# A Review of Pharmacologic Treatments for Obsessive-Compulsive Disorder

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***Objective:*** **Obsessive-compulsive disorder is a chronic and often disabling disorder that affects 2 to 3 percent of the U.S. population. Optimal treatment involves a combination of pharmacologic and cognitive-behavioral therapies. Advances in psychopharmacology have led to safe and effective treatments for obsessive-compulsive disorder that provide clinically significant improvement in symptoms. In this article the authors review studies of pharmacologic treatments.** ***Methods:*** **A MEDLINE search was conducted to identify relevant articles from 1991 to 2002. Double-blind, placebo-controlled studies as well as open-label studies and case reports were included.** ***Results and discussion:*** **The serotonin reuptake inhibitors (SRIs), including clomipramine, fluvoxamine, fluoxetine, sertraline, and paroxetine, have been approved by the U.S. Food and Drug Administration for the treatment of adults with obsessive-compulsive disorder; three of these (clomipramine, fluvoxamine, and sertraline) have been approved for treatment of children and adolescents. Clomipramine and the selective serotonin reuptake inhibitors (SSRIs) are first-line agents. However, 40 to 60 percent of patients with obsessive-compulsive disorder do not respond to adequate treatment trials with SRIs, and agents that alter serotonin receptors and other neurotransmitter systems, such as dopamine, norepinephrine, and second-messenger systems, may play a role in treatment. Treatment options for patients who do not respond to SRIs include switching, augmentation, or novel-agent strategies. Up to two-thirds of patients with obsessive-compulsive disorder have comorbid psychiatric disorders, which may present a challenge in pharmacologic treatment. Major depressive disorder is the most common comorbid condition. Nonpharmacologic invasive techniques may play a role in refractory cases of obsessive-compulsive disorder, but further research is warranted. (*Psychiatric Services* 54:1111–1118, 2003)**

Obsessive-compulsive disorder is a chronic and often disabling disorder that affects 2 to 3 percent of the U.S. population (1). One aspect of diagnosing obsessive-compulsive disorder centers on the marked distress caused by obsessions or compulsions as well as significant interference in social or occupational functioning (2).

Obsessive-compulsive disorder has a major impact on quality of life in several domains, including social functioning, employment, marriage and family relationships, and socioeconomic status (3–6). The more severe the symptoms, the poorer the quality of life in the domain of social functioning (4). More than half the patients in one study reported that obsessive-compulsive disorder interfered with work and social functioning, and 70 percent reported that the disorder caused problems in family relationships (5). Family members of persons with obsessive-compulsive disorder are often affected by the patient's symptoms (6). Many family members are forced to become enmeshed with the patient's obsessions or compulsions, such as by having to provide repeated reassurance or to take part in avoidance or checking rituals, which can be cumbersome and debilitating. Persons with obsessive-compulsive disorder have also demonstrated elevated levels of unemployment and receipt of disability payments (7).

Thus it is evident that impairment due to obsessive-compulsive disorder is widespread and affects all aspects of the individual's daily life. One study showed that the social impairment of persons with severe obsessive-compulsive disorder was equal to that of patients with schizophrenia (8).

## Methods

A MEDLINE search was conducted to identify relevant articles from 1991 to 2002. Double-blind, placebo-controlled studies as well as open-label studies and case reports were included.

## Results and discussion

Optimal treatment for obsessive-compulsive disorder involves a combination of both pharmacologic and cognitive-behavioral therapies. Although the cognitive-behavioral techniques of exposure and response prevention are effective in the treatment of obsessive-compulsive disorder, in this article we focus only on pharmacologic treatments.

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Obsessive-compulsive disorder is frequently associated with embarrassment and shame. As a result, often patients do not seek necessary treatment, which is unfortunate, because advances in psychopharmacology have led to safe and effective treatments that provide clinically significant improvement in symptoms. Manipulation of the serotonergic system is associated with improvement in obsessive-compulsive symptoms. At the neurochemical level, serotonin is transported back into the neuron through transporter proteins. Serotonin reuptake inhibitors (SRIs) work by blocking these transporter proteins, which results in a greater availability of serotonin neurotransmitter in the synaptic cleft. SRIs are first-line agents in the treatment of obsessive-compulsive disorder.

