A Comparison of the Efficacy of Clonazepam and Cognitive-Behavioral Group Therapy for the Treatment of Social Phobia

Michael W. Otto, Ph.D., Mark H. Pollack, M.D., Robert A. Gould, Ph.D., John J. Worthington III, M.D., Eliza T. McArdle, B.A., and Jerrold F. Rosenbaum, M.D.

Massachusetts General Hospital and Harvard Medical School, Boston, Massachusetts, USA

Richard G. Heimberg, Ph.D.

Department of Psychology, Temple University, Philadelphia, Pennsylvania, USA

Abstract—There is a growing body of evidence that social phobia may be treated effectively by either pharmacologic or cognitive-behavioral interventions, but few studies have examined the relative benefits of these treatments. In this study, we examined the relative efficacy of pharmacotherapy with clonazepam and cognitive-behavioral group therapy (CBGT) for treating social phobia. In addition, we examined potential predictors of differential treatment response. Outpatients meeting Diagnostic and Statistical Manual of Mental Disorders (3rd ed., revised) criteria for social phobia were randomly assigned to treatment. Clinician-rated and patient-rated symptom severity was examined at baseline and after 4, 8, and 12 weeks of treatment. All clinician-rated assessments were completed by individuals blind to treatment condition. Patients in both conditions improved significantly, and differences between treatment conditions were absent, except for greater improvement on clonazepam on several measures at the 12-week assessment. Symptom severity was negatively associated with treatment success for both methods of treatment, and additional predictors—sex, comorbidity with other...
anxiety or mood disorders, fear of anxiety symptoms, and dysfunctional attitudes—failed to predict treatment outcome above and beyond severity measures. In summary, we found that patients randomized to clinical care with clonazepam or CBGT were equally likely to respond to acute treatment, and pretreatment measures of symptom severity provided no guidance for the selection of one treatment over another. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Social phobia; Cognitive-behavioral therapy; Clonazepam; Social anxiety disorder; Benzodiazepine

The lifetime prevalence of social phobia is approximately 13%, making it the third most common psychiatric disorder in the community after major depression and alcohol dependence (Kessler et al., 1994). It is characterized by a persistent fear of one or more social or performance situations that invoke intense anxiety, distress, or avoidance, and which result in significant impairment in social or role functioning (American Psychiatric Association, 1994). The impairment resulting from social phobia may be extensive and includes disruption in educational and occupational achievement as well as family and social relationships (Davidson, Hughes, George, & Blazer, 1993; Schneier et al., 1994). Comorbid anxiety, mood, and substance use disorders are also common among individuals with social phobia (Davidson et al., 1993; Schneier, Hohnson, Hornig, Liebowitz, & Weisman, 1992).

Two modes of treatment, pharmacotherapy and cognitive-behavior therapy (CBT), have demonstrated efficacy in the treatment of social phobia (Gould, Buckminster, Pollack, Otto, & Yap, 1997). In controlled clinical trials, monoamine oxidase inhibitors (i.e., phenelzine; Liebowitz et al., 1992; Versiani et al., 1992) and high potency benzodiazepines (Davidson et al., 1993; Munjack, Baltazar, Bohn, Cabe, & Appleton, 1990) have demonstrated efficacy for social phobia. Initial support also has been provided for selective serotonin reuptake inhibitors (SSRIs), including fluvoxamine (van Vliet, den Boer, & Westenberg, 1994), sertraline (Katzelnick, Kobak, & Greist, 1995), and paroxetine (Stein et al., 1998). Beta-blockers, although effective for performance anxiety, appear less effective for social phobia (Pollack & Gould, 1996).

Cognitive-behavioral treatments for social phobia have typically emphasized cognitive-restructuring and exposure interventions, either alone or in combination, in either individual or group formats (Heimberg & Juster, 1995; Juster & Heimberg, 1995). Treatment programs emphasizing these interventions have met with consistent success (for metaanalytic reviews see Feske & Chamber, 1995; Gould et al., 1997; Taylor, 1996). For example, in a comparison of cognitive-behavioral group treatment (CBGT) with an educational-supportive psychotherapy program, significantly greater improvement was found for the CBGT program at both endpoint and 5-year follow-up assessments (Heimberg et al., 1990; Heimberg et al., 1993).

