Little more than a decade ago, virtually nothing was known about how to treat BDD with medication. In the early 1990s, the pharmacotherapy literature was limited to case reports and a case series of five patients.\textsuperscript{1,2} Even now, relatively few treatment studies have been done, but our knowledge has grown dramatically, and the findings are remarkably consistent. They indicate that BDD often responds to pharmacotherapy and that serotonin-reuptake inhibitors (SRIs) appear preferentially efficacious for this often-disabling illness.

This article offers practical suggestions on how to successfully treat BDD with pharmacotherapy. More detailed reviews of the empirical literature on this growing topic may be found elsewhere.\textsuperscript{3,4} It should be noted that no medications are approved by the Food and Drug Administration specifically for the treatment of BDD.

The following suggestions may need to be modified to appropriately treat an individual patient. For example, the presence of certain comorbid disorders may require additional

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treatment, such as a concomitant mood stabilizer for bipolar disorder. For certain patients with substance abuse or dependence, it may be unwise to use a benzodiazepine or a stimulant.

RECOGNIZING BDD

While this point may seem self-evident, numerous studies have found that BDD usually goes unrecognized in clinical practice. In particular, psychiatrists providing pharmacotherapy often are unaware that the patient they are treating has BDD. In an ongoing study in which 110 patients with BDD had received psychotrophic medication, only 51% had revealed their BDD symptoms to the pharmacotherapist. Because BDD often requires different treatment from depression or anxiety disorders, it is important to inquire about BDD symptoms so they can be targeted specifically.

It is also important to educate the patient about BDD and the potential benefits of medication. Websites and books for the layperson may be helpful, especially because these patients may be reluctant to accept psychiatric care. Their insight typically is poor, and many desire nonpsychiatric treatment such as surgery or dermatologic treatment. Many patients are relieved to learn they have a bona fide and often treatable condition.

SRI TREATMENT FOR BDD

The SSRIs — clomipramine and the selective serotonin reuptake inhibitors (SSRIs) — are the best-studied medications in BDD. Several early case reports suggested efficacy for fluoxetine and clomipramine in some cases after patients had failed to respond to many other medications. Reports from subsequent large clinical series, open-label studies, and controlled studies consistently have indicated that SSRIs often are efficacious for BDD and that they are probably more efficacious than other types of medication.

Two controlled pharmacotherapy studies have been completed. In a double-blind crossover trial of clomipramine versus desipramine conducted by Hollander and colleagues, 29 patients were randomized to 8 weeks of treatment with each medication. Clomipramine was more efficacious than desipramine for BDD symptoms and functional disability. Treatment efficacy was independent of the presence or severity of comorbid obsessive-compulsive disorder (OCD), depression, or social phobia. This study is important because it suggests BDD should be treated with an SRI rather than a non-SRI antidepressant.

In the only placebo-controlled study, 67 patients were randomized to 12 weeks of treatment with fluoxetine versus placebo in a double-blind, parallel-group trial. Fluoxetine was significantly more efficacious than placebo for BDD symptoms and functional disability. 53% responded to fluoxetine, while only 18% responded to placebo. As was found in the clomipramine versus desipramine study, treatment efficacy was independent of the presence of major depression or OCD.

Four systematic open-label SRI studies have been conducted, two with fluvoxamine, one with citalopram, and one with escitalopram. In a 16-week study of 30 patients with BDD, 19 (63%) responded to fluvoxamine. BDD severity significantly decreased between study baseline and termination. A 10-week, open-label study of fluvoxamine found 10 of 12 patients who completed the study were responders. In a recent open-label citalopram study, 73% of 15 patients improved. The same proportion of patients improved in a recently completed open-label trial of escitalopram (unpublished data, 2004).

While all of these studies have consistently found a majority of patients improve with SRI treatment, in most cases, response is only partial. In the fluoxetine study, 15% of patients were very much improved on the Clinical Global Impressions Scale (CGI), and in one of the fluvoxamine studies, this was the case for 30% of the participants. The proportion of patients who were very much improved was somewhat higher in the studies of citalopram (40%) and escitalopram (45%) (unpublished data, 2004). These findings suggest citalopram and escitalopram may produce a greater magnitude of response than other SSRIs, but this cannot be concluded definitively because of the small sample sizes and lack of head-to-head comparisons of these medications.

