

# Cognitive-Behavioral Therapy as an Adjunct to Serotonin Reuptake Inhibitors in Obsessive-Compulsive Disorder: An Open Trial

H. Blair Simpson, M.D., Ph.D.;  
Kenneth S. Gorfinkle, Ph.D.; and Michael R. Liebowitz, M.D.

---

**Background:** We report the results of an open trial of cognitive-behavioral therapy (CBT) using exposure and ritual prevention as an adjunct to serotonin reuptake inhibitors (SRIs) in obsessive-compulsive disorder (OCD). We hypothesized that exposure and ritual prevention would significantly reduce OCD symptoms in patients who remained symptomatic despite an adequate trial of an SRI and enable patients to discontinue their medication.

**Method:** OCD patients taking an adequate dose of an SRI  $\geq 12$  weeks who remained symptomatic (i.e., a Yale-Brown Obsessive Compulsive Scale [Y-BOCS] score  $\geq 16$ ) were eligible. While taking a stable dose of an SRI, patients received 17 sessions of exposure and ritual prevention. For the intent-to-treat group, the paired t test was used to compare scores on the Y-BOCS, the National Institute of Mental Health (NIMH) Global OCD scale, the Clinical Global Impressions scale, and the Hamilton Rating Scale for Depression before and after exposure and ritual prevention.

**Results:** Six of 7 eligible patients entered the study, and 5 completed it. All 6 improved on all OCD measures. The mean  $\pm$  SD Y-BOCS score was  $23.8 \pm 2.6$  prior to exposure and ritual prevention and  $12.2 \pm 4.3$  after it ( $p < .001$ ). The mean percentage decrease on the Y-BOCS was 49% (range, 26%–61%). Patients were rated by the therapist and rated themselves as much ( $N = 4$ ) or very much ( $N = 2$ ) improved. Blood drug levels did not change in most patients during exposure and ritual prevention; thus, the improvement was attributed to this type of therapy. No patients discontinued their medication.

**Conclusion:** This open trial suggests that CBT using exposure and ritual prevention can lead to a significant reduction in OCD symptoms in patients who remain symptomatic despite an adequate trial of an SRI.

(*J Clin Psychiatry* 1999;60:584–590)

---

Received July 28, 1998; accepted Dec. 29, 1998. From the New York State Psychiatric Institute, New York (Drs. Simpson and Liebowitz), and the Department of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, N.Y. (all authors).

This study was supported by National Institute of Mental Health grants 5T32-MH15144-20 and 2P30-MH30906 MHCRC-NYSPI.

We thank Donna Y. Vermes, R.N., for performing structured clinical interviews; Thomas B. Cooper, M.A., for performing the drug assays; James Broatch and the Obsessive-Compulsive Foundation, Inc., for help in recruiting patients; and Michael J. Kozak, Ph.D., for commenting on an earlier version of this article.

Reprint requests to: H. Blair Simpson, M.D., Ph.D., Anxiety Disorders Clinic, P.I. Unit 69, New York State Psychiatric Institute, 1051 Riverside Dr., New York, NY 10032.

Obsessive-compulsive disorder (OCD) is an illness characterized by intrusive and distressing thoughts, urges, and/or images (obsessions) and repetitive thoughts or behaviors (compulsions) aimed at reducing the distress triggered by the obsessions.<sup>1</sup> Once thought rare, OCD is now reported to have a lifetime prevalence as high as 2% to 3%.<sup>2,3</sup> Because of the significant suffering and impairment in social and occupational functioning caused by OCD,<sup>4,5</sup> the World Health Organization concluded that OCD was one of the world's most disabling illnesses.<sup>6</sup> Thus, improving treatments for OCD is important.

The best current treatments for OCD (reviewed in Kozak et al.<sup>7</sup>) are the serotonin reuptake inhibitors (SRIs; e.g., clomipramine, fluoxetine, fluvoxamine, sertraline, paroxetine) and cognitive-behavioral therapy (CBT) using exposure and ritual prevention. Problems with the SRIs include limited acute efficacy, occurrence of side effects, and a high relapse rate for patients who stop medication (e.g., because of pregnancy). For example, estimates for clomipramine, arguably the most potent available medication, are that approximately 50% of patients respond and that responders achieve, on average, about a 40% reduction in symptoms.<sup>7,8</sup> Those who stop clomipramine relapse up to 89% of the time.<sup>9</sup> The response rate for those who complete exposure and ritual prevention is higher: on average, 83%. However, up to 30% of patients refuse this type of therapy, and many who start it do not complete it.<sup>7</sup> Thus, OCD is a severe mental illness, and the 2 best treatments have serious limitations.