However, 40 to 60 percent of patients with obsessive-compulsive disorder do not respond to adequate treatment trials with SRIs (9), and agents that alter serotonin receptors and other neurotransmitter systems, such as dopamine and norepinephrine, as well as second-messenger systems may play a role in the treatment of obsessive-compulsive disorder. Treatment options for patients who do not respond to SRIs include switching, augmentation, and novel-agent strategies. Although patients who respond may experience clinically significant improvement in their obsessions and compulsions, they are not cured of the illness and may still have residual symptoms. Because obsessive-compulsive disorder is highly comorbid with other psychiatric disorders, pharmacotherapy is aimed at targeting associated conditions as well.

The treatment of obsessive-compulsive disorder with SRIs differs from that of depression, because higher dosages and longer treatment trials (ten to 12 weeks) are often required for a full effect. The SSRIs have a better-tolerated side-effect profile for most patients than clomipramine, a tricyclic antidepressant, because they do not produce antihistaminergic, anticholinergic, or antiadrenergic effects and are associated with a lower risk of cardiac toxicity. A review of SRI treatment studies suggested that 65 to 70 percent of patients with obsessive-compulsive disorder respond at least moderately to first-time SRI treatment (10).

***Editor's Note:*** This paper is part of a series of psychopharmacology updates edited by Ewald Horwath, M.D. Contributions are invited to address the psychopharmacological treatment of psychiatric disorders. Papers should focus on integrating new treatments and on issues faced in the public sector, such as psychiatric and medical comorbidity, severe and persistent illness, and weighing of risks versus benefits of medications. For more information, please contact Dr. Horwath at the New York State Psychiatric Institute, 1051 Riverside Drive, Unit 88, New York, New York 10032 (e-mail, eh10@columbia.edu).

Currently, five SRIs, including clomipramine, fluvoxamine, fluoxetine, sertraline, and paroxetine, have been approved by the U.S. Food and Drug Administration for the treatment of adults with obsessive-compulsive disorder; three of these—clomipramine, fluvoxamine, and sertraline—have been approved for treatment of children and adolescents with the disorder. Evidence is accumulating on the efficacy of citalopram (11,12) and venlafaxine (13–15) in the treatment of obsessive-compulsive disorder, although these agents do not have an FDA indication for this disorder.

### *Clomipramine*

Clomipramine not only works as an SRI but also blocks the reuptake of norepinephrine and dopamine. Multiple controlled studies have demonstrated clomipramine's efficacy in the treatment of obsessive-compulsive disorder (16–19). The first multicenter, randomized, placebo-controlled trial of clomipramine for obsessive-compulsive disorder was published in 1991. In two trials among patients with obsessive-compulsive disorder, scores on the Yale-Brown Obsessive-Compulsive Scale (YBOCS) decreased by an average of 38 percent and 44 percent with clomipramine, compared with 3 percent and 5 percent with placebo (18).

### *Selective serotonin reuptake inhibitors (SSRIs)*

SSRIs may have a better side-effect profile than tricyclic agents. However, SSRIs are not well tolerated by all patients and vary in their side-effect profiles and half-lives. Fluoxetine has the longest half-life—two to four days—and its active metabolite, norfluoxetine, has a half-life of seven to nine days. Clinically, fluoxetine may have an activating profile. Therefore, gradual dosage titration is needed to avoid insomnia and restlessness or potential exacerbation of anxiety early in treatment. Early open trials showed efficacy in the treatment of obsessive-compulsive disorder (20). In addition, one multicenter, double-blind, placebo-controlled study found clinically significant improvement at fluoxetine dosages of 20, 40, and 60 mg daily (21). In another double-blind placebo-controlled study with fluoxetine, dosages of 40 and 60 mg (but not 20 mg) were superior in efficacy to a placebo (22).

Fluvoxamine was shown to have efficacy in several double-blind, placebo-controlled studies (23–25). In one multisite study, fluvoxamine at a daily dosage of 100 to 300 mg was found to be superior to placebo, and 43 percent of patients who were treated with fluvoxamine responded after six weeks, compared with 12 percent of those who received placebo (26). Fluvoxamine was also shown in an eight-week trial to be significantly more effective than desipramine, mainly a noradrenergic reuptake inhibitor, demonstrating the selective efficacy of SSRIs in the treatment of obsessive-compulsive disorder (24). A controlled-release formulation of fluvoxamine has shown promising efficacy in the treatment of obsessive-compulsive disorder, with significantly superior efficacy to that of placebo as early as week 2 (27).