As specific pharmacologic and cognitive-behavioral treatments for social phobia are established, questions arise about the relative efficacy of these treat-
ments. In one of the few studies in this area, Gelernter et al., (1991) compared a CBGT with pharmacotherapy (with alprazolam, phenelzine, or pill placebo) plus instructions for exposure to phobic stimuli. All three treatments were associated with significant improvement, and no single treatment was consistently superior to the others. More recently, Heimberg, Juster, and Brown (1994) compared CBGT with phenelzine, pill-placebo, and attention-placebo conditions. At posttreatment (12 weeks), CBGT and phenelzine produced response rates that were equivalent to each other and superior to both the pill-placebo and the attention-placebo control group. Whereas phenelzine had a faster onset of therapeutic effect and demonstrated superior outcome on select measures at endpoint, CBGT tended to be associated with reduced risk of relapse during maintenance treatment and follow-up.

Findings of approximately equal efficacy of pharmacologic and cognitive-behavioral treatments for social phobia are reflected in the results of a recent metaanalysis (Gould et al., 1997). Sixteen studies with 27 treatment conditions achieved a mean effect size of 0.74 for CBT relative to control conditions (most frequently a wait-list control), and 11 studies of the efficacy of pharmacotherapy relative to pill placebo provided a mean effect size of 0.62. In this metaanalysis, the very highest, single-study effect size was obtained for the high potency benzodiazepine clonazepam. In addition, the overall average effect size for clonazepam was among the highest ($ES = .72$) for pharmacologic treatment and equal to the overall effect size for CBT ($ES = .74$).

The present study was designed to test the relative efficacy of two accepted treatments of social phobia: pharmacotherapy with clonazepam treatment and Heimberg’s CBGT (Heimberg, Juster, & Hope, 1995; Heimberg et al., 1993). Each treatment has demonstrated impressive effect sizes in earlier trials; the purpose of the present study was to examine the relative efficacy of these treatments and potential predictors of differential outcome.

**METHOD**

**Participants**

Participants in this study were 27 men and 18 women who met Diagnostic and Statistical Manual of Mental Disorders (3rd ed., revised [DSM-III-R]) criteria (American Psychiatric Association, 1987) for social phobia as a primary diagnosis. Mean age (± standard deviation) of participants was 39.8 ± 10.5 years; mean age of onset of the social phobia was 14.6 ± 7.9 years. Seventy-six percent of the sample met criteria for the generalized subtype of social phobia.

Participants were recruited from clinical referrals and local media advertisements, and all provided written informed consent. Exclusion criteria were the following: (1) a history of failure to respond to or hypersensitivity to 2 mg of clonazepam treatment, or known allergy to this or other benzodiazepines;
(2) a history of previous CBT for social phobia that included exposure and cognitive restructuring interventions; (3) the presence of a serious or unstable medical condition; (4) current pregnancy or lactation, or failure to take adequate precautions (medically acceptable means of birth control) against pregnancy; (5) a history of schizophrenia, other psychosis, or bipolar disorder; (6) a current diagnosis of alcoholism or other substance abuse or dependence; and (7) recent or current suicidal tendency. Patients were not taking psychotropic medications at baseline, and were not engaged in concurrent psychotherapy for anxiety difficulties, although patients undergoing other treatments (e.g., occasional couples therapy) were eligible as long as the focus of this treatment was not anxiety management.

Comorbid anxiety or affective disorders were permitted as long as social phobia was the primary diagnosis. Two patients met criteria for comorbid panic disorder (both received clonazepam), eight met criteria for comorbid generalized anxiety disorder (three received CBGT and five received clonazepam), and one met criteria for comorbid posttraumatic stress disorder (PTSD; who received clonazepam). Depressive disorders were equally distributed among patients in each treatment; 16 patients had a lifetime history of major depression, four had current major depression, and four had current dysthymia.

Rates of the generalized subtype of social phobia were almost identical in the two treatment groups (75% for CBGT and 76% for clonazepam). The two treatment groups did not differ in age (40.8 ± 10.9 for CBGT; 39.0 ± 10.2 for clonazepam) or in age of onset of social phobia (14.7 ± 8.3 for CBGT; 14.5 ± 7.6 for clonazepam). Seventy-five percent (15 of 20) of the patients who were treated with CBT were men, compared with 48% (12 of 25) of the patients treated with clonazepam (Fisher’s exact test, \( p < .08 \)).