Because of these limitations, combination treatments have been proposed. These combinations include augmenting SRIs with other medications (including buspirone, lithium, clonazepam, fenfluramine, tryptophan, and neuroleptics; reviewed in Pigott and Seay<sup>8</sup>) and combining SRI treatment with exposure and ritual prevention (reviewed in Kozak et al.<sup>7</sup> and van Balkom van Dyke<sup>10</sup>). The combination of exposure and ritual prevention therapy and SRIs has not been found to be clearly superior to therapy alone in most studies that have examined this question.<sup>7,10-17</sup> At least in some cases, this lack of superiority could be due to the way the 2 treatments are combined. For example, in the ongoing comparison of clomipramine and exposure and ritual prevention sponsored by the National Institute of Mental Health (NIMH),<sup>7</sup> OCD patients receiving the combination treatment start medication and therapy at the same time. However, since the intensive exposure and ritual prevention occurs 5 times a week for the first 3 weeks, and clomipramine can take 6 to 10 weeks to show much effect, the intensive phase of the therapy is over before the medication has time to take effect.

Some have reported that exposure and ritual prevention can reduce symptoms in OCD patients who are already taking medication.<sup>12,18-20</sup> However, we know of no published study that examined the benefit of exposure and ritual prevention in OCD patients who remained symptomatic despite an adequate dose (as defined below) and duration (e.g.,  $\geq 12$  weeks) of an SRI. In addition, we know of no such study that used blood drug levels to document medication compliance.

We report the results of an open pilot trial of exposure and ritual prevention as an adjunct to SRIs in OCD. We hypothesized that this type of therapy would lead to a reduction in OCD symptoms in patients who remained symptomatic despite an adequate trial of an SRI and that those who improved with this therapy would be able to discontinue their SRI with a lower relapse rate than that reported in the literature.

## METHOD

This study was conducted at an outpatient clinic that specializes in the treatment of anxiety disorders. Patients were recruited by advertisements and by referrals from other clinicians. To be eligible, patients had to meet DSM-IV<sup>1</sup> criteria for OCD as their primary disorder, have had OCD for at least 1 year, and be between the ages of 18 and 65 years. They had to have experienced some improvement (by verbal report) on an adequate dose (see below) and duration ( $\geq 12$  weeks) of an SRI (e.g., clomipramine, fluoxetine, fluvoxamine, paroxetine, sertraline), but remained symptomatic (i.e., a Yale-Brown Obsessive Compulsive Scale [Y-BOCS<sup>21,22</sup>] score of  $\geq 16$ ). Some improvement while taking an SRI was required so that it would be ethical to maintain the patients on this medication during

the study. An adequate dose of an SRI for a duration of  $\geq 12$  weeks was required so that patients were likely to have already experienced the maximum benefit from their SRI medication.<sup>8</sup> In congruence with the literature,<sup>8,23</sup> an adequate dose of medication for OCD was defined as clomipramine,  $\geq 225$  mg/day; fluoxetine,  $\geq 60$  mg/day; paroxetine,  $\geq 60$  mg/day; sertraline,  $\geq 200$  mg/day; and fluvoxamine,  $\geq 250$  mg/day. Patients were excluded if they had already had CBT using exposure and ritual prevention for OCD; were taking psychoactive substances other than SRIs; and/or had a current episode of major depression, a current or past history of psychosis or mania, and/or a diagnosis of alcohol or substance abuse or dependence in the past 6 months. Eligibility for the study was determined by one of the authors (H.B.S.) during a comprehensive psychiatric assessment; the psychiatric diagnosis was confirmed using the Structured Clinical Interview for DSM-IV.<sup>24</sup> Medication history was confirmed by the provider who prescribed the medication and/or by review of the medication record. Written informed consent was obtained after full explanation of the study procedures.

While taking medication, patients received 17 sessions of CBT using exposure and ritual prevention. This therapy involves live exposure to feared situations, imaginal exposure to feared consequences, and ritual prevention, in which patients refrain from compulsive rituals. We followed the exposure and ritual prevention protocol as described by Kozak and Foa<sup>25</sup> with one exception: instead of sessions 5 times a week, the therapy sessions were scheduled twice a week, with allowances made for occasional scheduling conflicts. This scheduling change was made to enhance recruitment and to examine a version of the therapy that would be more applicable to the typical clinical setting. The 17 exposure and ritual prevention sessions each lasted 1½ hours and included 2 treatment planning sessions and 2 home visits. The therapy was conducted by a psychiatrist (H.B.S.), trained and supervised in exposure and ritual prevention by an experienced cognitive-behavioral therapist (K.S.G.). Before and after the therapy, clinical symptoms were assessed using the Y-BOCS, the NIMH Global Obsessive Compulsive Severity Scale<sup>26,27</sup> (NIMH-OC), the Clinical Global Impressions (CGI) scale (severity and improvement subscales),<sup>28</sup> and the 17-item Hamilton Rating Scale for Depression (HAM-D<sup>29</sup>). In addition, patients filled out a patient version of the CGI-improvement scale. Blood drug levels were taken before and after exposure and ritual prevention to ensure compliance with medication and to exclude the possibility that changes in symptoms were due to changes in blood drug levels. For each patient, blood drug levels were taken at the same time of day in relationship to the last dose of medication. Patients with a  $\geq 50\%$  reduction in their Y-BOCS score at the end of exposure and ritual prevention were eligible to enter a medication discontinuation phase.