Sertraline has shown efficacy in the treatment of obsessive-compulsive dis-

order despite negative results in an early study (28). In an eight-week double-blind, controlled trial at daily dosages of up to 200 mg, sertraline was found to be more effective than placebo (29). A multicenter, 12-week, placebo-controlled, double-blind trial of sertraline at three fixed daily dosages—50, 100, and 200 mg—showed that the 50 and 200 mg dosages were more effective than placebo, but not the 100 mg dosage (30).

Furthermore, the ability of sertraline to maintain improvement was demonstrated in a double-blind, placebo-controlled study in which the patients described above who responded to treatment were assigned to a double-blind, fixed-dose trial for an additional 40 weeks. At the 52-week end point, mean scores on four primary outcome measures—the YBOCS, the Clinical Global Impression (CGI) severity-of-illness and improvement scales, and the National Institute of Mental Health Global Obsessive Compulsive Scale—showed significantly greater improvement ($p<.005$) in the sertraline group than in the placebo group (31).

In a recent 28-week double-blind trial of sertraline compared with placebo among patients who had achieved a sustained response during 52 weeks of single-blind therapy, sertraline had significantly better efficacy than placebo on two of three primary outcomes, including dropout due to relapse or insufficient clinical response (9 percent compared with 24 percent) and acute exacerbation of symptoms (12 percent compared with 35 percent) (32).

Paroxetine has also demonstrated effectiveness in the treatment of obsessive-compulsive disorder (33). In a recent multicenter, double-blind, placebo-controlled study of paroxetine among patients with obsessive-compulsive disorder, acute and long-term treatment and prevention of relapse were examined (33). For acute treatment—phase I (12 weeks)—paroxetine at daily dosages of 40 and 60 mg (but not 20 mg) was found to be clinically significant compared with placebo in the treatment of obsessive-compulsive disorder compared with placebo among 348 patients.

In phase II, 263 patients who had completed phase I underwent six months of treatment with flexibly dosed paroxetine in an open-label trial. In phase III, 105 patients who had responded to the open-label trial of paroxetine were randomly assigned to a six-month double-blind, fixed-dose, parallel trial with either paroxetine or placebo. During phase III, a greater proportion of placebo recipients than paroxetine-treated patients experienced relapse (59 percent and 38 percent, respectively) (33). As is the case with all SSRIs that have a short half-life, abrupt discontinuation of paroxetine may result in a discontinuation syndrome. Therefore, paroxetine dosages should be tapered gradually.

Citalopram does not have FDA approval for the treatment of obsessive-compulsive disorder. It is the most selective of the SSRIs, with less potential for drug interactions. In a recent 12-week, placebo-controlled, double-blind study of citalopram among 401 patients, all three daily dosages—20, 40, and 60 mg—were found to be statistically superior to placebo in the treatment of obsessive-compulsive disorder (11). In a ten-week single-blind study of 30 patients with obsessive-compulsive disorder who underwent randomized treatment with fluvoxamine, paroxetine, or citalopram, no significant differences were found between the three treatments (12).

### *Augmentation strategies*

Because not all patients respond to SRIs, one further treatment option is augmentation of an SRI with another agent that alters other neurotransmitter systems or different serotonin receptors. It is known that dopamine and serotonin have complex structural interactions in the brain. Furthermore, combinations of a dopamine antagonist and an SRI have been reported to be effective in the treatment of obsessive-compulsive disorder. Haloperidol was shown to be effective as an augmentation strategy for the treatment of patients with obsessive-compulsive disorder and comorbid tic-related disorders (34). In this double-blind, placebo-controlled study of patients with obsessive-compulsive disorder whose illness was refractory to fluvoxamine, haloperidol augmentation was found to be significantly more effective than that of placebo. All eight patients with a concurrent chronic tic-related disorder responded to ongoing treatment with fluvoxamine combined with haloperidol (34).

This finding suggests that a subtype of patients with obsessive-compulsive disorder who have comorbid tics may benefit from augmentation with a dopamine antagonist. Also, open case series have demonstrated the effectiveness of combinations of pimozide and SRIs among patients with obsessive-compulsive disorder with and without comorbid tic-related disorders (35). Case reports and open studies with combinations of risperidone and SRIs have shown effectiveness in the treatment of obsessive-compulsive disorder (36,37).

