to Klosko et al. (1990)

In the article "A Comparison of Alprazolam and Behavior Therapy in Treatment of Panic Disorder," by Janet S. Klosko, David H. Barlow, Robin Tassinari, and Jerome A. Cerny (*Journal of Consulting and Clinical Psychology,* 1990, Vol. 58, No. 1, 77–84), there was an error in the chi-square for the number of patients experiencing zero panic attacks among the four groups evaluated (alprazolam, placebo, panic-control treatment, and waiting list). On pages 82–83, the statistic is given as $\chi^2(3, N = 57) = 5.21, p < .05$. The correct statistic is $\chi^2(3, N = 57) = 10.42, p < .02$. Because follow-up analyses (2×2 tables) of this effect would have involved chi-square tests with expected cell frequencies of ≤ 5, Fisher's exact tests and z tests for proportions were conducted instead. Additional analyses now show that the panic-control treatment group was significantly different from the alprazolam group, as well as from the placebo and waiting-list groups as originally reported (Fisher's exact test $p < .04$; z test for proportions = 2.18, $p < .05$). The 95% confidence limits of this difference range from 3.7% to 69.6%. The authors thank Don Klein for suggesting the analysis that led to this discovery.