Other SSRIs have not been studied systematically, nor have any SSRIs been directly compared with one another in a methodologically rigorous study. However, in a chart-review study of 90 patients treated in clinical practice during a mean duration of 1.8 ± 1.8 years (range = 0.1 to 8.4 years), response rates were similar for each type of SRI (although these data did not include citalopram or escitalopram). Overall, 63% (n = 55) of adequate SRI trials led to improvement (much or very much improved on the CGI). To the author's knowledge, this is the only pharmacotherapy study that has examined remission; 18% (n = 10) of adequate SRI trials resulted in full remission and 32% (n = 18) in partial remission.

Response to an SRI in a patient with BDD usually includes decreases in preoccupation with the perceived defect, better ability to resist and control BDD-related thoughts and behaviors (eg, mirror checking and excessive grooming), and decreased distress secondary to BDD. Mood and suicidal thinking usually improve, as does functioning.

Several other medications with prominent effects on serotonin may also be efficacious for BDD, although they
have received much less investigation than the SRIs. In an unpublished open-label study of 11 patients, venlafaxine significantly improved BDD symptoms in study completers. This preliminary finding is consistent with the author’s clinical experience that BDD may respond to this medication. Monoamine oxidase inhibitors, too, may have a role in treating BDD, as discussed below. However, it seems prudent to reserve them for very treatment-refractory cases, because there are far less data supporting their efficacy than for SRIs and because of their potentially problematic side-effects.

**SRI TREATMENT FOR DELUSIONAL BDD**

The appearance beliefs of about half of all patients with BDD are delusional (i.e., they are completely convinced that their view of the "defect" is accurate and cannot be convinced otherwise). The *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV), classifies BDD’s delusional variant as a psychotic disorder — delusional disorder, somatic type. The delusional variant may be double coded with BDD (i.e., delusional patients may receive diagnoses of both BDD and delusional disorder), reflecting the likelihood that BDD’s delusional and non-delusional variants are actually one and the same disorder, characterized by a range of insight.

It may sound counterintuitive to treat delusional BDD with an SRI, because delusional symptoms in disorders other than BDD typically are treated with antipsychotics. However, every study that has examined this issue has had the same finding: BDD symptoms of delusional patients are as likely as those of non-delusional patients to respond to an SRI. In the previously noted desipramine versus clomipramine study, clomipramine was more effective than desipramine, regardless of whether patients had insight or held their dysmorphic misperception with delusional intensity. In fact, clomipramine was even more effective for delusional patients than for non-delusional patients. In the previously noted placebo-controlled fluoxetine study, a similar percentage of delusional and non-delusional patients experienced response of BDD symptoms to fluoxetine (50% and 55%, respectively). In contrast, antipsychotics as monotherapy do not appear efficacious for delusional BDD, although data are very limited (see discussion below).

Several studies have examined the different question of whether delusional insight (insight) improves with treatment. In other words, do treated patients develop more accurate beliefs about their appearance, realizing that they do not look abnormal? In an open-label fluvoxamine study, insight improved significantly, and in the blinded crossover study, clomipramine (but not desipramine) significantly improved insight. In the fluoxetine study, insight did not improve more with fluoxetine than with placebo, although insight improved significantly more in treatment responders (to either fluoxetine or placebo) than in treatment nonresponders.

**OTHER MEDICATIONS FOR BDD**

The above-noted clomipramine versus desipramine study is the most methodologically rigorous study to examine whether non-SRIs are efficacious for BDD. That study found that desipramine was inferior to clomipramine for both BDD and depressive symptoms. Other data, while limited, are consistent with this finding. In a retrospective study of 50 patients by Hollander and colleagues, 35 SRI trials resulted in improved BDD symptoms, whereas 18 non-SRI tricyclic trials led to no overall improvement.