**Table 1. Y-BOCS Scores at Intake, Before and After Exposure and Ritual Prevention Therapy, and During the Follow-Up in 6 OCD Patients Taking SRIs<sup>a</sup>**

Patient	Daily Medication (duration prior to exposure and ritual prevention) <sup>b</sup>	Intake	Start of Exposure and Ritual Prevention	End of Exposure and Ritual Prevention	1 mo	2 mo	3 mo	4 mo	5 mo	6 mo	9 mo	12 mo
1	Fluvoxamine 300 mg (12 weeks)	26	27	20	18	21	23	20	26	23	17	20
2	Fluoxetine 100 mg (> 1.5 years)	24	22	9	NA	13	12	11	NA	NA	15 <sup>c</sup>	NA
3	Fluoxetine 80 mg Clomipramine 50 mg (> 1 year)	22	26	13	11	12	13	12	12	12	10	11
4	Fluoxetine 60 mg (16 weeks) then fluoxetine 70 mg (8 weeks)	27	25	13	13	16	23	22	17	17	19	...
5	Fluvoxamine 300 mg (> 10 years)	22	20	9	33	4	16	8	25	20	13	...
6	Fluoxetine 40–60 mg (2.5 years) then fluoxetine 60 mg (26 weeks)	21	23	9	6	NA	NA	NA	NA	21	...	...

<sup>a</sup>Abbreviations: NA = not available, OCD = obsessive-compulsive disorder, SRI = serotonin reuptake inhibitor, Y-BOCS = Yale-Brown Obsessive Compulsive Scale.

<sup>b</sup>Prior to these medications for OCD, the pharmacotherapy history of the 6 patients was as follows. Patient 1 had a previous trial of paroxetine, which was of questionable benefit, and a previous trial of clomipramine, which was beneficial but was stopped because of intolerable side effects. Patient 2 had a previous trial of fluvoxamine that was stopped because of intolerable side effects. Patient 3 had a previous trial of fluoxetine monotherapy. Patient 4 had a previous trial of paroxetine, sertraline, fluvoxamine, and clomipramine; all were stopped because of intolerable side effects. Patient 5 had a history of augmenting fluvoxamine with clonazepam, lithium, buspirone, and methylphenidate hydrochloride. Patient 6 had no prior medication trials.

<sup>c</sup>Patient was unable to be assessed at 6, 9, and 12 months, but was seen at 7.5 months.

Patients were contacted at 1, 2, 3, 4, 5, 6, 9, and 12 months after the end of exposure and ritual prevention and reassessed over the phone and/or in person, each time for approximately 45 minutes. Ongoing problems with OCD symptoms were discussed, the principles of exposure and ritual prevention were reiterated, and clinical symptoms were assessed using the scales listed above; however, no live or imaginal exposure occurred. Patients were also asked about any additional treatment they had received in the intervening period.

A statistical analysis was done on the intent-to-treat sample, which included all patients who entered the study. For the analysis, the paired *t* test was used to compare scores on the Y-BOCS, the NIMH-OC, the CGI-severity scale, and the HAM-D before and after receiving exposure and ritual prevention. The last observations were carried forward for the patient who did not complete the study. For all tests, a *p* value < .05 (2-tailed) was considered significant.

## RESULTS

To recruit those who met the stringent inclusion criteria, 47 patients with OCD were screened, and 40 were excluded. Most (*N* = 21, 53%) were excluded because they either were not taking an adequate dose of an SRI (usually because of intolerable side effects) or were taking benzodiazepines or other excluded medications. Some (*N* = 7, 18%) were not currently taking medication and wanted

only exposure and ritual prevention. Some (*N* = 6, 15%) were excluded because they had previously received exposure and ritual prevention. Of 7 eligible patients, 1 declined to participate after learning more about exposure and ritual prevention.

Six patients entered the protocol, 5 men and 1 woman. Five completed all 17 sessions; 1 (Table 1; patient 6) stopped after the 14th session because of an unexpected medical problem that made him unable to complete the final 3 sessions. The mean  $\pm$  SD age of those who entered was  $35 \pm 3.3$  years. Six had entered college, 5 had completed college, and 2 (patients 3 and 5) had completed graduate school. Five patients were employed, and 1 was a full-time graduate student. The mean age at onset of OCD was  $13.5 \pm 5.3$  years.

