Bulimia nervosa
Persistent disorder requires
equally persistent treatment

Thousands of scientific papers have been written about bulimia, but not all patients receive effective treatments that produce remission.

To set the record straight and help psychiatrists avoid undertreating bulimia, this article discusses:

• evidence for using antidepressants, even when patients are not “depressed”

• merits of psychotherapies, including those shown to work and those that can harm

• augmentation therapies that can help increase response from partial to full remission.

INITIAL EVALUATION

Diagnosis. Bulimia nervosa is characterized by eating binges, followed by purging behaviors such as self-induced vomiting or laxative abuse (Table 1). It affects 1% to 3% of adolescent girls and young women and occurs in women 5 to 10 times more often than in men.

Bulimia is often persistent. About one-half of bulimic patients—including those who have been treated—continue to show eating disorder features on long-term follow-up.

Psychiatric comorbidity. Most bulimic patients
Bulimia nervosa

DSM-IV-TR diagnostic criteria for bulimia nervosa

A. Recurrent episodes of binge eating, characterized by both of the following:
   • eating, in a discrete period of time (as within any 2 hours), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances
   • a sense of lack of control over eating during the episode (a feeling that one cannot stop eating or control what or how much one is eating)

B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months

D. Self-evaluation is unduly influenced by body shape and weight

E. The disturbance does not occur exclusively during episodes of anorexia nervosa

Specify type:
  Purging type: during the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas
  Non-purging type: during the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas


Report a history of other psychiatric disorders, especially major depressive and bipolar disorders and anxiety disorders such as panic disorder, social phobia, and obsessive-compulsive disorder (OCD). Because these psychiatric comorbidities may occur before, during, or after bulimia nervosa, one cannot assume that mood or anxiety disorders are a cause or consequence of bulimia. Instead, bulimia nervosa, mood disorders, and anxiety disorders may be different expressions of a shared etiologic abnormality.

Evidence supporting this hypothesis comes from studies showing that these disorders:
   • respond to several chemically unrelated families of antidepressants
   • frequently co-occur in individual patients
   • frequently co-aggregate in families

We have published this evidence and proposed that bulimia nervosa may be one form of a larger underlying disorder, which we termed “affective spectrum disorder.” Antidepressants are often rapidly effective in treating bulimic symptoms, regardless of whether patients exhibit depressive symptoms. Thus, there is no reason to withhold antidepressant therapy simply because a bulimic patient is not depressed. The term “antidepressant” may be a misnomer; these drugs are effective for numerous conditions, of which depression is only one.

Anorexic symptoms. Co-occurring depressive or anxiety disorders in a bulimic patient will not greatly alter treatment. The antidepressants and psychotherapies typically used to treat bulimia are often equally effective for affective disorders. Co-occurring anorexia nervosa, however, is a more serious concern.

Bulimic patients often display a history of anorexia nervosa; in many cases, the patient develops anorexia nervosa as a teenager and then progresses to bulimia nervosa across several years. Her prognosis is much better if her weight normalizes with the shift to bulimia nervosa, than if her weight remains well below normal for her
height. It is unclear why medications and psychotherapy are much less effective in bulimic patients with anorexic symptoms than in those with bulimia alone. Watch for further details on anorexia nervosa as this series continues in future issues of CURRENT PSYCHIATRY.

Medical considerations. Potential medical complications—mostly consequences of vomiting or laxative use—are important to consider when you assess a bulimic patient:

- The acid in vomitus may gradually erode tooth enamel, requiring dental consultation.
- Vomitus may inflame salivary gland ducts, though the swelling is usually benign.
- Frequent vomiting may result in hypokalemia and alkalosis, although aggressive medical treatment usually is not needed.

Ask about ipecac use. To induce vomiting, some patients may abuse ipecac syrup, which can cause cardiomyopathy.

Inpatient or outpatient? Unless the bulimic patient displays severe and medically dangerous anorexic symptoms, she can usually be treated as an outpatient. However, evaluate her carefully for suicidal ideation—which is not uncommon in bulimia nervosa—and consider inpatient treatment if necessary.

MEDICATION VS. PSYCHOTHERAPY

The relative merits of medication versus psychotherapy in treating bulimia nervosa continue to be debated. The Cochrane Database of Systematic Reviews includes meta-analyses of both drug therapy and psychotherapy for bulimia nervosa. The 2001 drug therapy review found that “the use of a single antidepressant agent was clinically effective,” with no one drug clearly superior to another. Notably, this review was published before recent findings on topiramate (see page 19).

The corresponding 2002 review of psychotherapy concludes—somewhat more cautiously—that “there is a small body of evidence for the efficacy of cognitive-behavior therapy in bulimia nervosa and similar syndromes, but the quality of trials is very variable and sample sizes are often small.”

In bulimia nervosa and other psychiatric disorders, comparing psychotherapy with drug therapy is hazardous because several factors bias the comparison in favor of psychotherapy. These factors include an expectational effect, a responsibility effect, and differential generalizability of study results.