**Procedures**

Patients referred to the study were initially screened by telephone to determine whether they met basic diagnostic and inclusion criteria. Patients were subsequently scheduled for a diagnostic and medical assessment. Diagnostic evaluation was performed with the Structured Clinical Interview for DSM-III-R (Spitzer et al., 1990). To assess treatment outcome, symptom severity was evaluated at baseline, week 4, week 8, and week 12 (posttreatment). All clinician-rated assessments were completed by individuals blind to treatment condition and included the Clinical Global Impression Scale for severity (CGI; Guy, 1976) with scores ranging from 1 (“not ill”) to 7 (“among most severely ill”), the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), and the Hamilton Rating Scale for Anxiety (HAM-A; Hamilton, 1959). Patient-rated measures included the Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1989; Heimberg et al., 1992), the Social Phobia Scale (Mattick & Clarke,
For purposes of data analysis, the CGI severity score and the SIAS were chosen on an a priori basis to serve as the primary outcome measures. In addition to these continuous variables, treatment response was assessed according to a responder criteria of a CGI severity score of 1 or 2.

In addition to social phobia severity, five additional patient characteristics were examined as predictors of treatment outcome: patient gender, presence of a current comorbid depressive disorder (major depression or dysthymia), presence of a comorbid anxiety disorders (other than simple phobia), severity of fears of anxiety sensations as assessed by the Anxiety Sensitivity Index (Peterson & Reiss, 1992), and severity of depression-related dysfunctional cognitions as assessed by the Dysfunctional Attitudes Scale (Weissman, 1979).

**Treatment Conditions**

**Clonazepam treatment.** Patients in this treatment condition were treated with clonazepam and monitored on a weekly basis by a psychiatrist experienced in the pharmacologic treatment of anxiety disorders. Patients were prescribed 0.25 mg twice daily for the first 2 days, increasing to 0.5 mg twice daily by the end of the first week. Dose was then increased by 1.0 mg per week up to a maximum daily dose of 2.0 mg twice daily. Dose titration was flexible and based on the clinician’s assessment of the relative balance of efficacy and side effects. Weekly interactions between psychiatrists and patients included review of anxiety symptoms and potential medication side effects, adjustment of dosage schedule, supportive interactions encouraging attempts to complete new social interactions, and monitoring of the success of these interactions as they were attempted. Prohibited interventions included review of the cognitive-behavioral model of social phobia, identification or modification of catastrophic cognitions associated with social phobia, relaxation training, diaphragmatic breathing training, or structuring of specific exposure assignments.

**CBGT treatment.** The CBGT intervention consisted of 12 group treatment sessions of 2.5 hours duration each. This treatment was based on the successful social phobia treatment protocol developed by Heimberg and associates (Heimberg et al., 1995; Hope & Heimberg, 1993) and was guided by a detailed treatment manual (Heimberg, 1991). Core elements of CBGT include: (1) a review of the cognitive-behavioral model of social phobia; (2) training in the identification, analysis, and restructuring of dysfunctional cognitions; (3) exposure to anxiety-provoking events in the group in a true-to-life fashion; (4) rehearsal of cognitive restructuring procedures in the context of exposure exercises; and (5) assigned homework to complete cognitive-restructuring and exposure exercise situations rehearsed in the context of the group. Treatment groups
ranged in size between three and eight members, and each was led by two therapists. Therapists included interns, fellows, and staff psychologists experienced in behavior therapy and supervised by either the first or third author.

**Data Analysis**

Differences between treatment groups were examined using analysis of covariance (ANCOVA), treating the baseline value of each outcome variable as the covariate. ANCOVAs were used to achieve two goals: (1) statistical adjustment of outcome scores to equate participants on baseline severity, and (2) evaluation of baselines scores (the covariates) as predictors of treatment outcome. To examine whether baseline severity was differentially predictive of outcome for the two treatment groups (and thereby assess the homogeneity of regression assumption required by ANCOVA), preliminary analyses examined the significance of the interaction between the covariate and treatment group. Alternate procedures (allowing for heterogeneity of regression; Maxwell & Delaney, 1990) were applied if heterogeneity of regression was evident, as evaluated by the significance of the interaction term. All outcome measures were assessed at the end of the trial in both completer (examining data only from patients completing the trial) and endpoint (data from all patients, carrying data from the last available visit forward) analyses. To examine the time-course of significant differences at outcome further, follow-up tests examined differences between treatment groups at weeks 4 and 8. In the absence of significant differences between groups, improvement over the course of treatment was evaluated using \( t \)-tests for repeated measures. Attrition in each group, and all other contingency table analyses, were examined using Fisher’s exact tests. Alpha was set at \( p < .05 \) for all statistical tests.