Besides a current diagnosis of OCD, all 6 patients had other Axis I psychiatric disorders. Patient 1 had comorbid generalized anxiety disorder. Patient 2 had a history of social phobia and major depressive disorder. Patient 3 had specific phobia, dysthymia, and a history of major depressive disorder. Patients 4 and 5 both had histories of major depressive disorder. Patient 6 had a history of both major depressive disorder and panic disorder with agoraphobia. None had a current episode of major depression (HAM-D mean score =  $8.7 \pm 2.7$ ), and none reported a history of tics.

The patients had a range of OCD symptoms. Two patients (patients 2 and 6; see Table 1) primarily experienced contamination and somatic obsessions and cleaning compulsions. The remaining 4 (patients 1, 3, 4, 5) had

multiple obsessions and compulsions from the categories listed in the Y-BOCS symptom checklist. Patient 1 primarily experienced aggressive, contamination, and symmetry obsessions and cleaning, checking, and ordering compulsions. Patient 3 primarily had aggressive, contamination, and hoarding obsessions and cleaning, checking, counting, and hoarding compulsions. Patient 4 mostly experienced aggressive, contamination, hoarding, and symmetry obsessions and cleaning, checking, repeating, counting, ordering, and hoarding compulsions. Patient 5 mostly had aggressive, sexual, and religious obsessions and mental and checking compulsions. As shown in Table 1, 3 patients were taking fluoxetine only (60, 70, and 100 mg/day, respectively), 2 were taking fluvoxamine (300 mg/day), and 1 was taking fluoxetine (80 mg/day) and clomipramine (50 mg/day) at the time they received exposure and ritual prevention therapy. All but 1 had previously taken other OCD medications as well (see Table 1).

Prior to receiving exposure and ritual prevention, all patients had Y-BOCS ratings twice: at intake and at the start of therapy (see Table 1). The mean time interval between these 2 ratings was  $21.2 \pm 11$  days (range, 6–39 days). There was no significant difference in the Y-BOCS scores between intake and the start of exposure and ritual prevention therapy (mean at intake =  $23.7 \pm 2.4$ ; mean at start of therapy =  $23.8 \pm 2.6$ ;  $t = -0.16$ ,  $df = 5$ ,  $p = .88$ ).

After receiving exposure and ritual prevention, all 6 patients improved significantly on all outcome measures that assessed OCD symptoms, as shown in Table 2. The mean Y-BOCS score was  $23.8 \pm 2.6$  prior to exposure and ritual prevention and  $12.2 \pm 4.3$  after the therapy ( $p < .001$ ). The mean percent decrease in the Y-BOCS score was 49% (range, 26%–61%). Individual Y-BOCS scores before and after exposure and ritual prevention are shown in Table 1. All patients were rated by the therapist and rated themselves as much ( $N = 4$ ) or very much improved ( $N = 2$ ).

The clinical improvement was attributable to exposure and ritual prevention. All patients remained on the same dose of medication while receiving this type of therapy. In 4 patients, the blood drug levels were nearly identical before and after therapy (Table 3; patients 1, 2, 4, 5). In another patient (patient 3), blood drug levels were higher at the end of therapy, but 1 month later, the levels had decreased again with continued clinical improvement. Thus, the clinical improvement in this patient was also attributable to exposure and ritual prevention. Blood drug levels after exposure and ritual prevention were not available for the sixth patient. Of the 5 patients with drug levels before and after therapy, 3 were taking fluoxetine, and 2 were taking fluvoxamine. For the 3 patients taking fluoxetine (pa-

**Table 2. Response to Exposure and Ritual Prevention Therapy in 6 OCD Patients Taking SRIs<sup>a</sup>**

Scale	Exposure and Ritual Prevention						t	df	p Value	95% CI of the Difference
	Start		End		Difference					
	Mean	SD	Mean	SD	Mean	SD				
Y-BOCS score	23.8	2.6	12.2	4.3	-11.7	2.5	11.4	5	<.001	9.0 to 14.3
NIMH-OC score	7.8	1.0	5.7	1.5	-2.2	0.8	7.1	5	.001	1.4 to 3.0
CGI-severity score	4.5	0.5	3.2	0.7	-1.3	0.5	6.3	5	.001	0.8 to 1.9
HAM-D score	8.7	2.5	6.7	1.4	-2.0	2.8	1.8	5	.14	-9 to 4.9

<sup>a</sup>Abbreviations: CGI = Clinical Global Impressions scale, CI = confidence interval, HAM-D = 17-item Hamilton Rating Scale for Depression, NIMH-OC = National Institute of Mental Health Global Obsessive Compulsive Severity Scale.