In the first double-blind, placebo-controlled trial of risperidone augmentation among patients with SRI-refractory obsessive-compulsive disorder, nine (50 percent) of the 18 patients who completed the risperidone trial responded, compared with none of the 18 patients in the placebo group (38). Also, no differences in response were noted between patients with obsessive-compulsive disorder with and without comorbid diagnoses of a chronic tic-related disorder or schizotypal personality disorder. In addition, more recent case studies have shown some benefit of augmentation of SRIs with olanzapine among patients with treatment-refractory obsessive-compulsive disorder (39,40). A double-blind, placebo-controlled olanzapine augmentation study documented efficacy as well (41).

The $5HT_{1A}$ agonist buspirone has been reported to effectively augment fluoxetine in open-label studies of obsessive-compulsive disorder (42,43) but not in a controlled study (44). In two double-blind augmentation trials of SRIs, the addition of buspirone was not found to be significantly better than placebo in reducing obsessive-compulsive symptoms (45,46). One open-label addition of D,l-fenfluramine, an indirect 5-HT agonist, to ongoing SRI treatment was associated with improvement in obsessive-compulsive symptoms among six of seven patients (47). Fenfluramine is no longer available on the U.S. market.

Lithium, which is thought to en-

hance presynaptic 5-HT release in the brain (48), was found to improve obsessive-compulsive symptoms when used to augment fluoxetine in an open-label trial (49) but not in a double-blind augmentation study among partial responders with obsessive-compulsive disorder who were receiving clomipramine (50). Two double-blind, placebo-controlled trials of lithium in addition to ongoing fluvoxamine treatment among nonresponders with obsessive-compulsive disorder have been conducted—a two-week study with 20 patients and a four-week study with ten patients (51). A small statistically significant reduction in obsessive-compulsive symptoms was reported in the two-week trial but not in the four-week trial.

Clonazepam, a benzodiazepine with serotonergic properties, has been found to be effective in the treatment of obsessive-compulsive disorder when used to augment SRIs in case series (52) and in a double-blind, placebo-controlled crossover trial with fluoxetine or clomipramine (53). Trazodone, a 5-$HT_2$-blocker with weak 5-HT reuptake properties, was reported to be effective when used to augment various SRIs in five case reports of refractory obsessive-compulsive disorder (54). Tryptophan, a 5-HT precursor, has shown varying degrees of effectiveness in case reports of SRI augmentation in the treatment of obsessive-compulsive disorder (55).

*Switching antidepressants*

An alternative to augmentation strategies involves switching to another SSRI, to clomipramine, or to the serotonin norepinephrine reuptake inhibitor (SNRI) venlafaxine. Although meta-analyses have suggested that clomipramine has a more potent effect than SSRIs in treating obsessive-compulsive disorder (56–58), the studies on which these analyses were based were not head-to-head comparisons, and the patient samples differed in their severity of illness.

In head-to-head trials, no individual SRI has been shown to be superior to another (59,60). Side effects of clomipramine may include dry mouth and urinary retention and constipation (anticholinergic blockade), sedation and weight gain (antihistaminergic blockade), and orthostatic hypotension (alpha adrenergic blockade), and there is also potential for prolongation of the QT interval and seizures. Only about 25 percent of patients who fail to respond to one SRI will respond to a second SRI trial (61). Venlafaxine is unique in that it acts mostly as an SSRI at dosages below 225 mg daily and begins to have substantial noradrenergic effects at 225 mg daily, as an SNRI. Venlafaxine has been shown to be effective in case reports of patients with obsessive-compulsive disorder (13,14). In a recent 12-week single-blind study of venlafaxine compared with clomipramine for acute treatment of obsessive-compulsive disorder, no statistically significant difference was found between the two drugs (15).

*For some patients, obsessive-compulsive disorder remains refractory to treatment even after switching or augmentation trials. For these patients, alternative novel pharmacotherapy may provide relief.*

In a second 12-week single-blind study of venlafaxine versus clomipramine versus citalopram among patients who were unresponsive to at least two trials of SSRIs other than citalopram, 14 percent of patients responded to citalopram, 42 percent to venlafaxine, and 37.5 percent to clomipramine (15). These findings suggest that patients with obsessive-compulsive disorder who fail to respond to two SSRI trials might benefit from being switched to an agent that has a different mechanism of action, such as clomipramine or venlafaxine (62).