Numerous studies have found that BDD usually goes unrecognized in clinical practice. In particular, psychiatrists providing pharmacotherapy often are unaware that the patient they are treating has BDD. It is important to inquire about BDD symptoms so that they can be targeted specifically.
Other data, while limited, similarly suggest non-SRIs are not efficacious for BDD as monotherapy, although some may have a role as SRI augmentation agents (as discussed below). In a retrospectively assessed series of 30 patients with BDD, 58% responded to an SRI, whereas only 5% responded to other medications. In an expansion of this series, consisting of 130 patients who had received a total of 316 medication trials, 42% of 65 SRI trials led to improvement on the CGI, whereas improvement followed 30% of MAOIs trials, 15% of trials with non-SRI tricyclics, 3% of neuroleptic trials, 6% of trials with a variety of other medications (eg, benzodiazepines and mood stabilizers), and 0% of ECT trials. In this series the SRI response rate was lower than in subsequent studies, probably because at this early stage of BDD research, it was not known what an adequate SRI trial consisted of. Of note, about half of the patients in this series had delusional BDD, but neuroleptics were efficacious in only one of 49 trials.

Pimozide has long been reputed to be effective for monosymptomatic hypochondriacal psychosis, a broad category currently known as delusional disorder, somatic type, which includes delusional BDD. Data from a small number of case reports suggested that pimozide may be effective for BDD, although pimozide alone has been found ineffective in a small number of cases (*n* = 8). Pimozide also appears ineffective as an SRI augmenting agent (see discussion below). However, this medication warrants further and more rigorous investigation. A recent case report, in contrast, noted improvement in BDD symptoms with olanzapine monotherapy.

**HIGHER SRI DOSES**

Fixed-dose studies have not been conducted; however, BDD often appears to require higher SRI doses than those typically used for depression. The following mean doses have been used in clinical practice:

- Escitalopram, 29 ±11.9 mg per day.
- Citalopram, 66 ±35.9 mg per day.
- Fluoxetine: 66.7 ±23.5 mg per day.
- Fluvoxamine: 308.3 ±49.2 mg per day.
- Paroxetine: 55.0 ±12.9 mg per day.
- Sertraline: 202.1 ±45.8 mg per day.
- Clomipramine: 203.3 ±52.5 mg per day.

Some patients clearly benefit from SSRI doses that exceed the maximum recommended dose — for example, mg per day of escitalopram, mg per day of citalopram, mg per day of fluvoxamine, or mg per day of paroxetine. These higher doses are best suited to patients who have failed many SSRIs or those who have responded partially to the highest recommended dose and are tolerating the medication well. Clomipramine doses, however, should not exceed mg per day because of the risk of seizure.

How rapidly the dose is raised will depend on a number of factors, including illness severity, with quicker titration generally advisable for sicker patients, especially those who are suicidal. Other considerations include medication tolerability and patient preference. Generally, however, it is advisable to reach the maximum recommended medication dose by week 5 to 9 of treatment, if tolerated.

**SLOW SRI RESPONSE**

Most studies have found a mean time to BDD response of 6 to 9 weeks and as long as 16 weeks. However, recent studies of citalopram and escitalopram found a quicker onset of action, with a mean time to response of only 4.6 ±2.6 weeks and 4.7 ±3.7 weeks (unpublished data, 2004), respectively. Most studies used a fairly rapid titration schedule, so an even longer time to response might be expected with a slower dose increase. If an adequate...

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dose has been reached (the highest dose that is tolerated or recommended by the manufacturer) and the response is inadequate after 12 to 16 weeks, it is recommended that the medication be changed at that time.

LENGTH OF TREATMENT

No studies have examined the important question of how long to continue an efficacious SRI. To the author’s knowledge, the only data on continuation treatment come from a chart-review study from clinical practice. This study found that, among patients who had responded to an acute SRI trial and then continued the efficacious SRI for 6 more months, only 8% (n = 2) relapsed during the 6 months. Thirty-nine percent (n = 9) of subjects who continued the SRI for 6 months had further improvement in BDD symptoms.

The recommendation to continue an effective SRI for at least 1 year is somewhat arbitrary, because the optimal treatment duration has not been studied systematically. It seems wise to tailor treatment duration to the individual patient. Patients who have had several previous relapses when discontinuing medication may need long-term pharmacotherapy. For severely ill patients (e.g., those who have made numerous, potentially lethal suicide attempts due to BDD), it is probably best to continue the medication for life.

DISCONTINUING AN SRI

Methodologically rigorous medication discontinuation studies have not been conducted in BDD. However, a chart review study from clinical practice found that, among patients who responded to an acute trial of an SRI but discontinued it, 87% (n = 20) relapsed within 6 months, compared with a relapse rate of 8% in those who continued the SRI for 6 months. Relapse occurred significantly earlier in the SRI discontinuation group than in the SRI continuation group. The median time to relapse in the SRI discontinuation group was approximately 36 days.