**Table 3. Blood SRI Levels Before and After Exposure and Ritual Prevention Therapy**

Patient	Medication	Daily Dose	Exposure and Ritual Prevention		
			Start (ng/mL)	End (ng/mL)	1 mo later (ng/mL)
1	Fluvoxamine	300 mg	567	563	...
2	Fluoxetine	100 mg	598	659	...
3	Norfluoxetine		618	623	...
	Fluoxetine	80 mg	347	535	243
	Norfluoxetine		277	397	392
4	Clomipramine	50 mg	150	208	98
	Desmethyl-clomipramine		83	140	120
5	Fluoxetine	70 mg	604	569	...
	Norfluoxetine		307	285	...
6	Fluvoxamine	300 mg	294	271	...
6	Fluoxetine	60 mg	427	...	...
	Norfluoxetine		271	...	...

tients 2, 3, and 4), the mean blood fluoxetine levels before and after exposure and ritual prevention were  $516 \pm 147$  ng/mL and  $588 \pm 64$  ng/mL, respectively. For the 2 patients taking fluvoxamine (patients 1 and 5), the mean blood fluvoxamine levels before and after exposure and ritual prevention were  $431 \pm 193$  ng/mL and  $417 \pm 206$  ng/mL, respectively. A repeated measures analysis of variance was performed using a univariate approach with a between-subject factor of medication (fluoxetine, fluvoxamine) and a within-subject factor of time (start of therapy, end of therapy). Neither medication ( $F = 0.999$ ,  $df = 1$ ,  $p = .111$ ) nor time ( $F = 0.478$ ,  $df = 1$ ,  $p = .539$ ) had a significant effect on blood drug levels. Moreover, there was no significant medication  $\times$  time interaction ( $F = 1.028$ ,  $df = 1$ ,  $p = .385$ ).

Despite significant improvement, no patient entered the medication discontinuation phase. The reasons included patient's fear of symptom return ( $N = 2$ ), necessity of ongoing medication for maintenance treatment of severe recurrent depression ( $N = 2$ ), and failure of the Y-BOCS score to decrease by the required  $\geq 50\%$  to enter the discontinuation phase ( $N = 2$ ).

Patients were followed for up to 1 year after the end of exposure and ritual prevention. Because they entered the

protocol at different times, they were followed for different periods of time (see Table 1); the mean length of follow-up was 9 months (range, 6–12 months). If the Y-BOCS and CGI ratings at the last available follow-up visit are examined, 5 (83%) of 6 patients (1 through 5) remained much improved with a  $\geq 6$  point decrease in their Y-BOCS score compared with their Y-BOCS score at the start of exposure and ritual prevention. Two of these patients (patients 1 and 3) had a Y-BOCS score at the last available follow-up visit as good or better than their score at the end of exposure and ritual prevention. However, if the individual pattern of monthly ratings is examined (see Table 1), 2 patients maintained their gains in the follow-up phase with no (patient 3) or only gradual slippage (patient 2). The other 4 patients (patients 1, 4, 5, and 6) suffered essentially full relapses during certain months of follow-up, which they ascribed to not having applied the principles of exposure and ritual prevention those months and to decreasing (patient 5) or stopping their medication (patient 6).

Two patients (patients 1 and 2) did not change their medication or seek further treatment during the follow-up period; both remained much improved at the final follow-up visit. Four patients (patients 3 through 6) changed their treatment during the follow-up period. Patient 3 sought couples therapy and individual cognitive-behavioral therapy for dysthymia; the latter permitted booster sessions for OCD as needed (approximately 1 time per month). In the follow-up period, he reduced his fluoxetine from 80 to 40 mg/day (from 80 to 60 mg/day 1 week prior to the 5-month follow-up and from 60 to 40 mg/day 10 weeks before the 9-month follow-up) and his clomipramine from 50 to 25 mg/day (1 week prior to the 4-month follow-up) while maintaining his gains. After the 9-month visit, he briefly increased his fluoxetine and clomipramine because of increasing OCD symptoms and then reduced them again to fluoxetine, 40 mg/day, and clomipramine, 25 mg/day, 2 weeks prior to the 12-month visit. Patient 4, because of ongoing struggles with his OCD symptoms, increased his fluoxetine from 70 to 80 mg/day 2 weeks prior to the 1-month follow-up, augmented his fluoxetine with buspirone (up to 60 mg/day: 10 to 20 mg/day 2 weeks prior to the 3-month follow-up visit, 30 mg/day prior to the 6-month follow-up visit, and 60 mg/day 1 week prior to the 9-month follow-up visit), and attended a weekly OCD self-help group. Patient 5, despite struggling with relapses, decreased his fluvoxamine from 300 to 200 mg/day 2 weeks prior to the 3-month follow-up because he believed that the exposure and ritual prevention skills he had learned were more effective at combating his OCD symptoms than the higher dose of medication, and he wanted fewer medication side effects. He subsequently increased his fluvoxamine to 300 mg/day (to 250 mg/day after the 5-month follow-up and to 300 mg/day 5 weeks before the 9-month follow-up) and