**RESULTS**

**Attrition**

During the course of the study, 10 of 25 patients (40%) starting clonazepam discontinued treatment before reaching study endpoint (week 12). Five of these 10 patients dropped out because of excessive side effects; the remaining six patients reported lack of efficacy or were lost to follow-up. Five of 20 patients (25%) who started CBGT discontinued treatment before endpoint. Two patients attributed their attrition to lack of efficacy and three to inconvenience or time constraints associated with the group treatment. Patients who reached week 12 were considered “completers” \( (n = 30) \). Differences between groups in attrition during treatment did not reach significance \( (p < .35) \).
Comparisons Between Treatments

Table 1 presents means and standard deviations for outcome measures at each assessment point. Endpoint ANCOVAs, carrying forward the last observation for each outcome variable, revealed no significant differences between treatment groups for any clinician- or patient-rated variable. The two treatments were also similar in rates of remission (defined as CGI-severity < 2). Twenty percent (5 of 25) of the patients treated with clonazepam and 25% (5 of 20) of the patients treated with CBGT met criterion for remission at endpoint. CGI data at each assessment point are provided in Figure 1.

Completer analyses, examining only those patients completing the week-12 assessments, indicated no significant differences on clinician-rated variables. Patient-rated outcome variables were marked by missing scores for a large proportion of patients in each treatment group; nonetheless, available scores revealed significant differences between groups for three outcome variables: the SIAS ($F = 6.28; df = 1, 16; p < .05$), FNE ($F = 4.15; df = 1, 20; p < .05$), and RAS ($F = 5.67; df = 1, 20; p < .05$). In all three cases, outcome was better at visit 12 for patients treated with clonazepam relative to patients treated with CBGT. The time course of these significant effects was further evaluated by examining differences between groups at weeks 4 and 8; no significant differences between treatments were evident at these evaluation periods. Examination of patients who completed week 8 but not week 12 assessments indicated that on average, patients dropping out of clonazepam treatment were more severe on the SIAS and FNE at week 8 than their counterparts; in contrast, this trend was not evident for these measures for CBGT patients who did not complete week 12, but was evident for the RAS.

Improvement Across Treatment

Examination of change in outcome between baseline and endpoint revealed significant improvements for all measures (all $p$ values < .001). Likewise, significant changes were evident in all completer analysis (all $p$ values < .005). For examination of changes in the SIAS, FNE, and RAS (which demonstrated differences between groups at week 12), within-group changes were considered for each group individually; each group demonstrated significant improvement across treatment ($p$ values < .05). To allow comparison with published estimates of the pre- to posttreatment effect sizes of CBT for social phobia (Feske & Chambless, 1995), we used the following equation to compute the CBGT effect size for the present study: $\frac{(M_{\text{pretest}} - M_{\text{posttest}})}{SD_{\text{pretest}}}$. The average effect size for measures specific to social anxiety or avoidance (the CGI, SIAS, LSAS, Social Phobia Scale, and FNE) was 0.92; the effect size
<table>
<thead>
<tr>
<th>Outcome Variable</th>
<th>Baseline</th>
<th>Week 4</th>
<th>Week 8</th>
<th>Week 12</th>
<th>Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CLZP</td>
<td>CBGT</td>
<td>CLZP</td>
<td>CBGT</td>
<td>CLZP</td>
</tr>
<tr>
<td>CGI±severity</td>
<td>4.8</td>
<td>4.9</td>
<td>3.8</td>
<td>4.0</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td>(0.8)</td>
<td>(0.8)</td>
<td>(0.7)</td>
<td>(1.1)</td>
<td>(1.1)</td>
</tr>
<tr>
<td>LSAS-fear</td>
<td>46.9</td>
<td>43.1</td>
<td>37.0</td>
<td>42.1</td>
<td>33.1</td>
</tr>
<tr>
<td></td>
<td>(17.9)</td>
<td>(12.9)</td>
<td>(18.3)</td>
<td>(16.3)</td>
<td>(17.3)</td>
</tr>
<tr>
<td>LSAS-avoid</td>
<td>37.1</td>
<td>34.7</td>
<td>25.3</td>
<td>26.7</td>
<td>21.5</td>
</tr>
<tr>
<td></td>
<td>(12.9)</td>
<td>(11.1)</td>
<td>(11.5)</td>
<td>(13.3)</td>
<td>(13.0)</td>
</tr>
<tr>
<td>HAM-A</td>
<td>32.4</td>
<td>30.5</td>
<td>21.2</td>
<td>22.5</td>
<td>17.6</td>
</tr>
<tr>
<td></td>
<td>(14.0)</td>
<td>(12.9)</td>
<td>(12.5)</td>
<td>(13.8)</td>
<td>(12.1)</td>
</tr>
<tr>
<td>SPS</td>
<td>15.6</td>
<td>11.8</td>
<td>9.0</td>
<td>9.2</td>
<td>8.9</td>
</tr>
<tr>
<td></td>
<td>(6.8)</td>
<td>(5.8)</td>
<td>(4.8)</td>
<td>(5.2)</td>
<td>(6.3)</td>
</tr>
<tr>
<td>FNE</td>
<td>34.7</td>
<td>25.5</td>
<td>26.1</td>
<td>24.3</td>
<td>18.8</td>
</tr>
<tr>
<td></td>
<td>(18.0)</td>
<td>(9.4)</td>
<td>(17.3)</td>
<td>(12.0)</td>
<td>(13.0)</td>
</tr>
<tr>
<td>RAS</td>
<td>44.8</td>
<td>44.2</td>
<td>39.6</td>
<td>40.1</td>
<td>35.1</td>
</tr>
<tr>
<td></td>
<td>(9.6)</td>
<td>(10.1)</td>
<td>(9.2)</td>
<td>(13.9)</td>
<td>(8.7)</td>
</tr>
</tbody>
</table>