Expectational effect. Patients in clinical trials are aware that they are receiving psychotherapy and, presumably, that study investigators hope to demonstrate its efficacy. This might account for much of psychotherapy’s apparent effect, as even placebos can produce 30% to 50% improvement in bulimia.

Responsibility effect. If a patient fails to improve in a drug study, she will conclude that the drug has failed. But if she fails to improve in a psychotherapy study, she may conclude that she has failed. Because psychological treatments generally require patients to work in therapy, the patient may feel partially responsible for the outcome. Thus, to avoid cognitive dissonance, she may consciously or unconsciously exaggerate her improvement, both in her own mind and when reporting to treaters.

Differential generalizability. Psychological study protocols, such as administering several months of a behavioral treatment, usually mimic clinical practice fairly well. This is not the case with drug study protocols.

No responsible clinician would inflexibly administer a single dosage of a single drug for a fixed period to every bulimic patient and then declare failure for all nonresponders, as is done...
**Table 2**

How effective are medications in treating bulimia nervosa?

<table>
<thead>
<tr>
<th>Medication</th>
<th>Evidence for efficacy</th>
<th>Remarks</th>
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</thead>
<tbody>
<tr>
<td><strong>Antidepressants</strong></td>
<td></td>
<td></td>
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<tr>
<td>Selective serotonin reuptake inhibitors</td>
<td>+++</td>
<td>Fluoxetine is only SSRI studied in controlled trials</td>
</tr>
<tr>
<td>Tricyclics</td>
<td>+++</td>
<td>Generally more side effects than SSRIs</td>
</tr>
<tr>
<td>Monoamine oxidase inhibitors</td>
<td>++</td>
<td>High rates of remission, but dietary restrictions</td>
</tr>
<tr>
<td>Trazodone</td>
<td>++</td>
<td>Only one controlled trial</td>
</tr>
<tr>
<td>Venlafaxine, mirtazapine, nefazodone</td>
<td>?</td>
<td>No controlled trials, but probably effective</td>
</tr>
<tr>
<td>Bupropion</td>
<td>(+++)</td>
<td>Not recommended; caused seizures in bulimic patients</td>
</tr>
<tr>
<td><strong>Anticonvulsants</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Topiramate</td>
<td>++</td>
<td>Only one controlled trial, but substantial effect size</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>+</td>
<td>Little efficacy in only controlled study</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>+</td>
<td>May be useful in bulimia with comorbid bipolar disorder</td>
</tr>
<tr>
<td>Valproate</td>
<td>+</td>
<td>May be useful in bulimia with comorbid bipolar disorder</td>
</tr>
<tr>
<td><strong>Other agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liothyronine</td>
<td>+</td>
<td>Augmentation agent in patients with incomplete antidepressant response</td>
</tr>
<tr>
<td>Lithium</td>
<td>+</td>
<td>Ineffective in only controlled trial; possible augmentation strategy</td>
</tr>
<tr>
<td>Naltrexone</td>
<td>0</td>
<td>Ineffective in two controlled trials</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>+</td>
<td>One controlled trial</td>
</tr>
</tbody>
</table>

0 No apparent efficacy       
+ Occasional effect; limited evidence       
++ Clear effect; good evidence from controlled trial(s)       
+++ Strongly documented effect; evidence from multiple controlled trials.       
( ) Negative effect

in study protocols. In practice, the clinician can offer nonresponders augmentation strategies and additional drug trials. Thus, calculations of bulimia response rates in drug studies substantially underestimate response to drug therapy in clinical practice.

One also might note that psychological study findings have not “sold” well in the clinical practice marketplace. For example, in a recent survey of more than 220 bulimic women treated with psychotherapy, only 6.9% said they received a full course of cognitive behavioral therapy (CBT)\(^4\)—despite two decades of evidence of its efficacy. By contrast, untested, ineffectual, and possibly harmful psychotherapies for bulimia—including recovered-memory therapy—appear to be thriving.

**Recommendation.** Interpret with caution any head-to-head comparisons of psychological versus drug therapies—especially when clinical practice recommendations are made. Certain psychological therapies provided by specifically-trained individuals likely do help patients with bulimia nervosa. However, biases inherent to the studies may inflate psychological therapies’ efficacy when compared with that of drug therapy.

continued on page 19
Therefore, for a psychiatrist who does not specialize in eating disorders to offer exclusively psychological therapy to a bulimic patient—while withholding or postponing drug therapy—may now be a questionable practice.

**CHOOSING DRUG THERAPIES**

Although consensus is lacking on an optimal treatment trial sequence for bulimia nervosa, we suggest a rational approach based on the evidence and our experience (Algorithm, page 20).