*Novel treatment strategies*

For some patients, obsessive-compulsive disorder remains refractory to treatment even after switching or augmentation trials. For these patients, alternative novel pharmacotherapy may provide relief from obsessions and compulsions. Intravenous clomipramine has been reported to be successful in the treatment of obsessive-compulsive disorder (63). In a randomized, double-blind, placebo-controlled trial of intravenous versus oral pulse loading of clomipramine among 15 patients with obsessive-compulsive disorder, six out of seven patients who were treated with intravenous clomipramine responded after 4.5 days from the second pulse, but only one of eight patients responded to oral clomipramine, indicating greater immediate improvement (63).

Monoamine oxidase inhibitors have shown some efficacy in case reports of refractory obsessive-compulsive disorder (64). In a placebo-controlled trial of fluoxetine and phenelzine among 54 patients with obsessive-compulsive disorder, the patients who were treated with fluoxetine improved significantly more than the patients who received phenelzine or placebo, except for a subgroup of patients with symmetry obsessions who responded to phenelzine (65). Trazodone demonstrated efficacy in reducing obsessive-compulsive symptoms in case reports and open studies (66) but not in a double-blind, placebo-controlled trial (67). In addition, buspirone has shown varying effectiveness in treating patients with obsessive-compulsive disorder (68,69).

Clonidine, an $\alpha_2$ agonist, has been shown to be effective in both oral (70) and intravenous (71) form in case reports but not in a double-blind, controlled crossover trial of clomip-

ramine, clonazepam, and clonidine among 28 patients with obsessive-compulsive disorder (72). Clonazepam has also shown efficacy in case reports (73,74). Other benzodiazepines, such as alprazolam, have not been found to be effective in the treatment of obsessive-compulsive disorder (75).

Agents that affect autoimmune mechanisms, steroids, and peptide hormones may play a role in the treatment of obsessive-compulsive disorder. The sudden onset of obsessive-compulsive symptoms after infection by group A B-hemolytic streptococci has been reported among children. Plasmapheresis and intravenous immunoglobulin have been reported to be effective in case studies of such patients (76,77). Among 30 children with infection-triggered exacerbations of obsessive-compulsive disorder or tic-related disorders who received intravenous immunoglobulin, plasma exchange, or placebo, both the intravenous immunoglobulin group and the plasma-exchange group demonstrated significant improvement at one month (78). An open trial with flutamide, an androgen receptor antagonist, among eight patients with obsessive-compulsive disorder demonstrated a lack of response (79).

Medications that affect second-messenger systems may also be effective in the treatment of patients with obsessive-compulsive disorder. In a six-week double-blind, controlled crossover trial of inositol compared with placebo among 13 patients with obsessive-compulsive disorder, YBOCS scores were significantly lower when the patients were receiving inositol than when they were receiving placebo (80).

*Comorbid conditions*

Up to two-thirds of patients with obsessive-compulsive disorder have comorbid psychiatric disorders (81,82), which may present a challenge in pharmacologic treatment. Major depressive disorder is cited as the most common comorbid condition among patients with obsessive-compulsive disorder—up to 55 percent of patients in one study (83). SSRIs may target both conditions, with clinical improvement seen earlier for depressive symptoms (four to six weeks) and later for obsessive-compulsive symptoms (up to 12 weeks).

The co-occurrence of obsessive-compulsive disorder and bipolar disorder may be as high as 30 percent (84,85). This rate of comorbidity presents the biggest clinical challenge, because SRIs, which are often optimal for treating obsessive-compulsive disorder, may convert predisposed patients into manic states. Thus it is important to first initiate a mood stabilizer, such as lithium, valproate, or carbamazepine, and then exercise caution when using SRIs.

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The prevalence of obsessive-compulsive symptoms among patients with schizophrenia has been estimated to range from 7.8 percent to 46.6 percent (86–88). Of interest, in one study, patients with both schizophrenia and obsessive-compulsive disorder showed greater functional impairment with worsened clinical course and treatment response than matched patients with schizophrenia who did not have obsessive-compulsive disorder (89). The possibility that there exists a "schizo-obsessive" subtype of schizophrenia (90) or that there is an overlap in pathology of the comorbid conditions has been discussed. There is evidence of emergence or worsening of obsessive-compulsive symptoms among some patients with schizophrenia after they begin the atypical neuroleptics clozapine (91,92), olanzapine (93), and risperidone (94). This result may be due to compensatory upregulation of postsynaptic serotonin receptors. The addition of an SRI to the atypical agent may improve obsessive-compulsive symptoms in this subgroup (95,96).