Note. CLZP = clonazepam; CBGT = cognitive-behavior group therapy; CGI = clinician global impression of severity; SIAS = Social Interaction Anxiety Scale; LSAS = Liebowitz Social Anxiety Scale (fear and avoidance scales); HAM-A = Hamilton Rating Scale for Anxiety; SPS = Social Phobia Scale, FNE = Fear of Negative Evaluation scale; RAS = Rathus Assertiveness Schedule.

Table values are for study completers, except for the endpoint column, which provides data for all participants, with the data from last available visit carried forward.

Significant differences between groups at the designated assessment point are: *p < .05; **p < .005.
for severity of general anxiety symptoms was 0.62 as assessed by the HAM-A. Comparable metaanalytic results have not been published for high-potency benzodiazepines.

**Prediction of Treatment Response**

For each of the endpoint analyses, excluding CGI, baseline severity was a significant and positive predictor of endpoint outcome ($R$ values ranged from .48 to .60; all $p$ values < .005). For these analyses, there was no evidence of heterogeneity of regression, indicating that baseline severity was not differentially predictive of outcome for CBGT versus clonazepam.

For the completer analyses, baseline values were predictive of outcome scores for three measures: the SIAS, RAS, and HAM-A ($R$ values from .45 to .51; all $p$ values < .05). The analysis of the LSAS avoidance score revealed significant heterogeneity of regression. Examination of each treatment group separately indicated a significant positive association between the baseline and week 12 scores for the CBGT group, but no such association for patients treated with clonazepam. This results suggest that for clonazepam treatment, improvement in avoidance was not similar across levels of severity (as tended to be true for CBGT and for the analyses of other outcome measures).

In addition to the baseline values of the outcome measures, gender, depression comorbidity, anxiety comorbidity, and Anxiety Sensitivity Index and Dysfunctional Attitudes Scale scores were examined individually and in interaction with treatment group as predictors of the primary outcome measures, the CGI and the SIAS. None of these variables increased prediction over that provided by baseline severity scores.
DISCUSSION

Overall, our comparative trial of clonazepam and CBGT for social phobia indicated that both were effective treatments and revealed few significant differences in efficacy. Our sample size was not large, but adequate power, in excess of .95, was available for detection of large treatment or predictor effect sizes in the endpoint ANCOVAs. In the endpoint analyses, there was no evidence of significant differences between treatment groups, and both treatment groups achieved significant pre- to posttreatment changes. In addition, there were no differences in early treatment effects, as evaluated by week 4 and week 8 results, and no differences in clinician-rated measures at week 12. However, there was evidence of significantly better outcome on several patient-rated measures of social anxiety and avoidance, anxiety-related cognitions, and assertiveness at week 12 for patients treated with clonazepam. This treatment advantage was obtained for a subsample of patients completing the SIAS, FNE, and RAS, and may be partially explained by attrition of the more severe patients in the clonazepam treatment group.