started clonazepam (up to 1 mg/day: 0.5 mg p.r.n. for insomnia prior to the 5-month follow-up visit, 1 mg/day approximately 8 weeks prior to the 9-month follow-up visit) because of returning OCD symptoms. At his last follow-up visit (at 9 months), he had recently stopped his fluvoxamine altogether because he felt it was not helping him and that he was more successful with the exposure and ritual prevention techniques when he was not taking the drug. Patient 6 was lost to follow-up during the 2nd, 3rd, 4th, and 5th months. During the 2nd month, he stopped taking fluoxetine. At the 6-month follow-up visit, he had been off all medication for 4 months, had stopped practicing his exposure and ritual prevention techniques, and had experienced a full relapse of OCD.

## DISCUSSION

The results of this open trial of exposure and ritual prevention as an adjunct to SRI treatment are striking: for these OCD patients who remained symptomatic despite an adequate trial of an SRI, exposure and ritual prevention resulted in a significant further reduction in OCD symptoms. All patients benefited, some quite dramatically. The benefit occurred despite the fact that the therapy was conducted twice a week (instead of 5 times a week) and performed by a therapist who had not previously conducted CBT using exposure and ritual prevention. The degree of additional benefit (mean percentage decrease in Y-BOCS score = 49%) is as great or greater than that reported for placebo-controlled trials of SRIs in OCD (reviewed in Pigott and Seay<sup>8</sup>). Moreover, this degree of improvement is as great or greater than that reported for the augmentation of SRIs with other medications, including buspirone,<sup>30–32</sup> lithium,<sup>33,34</sup> clonazepam,<sup>35</sup> and neuroleptics (haloperidol<sup>36</sup> and risperidone<sup>37</sup>). In the follow-up period, 5 of 6 patients remained much improved at the last available follow-up visit, although only 2 of these maintained their improvement without any significant periods of relapse.

Whether all OCD patients who remain symptomatic despite SRI medication can benefit from exposure and ritual prevention is unclear. The results of this open study were based on a small sample of highly educated and employed patients who were motivated to try this type of therapy. These findings need to be replicated in a larger sample of OCD patients, with blind assessments and a placebo-control group. Moreover, the durability of the benefit needs to be further studied. It would be useful to know whether patients with comorbid conditions such as tic disorders and psychotic disorders can benefit from exposure and ritual prevention augmentation. In addition, it would be useful to know whether exposure and ritual prevention can help patients who are taking lower doses of SRIs and/or who are taking medications in addition to SRIs (e.g., benzodiazepines), since these patients were excluded from this study, but are common among the pa-

tients who were initially screened. Despite the limitations of this study, the results suggest that exposure and ritual prevention may be one of the best methods for SRI augmentation in OCD patients who are willing to participate in this therapy, both because of the magnitude of the potential benefit and the safety of the treatment.

Others have also reported that exposure and ritual prevention can help OCD patients who are taking medication.<sup>12,18-20</sup> The advantage of the present study is that it was designed to separate the medication and exposure and ritual prevention effects. In this study, all patients were on an adequate dose of an SRI for a minimum of 12 weeks (and most for much longer; see Table 1), and thus these patients presumably could expect minimal further improvement from their medication. In addition, this study used blood drug levels to confirm compliance with medication, and in 4 cases, to document negligible changes in blood SRI levels during exposure and ritual prevention therapy. Our results illustrate that this type of therapy can augment an adequate trial of an SRI.

Despite the dramatic improvement, no patient elected to discontinue the medication at the end of exposure and ritual prevention. This could be due to the underlying psychopathology of the sample: all had Y-BOCS scores at baseline  $\geq 20$  despite an adequate trial of an SRI, most had obsessions and compulsions in multiple domains, and 2 had histories of severe recurrent major depression and thus required maintenance medication for this reason. Medicated OCD patients who started the therapy with fewer residual OCD symptoms might be more capable (and less fearful) of tapering their medication with adjunctive exposure and ritual prevention. In addition, patients may be more successful at tapering or stopping their medication if they are receiving ongoing therapy during this process (although see Baer et al.<sup>38</sup>). Of note, 1 patient (Table 1; patient 3) cut his medication dose in half during the follow-up period while maintaining his gains; whether the additional cognitive-behavioral therapy that he received was necessary to achieve this outcome is unknown. The 2 other patients who either reduced (patient 5) or stopped (patient 6) their medication during the follow-up period suffered a return of OCD symptoms; neither received additional cognitive-behavioral therapy.