Comorbid anxiety disorders are prevalent among patients with obsessive-compulsive disorder. In a two-year prospective study, the rate of disorders comorbid with obsessive-compulsive disorder was high—23 percent for social phobia, 21 percent for simple phobia, and 20 percent for generalized anxiety disorder (97). Among 100 study participants with primary obsessive-compulsive disorder, high lifetime rates of social phobia (18 percent), panic disorder (12 percent), and specific phobia (22 percent) were reported (98). SRIs are the pharmacologic treatment of choice for patients with comorbid panic disorder, social phobia, generalized anxiety disorder, and specific phobias.

However, some patients with panic disorder may be sensitive to the activation of SRIs and may initially experience worsening symptoms. Therefore, other treatment options include use of a benzodiazepine, such as clonazepam, in addition to the SRI or use of a monoamine oxidase inhibitor. Although these agents come with the need for dietary restrictions and have the potential for serious side effects, such as hypertensive crises, they are a treatment option for treatment-refractory obsessive-compulsive disorder with comorbid panic disorder.

*Nonpharmacologic strategies*

Other biological approaches for obsessive-compulsive disorder include neurosurgery, deep-brain stimulation, electroconvulsive therapy, and repetitive transcranial magnetic stimulation. The neurosurgical techniques of cingulotomy and capsulotomy may provide clinical improvement among some patients with treatment-refractory obsessive-compulsive disorder. In

one prospective study of 18 patients with obsessive-compulsive disorder who were evaluated before and six months after bilateral cingulotomy, five patients (28 percent) met the criteria for treatment response (99).

Overall, stereotactic surgery should be viewed as a last option in treating refractory obsessive-compulsive disorder, and certain criteria, including several failed treatment trials with cognitive-behavioral therapy, must be met. Deep-brain stimulation, involving insertion of a pacemaker electrode into similar brain regions, may hold promise and does not cause lesions in brain tissue, but this approach requires further study. In one controlled study, repetitive transcranial magnetic stimulation, when applied to the right prefrontal cortex, was shown to produce a transient reduction in compulsive urges (100). Electroconvulsive therapy has not shown much benefit among patients with treatment-refractory obsessive-compulsive disorder. Further research in all these areas is required.

## Conclusions

Obsessive-compulsive disorder is a common and disabling disorder but it often responds to appropriate treatment in the form of cognitive-behavioral therapy and pharmacotherapy. The SSRIs and the tricyclic antidepressant clomipramine are first-line pharmacotherapeutic treatments for patients with obsessive-compulsive disorder. SSRIs have a better-tolerated side-effect profile than clomipramine for many patients. Because 40 to 60 percent of patients with obsessive-compulsive disorder do not respond to an SRI, augmentation with agents that affect serotonin and other neurotransmitter systems may play a role in treatment among nonresponders with obsessive-compulsive disorder.

Of importance, a subtype of patients with obsessive-compulsive disorder who have comorbid tic-related disorders may preferentially benefit from augmentation with a dopamine antagonist. Switching from an SSRI to an SNRI, such as clomipramine or venlafaxine, may also be effective. Novel treatments with agents that target autoimmune mechanisms, second-messenger systems, and steroid-peptide hormones may be effective in some cases.

Comorbid psychiatric disorders are highly prevalent among persons with obsessive-compulsive disorder. One subtype of patients with schizophrenia may experience exacerbation of obsessive-compulsive symptoms after treatment with atypical neuroleptics. In these cases, the addition of an SRI may be helpful. Treatment should be ultimately aimed at targeting all core and associated symptoms among patients with obsessive-compulsive disorder. Finally, nonpharmacologic invasive techniques may play a role in refractory cases of obsessive-compulsive disorder, but further research is warranted. ♦


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## First-Person Accounts Invited for Column

Patients, former patients, family members, and mental health professionals are invited to submit first-person accounts of experiences with mental illness and treatment for the Personal Accounts column of *Psychiatric Services.* Maximum length is 1,600 words. The column appears every two months.

Material to be considered for publication should be sent to the column editor, Jeffrey L. Geller, M.D., M.P.H., at the Department of Psychiatry, University of Massachusetts Medical School, 55 Lake Avenue North, Worcester, Massachusetts 01655. Authors may publish under a pseudonym if they wish.