Examination of predictors of treatment outcome in the ANCOVAs provided no consistent evidence of differential prediction of outcome for the two treatments. Endpoint analyses provided consistent evidence for a positive relationship between baseline and outcome severity for both treatment groups; higher pretreatment severity was associated with higher posttreatment severity. Similar, but less consistent, evidence for this relationship was evident in completer analyses. In multiple regression equations, no other predictor (including patient gender, anxiety comorbidity, depression comorbidity, anxiety sensitivity, and dysfunctional attitudes), examined alone or in interaction with treatment group, offered a significant increase in prediction over that provided by baseline severity for the main outcome measures. There was evidence for differential prediction for clonazepam and CBGT treatment for only one measure; for LSAS avoidance scores, pretreatment severity was associated with posttreatment outcome only for the CBGT group.

Our results at endpoint are consistent with efficacy estimates provided in effect-size analysis of controlled studies; clonazepam and CBGT have almost identical between-group effect size estimates in the Gould et al. (1997) review of controlled treatments. Evaluation of within-group effect sizes also revealed consistency with the available treatment-outcome literature. Feske and Chambless (1995) derived effect sizes based on within-group change from pre- to posttreatment in 12 studies of CBT for social phobia. For measures of social anxiety, the mean pre- to posttreatment effect size was 0.90. In the present study, we found a near identical pre- to posttreatment effect size of 0.92 for CBGT treatment for measures of social anxiety and avoidance. Likewise, Feske and Chambless (1995) reported a mean effect size of 0.58 for measures of anxiety and depression severity; we obtained a CBGT effect size of 0.62 for changes in general anxiety as assessed by the HAM-A.
The results of our study suggest that, on average, patients randomized to clonazepam and CBGT treatment are equally likely to respond during the acute treatment phase, although treatment completers may enjoy a stronger advantage with clonazepam. It is important to note that clonazepam treatment, similar to the pharmacotherapy conditions examined by Gelertner et al., (1991), was conducted in the context of clinicians’ encouragement of patients to enter avoided social situations. This encouragement to conduct self-guided exposure may well have aided treatment gains for the clonazepam condition, providing a clinically meaningful index of improvement when this element of psychosocial care is used as part of standard psychopharmacology. To disallow these supportive interventions is to construct a treatment that diverges from recommendations for standard pharmacologic treatment of social phobia (Sutherland & Davidson, 1995). Nonetheless, it should be understood that these procedures may have increased efficacy estimates for the clonazepam condition and may have provided a more stringent test of the relative efficacy of CBGT.

Examination of measures of social phobia severity revealed no consistent evidence for the selection of one treatment over another; predictors of poorer response to one treatment were also predictors of poorer response to the other treatment. As such, there is little empirical evidence to guide treatment choice firmly in the short-term, and choice of treatment may depend more on treatment availability and patient preference. The issue of treatment preference is clouded by the exceptionally low rates at which individuals with social phobia seek treatment (Schneier et al., 1992), and in our sample we observed an average 24-year interval between the onset of the social phobia and entry into our treatment trial. Treatment seeking may be limited by the tendency of those with social phobia to avoid the social anxiety associated with the interpersonal interactions inherent in treatment, but our study provided no evidence that once initiated, a psychosocial treatment is less tolerable than a pharmacologic treatment. Overall, we observed a 40% drop-out rate for clonazepam treatment and a 25% percent drop-out rate for CBGT.

Treatment choice should also be informed by long-term outcome, but few studies have addressed this issue in social phobia. Available studies indicate the return of symptoms after medication discontinuation (Davidson, Tupler, & Potts, 1994; Versiani et al., 1992) and that maintenance or additional treatment is common among individuals who initiated pharmacotherapy (Sutherland, Tupler, Colket, & Davidson, 1996). There is also tentative evidence for slippage of treatment effects even if pharmacotherapy is maintained (Liebowitz et al., 1999). In contrast, a number of studies of CBT for social phobia indicate that additional treatment gains are achieved over follow-up intervals (Gould et al., 1997; Heimberg et al., 1993; Mersch, Emmelkamp, & Lips, 1991; Turner, Beidel, & Cooley-Quille, 1995), suggesting that patients continue to apply principles and skills learned during the short-term treatment phase.
In summary, our study provides evidence of generally equal efficacy for clonazepam and CBGT, with some evidence of more optimal acute outcome for patients maintaining clonazepam treatment at the end of the trial. Our study is limited by a relatively low sample size and a relatively high attrition rate. However, effect size comparisons indicate our acute outcome findings are consistent with previous studies and are consistent with other recent comparisons of CBGT and pharmacotherapy (Heimberg et al., 1994). The best strategy for long-term treatment of patients with social phobia remains an open question, with some evidence suggesting consolidation and extension of treatment gains at follow-up in patients treated with CBT.

REFERENCES