If a decision is made to discontinue effective medication, it seems prudent to slowly taper it (e.g., over months) rather than suddenly discontinuing it. Because the relapse risk appears high, the medication ideally should be discontinued at a time when the patient is not highly stressed and can tolerate a relapse if it occurs. Reinitiating treatment with a previously effective medication usually will improve symptoms again, although occasionally patients do not have a robust response as they did initially.

SWITCHING SRIs

All SRIs appear effective, but one may be more effective than another for an individual patient. If one SRI fails, another should be tried, as some patients will respond. In the only study to examine this question, 43% of patients who failed an initial adequate SRI trial responded to at least one subsequent adequate SRI trial. Among responders to an initial SRI who were subsequently treated with a different SRI, 92% of subsequent trials also led to improvement.

SRI AUGMENTATION

It is not known whether it is better to augment a partially effective SRI or switch to another SRI. The only study on this issue (a chart-review study) found that, in patients who had failed an adequate SRI trial, 44% of subsequent adequate SRI trials were effective versus 33% of various SRI augmentation strategies.

SRI augmentation may be more effective for patients who have partially responded to an SRI, rather than those who have had no SRI response. In the chart-review study, augmentation was more likely to be effective when the augmenting agent was added to a partially effective (as opposed to an ineffective) SRI, with response rates of 41% versus 18%. Augmentation also is more appealing for patients who have partially responded to an SRI in the sense that relapse—which could occur with a switch to another SRI—could be risky (e.g., leading to increased suicidality).

SRI augmentation strategies in BDD have received little investigation. The only placebo-controlled study (n = 28), which examined pimozide versus placebo augmentation of fluoxetine, found that pimozide was not more effective than placebo. The response rate to pimozide was 18.2% and to placebo was 17.6%. There was no significant effect of baseline delusional severity on endpoint BDD severity (i.e., more delusional patients did not have a better response). Furthermore, delusionality did not decrease (i.e., insight did not improve) significantly more with pimozide than with placebo. In the author’s clinical experience, atypical antipsychotics are more promising and may be particularly helpful in decreasing anxiety and agitation in severely ill patients.

Other sometimes-helpful SSRI augmentation choices are bupropine, generally at doses of 30 to 60 mg per day, and clomipramine, although lower doses of clomipramine generally must be used in combination with an SSRI, and clomipramine levels must be closely monitored. Occasionally, augmentation with lithium, methylphenidate, or bupropion is helpful. Another option is to combine an SSRI with venlafaxine if treatment with either one alone is not successful, while monitoring patients for the possible (although unlikely) emergence of serotonin syndrome. The
duration of an adequate augmentation trial is unclear, but 6 to 8 weeks is probably adequate (although it seems best to try clomipramine or venlafaxine augmentation for 12 weeks).

Adjunctive benzodiazepines should be considered for very distressed, anxious, or agitated patients. They may be used for a short duration (i.e., until an SRI begins to work) or for the longer term if clinically indicated. Benzodiazepines may enable treatment with an SRI if an SRI causes agitation. The potential for substance abuse or dependence must be considered, although in the author's clinical experience, relatively few patients abuse these medications.

ECT for BDD
There are only very limited data on electroconvulsive therapy (ECT). A 1991 review found seven published case reports, six of which reported an unsuccessful outcome with ECT, although one subsequent report noted a good response. In a series of 130 cases, none of eight ECT trials were successful, although the data were largely retrospective. There are approximately 10 additional cases of which the author is aware, in none of which ECT was effective for BDD, although several patients had a transient improvement, primarily in depressive symptoms.

SUMMARY
It must be emphasized that virtually all aspects of pharmacotherapy for BDD need to be studied, as research is still in its early stages. Needed studies include additional placebo-controlled SRI trials, studies that compare SRLs to other medication classes, augmentation studies, continuation and maintenance studies, relapse prevention studies, and effectiveness studies with broad inclusion criteria that examine medications in “real world” settings. Finally, elucidation of BDD’s underlying neurobiology, which has received very little investigation, may provide fruitful leads for new pharmacologic approaches.

REFERENCES