There are limitations of this study: the sample is small, there is no control group, and the ratings were done by the treating therapist and thus are subject to bias. Nonetheless, this open trial suggests that exposure and ritual prevention can acutely reduce OCD symptoms in patients who remain symptomatic despite an adequate trial of an SRI. The long-term benefits of adjunctive exposure and ritual prevention need further study. The 2 first-line treatments for OCD are the SRIs and exposure and ritual prevention.<sup>39</sup> For OCD patients who first receive SRIs, CBT using exposure and ritual prevention is worth considering if residual OCD symptoms persist.

*Drug names:* buspirone (BuSpar), clomipramine (Anafranil and others), clonazepam (Klonopin and others), fluoxetine (Prozac), fluvoxamine (Luvox), haloperidol (Haldol and others), methylphenidate (Ritalin), paroxetine (Paxil), risperidone (Risperdal), sertraline (Zoloft).

## REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
2. Robins LN, Helzer JE, Weissman MM, et al. Lifetime prevalence of specific psychiatric disorders in three sites. *Arch Gen Psychiatry* 1984;41: 949-958
3. Antony MM, Downie F, Swinson RP. Diagnostic issues and epidemiology in obsessive-compulsive disorder. In: Swinson RP, Antony MM, Rachman S, et al, eds. *Obsessive-Compulsive Disorder: Theory, Research, and Treatment*. New York, NY: Guilford Publications; 1998:3-32
4. Leon AC, Portera L, Weissman MM. The social costs of anxiety disorders. *Br J Psychiatry* 1995;166(suppl 27):19-22
5. Koran LM, Thienemann ML, Davenport R. Quality of life for patients with obsessive-compulsive disorder. *Am J Psychiatry* 1996;153:783-788
6. Murray CJ, Lopez AD, eds. *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability From Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020*. Cambridge, Mass: Harvard University Press; 1996
7. Kozak MJ, Liebowitz ML, Foa EB. Cognitive behavior therapy and pharmacotherapy for OCD. In: Goodman WK, Maser JD, Rudorfer M, eds. *Treatment Challenges in Obsessive-Compulsive Disorder*. Mahwah, NJ: Lawrence Erlbaum Associates. In press
8. Pigott TA, Seay S. Biological treatments for obsessive-compulsive disorder: literature review. In: Swinson RP, Antony MM, Rachman S, et al, eds. *Obsessive-Compulsive Disorder: Theory, Research, and Treatment*. New York, NY: Guilford Publications; 1998:298-326
9. Pato MT, Zoka-Kadouch R, Zohar J, et al. Return of symptoms after desensitization of clomipramine and patients with obsessive-compulsive disorder. *Am J Psychiatry* 1988;145:1521-1525
10. Van Balkom AJLM, van Dyck R. Combination treatments for obsessive-compulsive disorder. In: Swinson RP, Antony MM, Rachman S, et al, eds. *Obsessive-Compulsive Disorder: Theory, Research, and Treatment*. New York, NY: Guilford Publications; 1998:349-366
11. Amin MD, Ban TA, Pecknold JC, et al. Clomipramine (Anafranil) and behavior therapy in obsessive-compulsive and phobic disorders. *J Int Med Res* 1977;5(suppl 5):33-37
12. Neziroglu F. A combined behavioral-pharmacotherapy approach to obsessive-compulsive disorders. In: Oriols J, Ballus C, Gonzalez M, et al, eds. *Biological Psychiatry Today*. Amsterdam, the Netherlands: Elsevier/North-Holland Press; 1979:591-596
13. Marks IM, Stern RS, Mawson D, et al. Clomipramine and exposure for obsessive-compulsive rituals. *Br J Psychiatry* 1980;136:1-25
14. Marks IM, Lelliott PT, Basoglu M, et al. Clomipramine, self-exposure and therapist-aided exposure for obsessive-compulsive rituals. *Br J Psychiatry* 1988;152:522-534
15. Cottraux J, Mollard E, Bouvard M, et al. A controlled study of fluvoxamine and exposure in obsessive-compulsive disorder. *Int Clin Psychopharmacol* 1990;5:17-30
16. Van Balkom AJL, de Hann E, van Oppen P, et al. Cognitive and behavioral therapies alone versus in combination with fluvoxamine in the treatment of obsessive compulsive disorder. *J Nerv Ment Dis* 1998;186:492-499
17. Hohagen F, Winkelmann G, Rasche-Rauchle H, et al. Combination of behaviour therapy with fluvoxamine in comparison with behaviour therapy and placebo: results of a multicentre study. *Br J Psychiatry* 1998; 173(suppl 35):71-78
18. Neziroglu FA, Yarura-Tobias J. Long-term outcome in OCD. In: Syllabus and Proceedings Summary of the 149th Annual Meeting of the American Psychiatric Association; May 8, 1996; New York, NY. Abstract 94B:162
19. Pato MT, Pato CN, Gunn SA. Biological treatments for obsessive-compulsive disorder: clinical applications. In: Swinson RP, Antony MM, Rachman S, et al, eds. *Obsessive-Compulsive Disorder: Theory, Research, and Treatment*. New York, NY: Guilford Publications; 1998:327-348
20. Steketee G, Chambless DL, Eisen JL. Long-term behavioral treatment for OCD. In: Syllabus and Proceedings Summary of the 149th Annual Meeting of the American Psychiatric Association; May 8, 1996; New York, NY. Abstract 94E:163

21. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale, I: development, use, and reliability. *Arch Gen Psychiatry* 1989;46:1006-1011
22. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale, II: validity. *Arch Gen Psychiatry* 1989;46:1012-1016
23. Greist JH, Jefferson JW, Kobak KA, et al. Efficacy and tolerability of serotonin transport inhibitors in obsessive-compulsive disorder. *Arch Gen Psychiatry* 1995;52:53-60
24. First MB, Spitzer RL, Gibbon M, et al. *Structured Clinical Interview for DSM-IV Axis I Disorders, Patient Edition*. New York, NY: Biometric Research, New York State Psychiatric Institute; 1996
25. Kozak MJ, Foa EB. *Mastery of Obsessive-Compulsive Disorder: A Cognitive-Behavioral Approach—Therapist Guide*. San Antonio, Tex: Graywind Publications; 1997
26. Insel TR, Murphy DL, Cohen RM, et al. Obsessive-compulsive disorder: a double-blind trial of clomipramine and clorgyline. *Arch Gen Psychiatry* 1983;40:605-612
27. Murphy DL, Pickar D, Alterman IS. Methods for the quantitative assessment of depressive and manic behavior. In: Burdock EI, Sudilovsky A, Gershon S, eds. *The Behavior of Psychiatric Patients*. New York, NY: Marcel Dekker; 1982
28. Guy W. *ECDEU Assessment Manual for Psychopharmacology*. US Dept Health, Education, and Welfare, publication (ADM 76-338). Rockville, Md: National Institute of Mental Health; 1976:218-222
29. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry* 1960;23:56-62
30. Pigott TA, L'Heureux F, Hill JL, et al. A double-blind study of adjuvant buspirone hydrochloride in clomipramine-treated OCD patients. *J Clin Psychopharmacol* 1992;12:11-18
31. Grady TA, Pigott TA, L'Heureux F, et al. A double-blind study of adjuvant buspirone hydrochloride in fluoxetine-treated patients with OCD. *Am J Psychiatry* 1993;150:819-821
32. McDougle C, Goodman W, Leckman J, et al. Limited therapeutic effect of the addition of buspirone in fluvoxamine-refractory OCD. *Am J Psychiatry* 1993;150:647-649
33. McDougle C, Price L, Goodman W, et al. A controlled trial of lithium augmentation in fluvoxamine-refractory obsessive-compulsive disorder: lack of efficacy. *J Clin Psychopharmacol* 1991;11:175-184
34. Pigott TA, Pato MT, L'Heureux F, et al. A controlled comparison of adjuvant lithium carbonate or thyroid hormone in clomipramine-treated OCD patients. *J Clin Psychopharmacol* 1991;11:242-248
35. Leonard H, Topol D, Bukstein O, et al. Clonazepam as an augmenting agent in the treatment of childhood-onset obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 1994;33:792-794
36. McDougle CJ, Goodman WK, Leckman JF, et al. Haloperidol addition in fluvoxamine-refractory obsessive-compulsive disorder. *Arch Gen Psychiatry* 1994;51:302-308
37. Saxena S, Wang D, Bystritsky A, et al. Risperidone augmentation of SRI treatment for refractory obsessive-compulsive disorder. *J Clin Psychiatry* 1996;57:303-306
38. Baer L, Ricciardi J, Keuthen N, et al. Discontinuing obsessive-compulsive disorder medication with behavior therapy [letter]. *Am J Psychiatry* 1994; 151:1842-1843
39. March JS, Frances A, Carpenter D, et al. Expert Consensus Treatment Guidelines for Obsessive-Compulsive Disorder: a guide for patients and families. *J Clin Psychiatry* 1997;58(suppl 4):65-72