**First-line antidepressants.** A selective serotonin reuptake inhibitor (SSRI) trial is usually the first choice (Table 2, page 16), and some data suggest that higher-than-usual dosages may be required. For example, in a large multicenter trial of fluoxetine in bulimia nervosa, 60 mg/d was considerably more effective than 20 mg/d for reducing binge eating behavior and vomiting frequency.15

Based on our observations, however, we believe that noncompliance or irregular compliance may account for this difference in response. Bulimic patients’ impulsive and obsessive behavior may keep them from taking their medications as prescribed. The higher fluoxetine dosage may therefore have been more effective simply because it ensured adequate plasma levels, even when patients missed or forgot multiple doses.

**Augmenting agents.** A first antidepressant trial rarely leads to complete remission of bulimic symptoms. This is not a serious concern, however, because many other options are available.

**Liothyronine.** Partial responders to SSRIs often become complete responders when we add a 10-day trial of liothyronine (T3), 25 µg/d. If this fails, we may try augmenting with lithium carbonate, although bulimic patients are often afraid of weight gain or lithium’s other side effects.

**Topiramate.** A newer augmentation strategy is to add the anticonvulsant topiramate. Used alone, topiramate demonstrated effectiveness for bulimia nervosa in one placebo-controlled, double-blind trial.16

Adding topiramate to an antidepressant regimen will likely reduce any remaining bulimic symptoms. In addition, topiramate often produces weight loss—a side effect that bulimic patients usually welcome. It remains unclear whether topiramate’s weight-loss effects might pose a hazard in patients with simultaneous bulimic and anorexic symptoms.

**Other antidepressants.** If the above strategies fail, other antidepressant options include venlafaxine, tricyclics, and monoamine oxidase inhibitors.

Bupropion is not recommended in bulimia nervosa; one trial of this agent resulted in a much higher rate of grand mal seizures in bulimic patients than in patients taking bupropion for depression.

In bulimic patients with concomitant bipolar disorder, the anticonvulsants carbamazepine and valproate often reduce affective and bulimic symptoms. By contrast, the anticonvulsant phenytoin—once thought to be useful in bulimia nervosa10—offers little benefit for either bulimic or affective symptoms.

Persistence is important when initial medication trials fail. One unblinded study followed 36 bulimic patients 9 to 19 months after they completed a controlled study with trazodone.18 Of the 26 patients who tried a second or third antidepressant, 17 (65%) achieved remission of bulimia on follow-up. Of the 10 patients who declined a second or third trial, only 1 (10%) attained remission.

Notably, these study results were obtained before the SSRIs and other newer antidepressants or topiramate became available. Cooperative patients using present-day medications might be able to achieve remission rates that exceed 65%.
Bulimia nervosa

Proposed treatment approach to bulimia nervosa

**Patient meets DSM-IV-TR diagnostic criteria for bulimia nervosa**

- **Initiate an SSRI, such as:**
  - Fluoxetine, 40-60 mg/d
  - Sertraline, 100-200 mg/d
  - Citalopram, 40-60 mg/d

  - **Remission**
  - **Inadequate response, or side effects limit dosage**

- **Continue treatment at least 6 months,** and monitor patient at least monthly

- **Consider tapering medication** if remission continues

  - **Remission**
  - **Inadequate response, or side effects limit dosage**

- **Consider switching to another SSRI**

  - **Remission**
  - **Inadequate response, or side effects limit dosage**

- **Consider augmentation with liothyronine,** 25 mcg/d

  - **Remission**
  - **Inadequate response**

  - **Consider augmentation with topiramate,** titrated to 100-300 mg qhs

- **Try (in the following order):**
  - Venlafaxine, 150-300 mg/d, or a tricyclic (eg, desipramine or nortriptyline), with dosage adjusted to therapeutic serum concentration
  - A monoamine oxidase inhibitor

  - **Remission**
  - **Inadequate response**

CBT: Cognitive-behavioral therapy
How effective are psychotherapies in treating bulimia nervosa?

<table>
<thead>
<tr>
<th>Psychotherapy</th>
<th>Evidence for efficacy</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive behavioral therapy (CBT)</td>
<td>+++</td>
<td>Controlled evidence for efficacy in individual and group treatment</td>
</tr>
<tr>
<td>Interpersonal psychotherapy (IPT)</td>
<td>++</td>
<td>Effective, but slower than CBT</td>
</tr>
<tr>
<td>Exposure with response prevention</td>
<td>+</td>
<td>May be added to other behavioral techniques, though additive benefit questionable</td>
</tr>
<tr>
<td>Dialectical behavior therapy</td>
<td>+</td>
<td>Highly structured behavioral technique originally developed for borderline personality disorder</td>
</tr>
<tr>
<td>Self-help groups</td>
<td>+</td>
<td>Frequently considered very helpful by patients</td>
</tr>
<tr>
<td>Psychodynamic psychotherapy</td>
<td>0</td>
<td>“Recovered memory” approaches are frankly harmful</td>
</tr>
<tr>
<td>Eye movement desensitization and reprocessing (EMDR)</td>
<td>0</td>
<td>Dubious theoretical basis; no methodologically acceptable evidence for efficacy</td>
</tr>
</tbody>
</table>

0 = No apparent efficacy
+ = Occasional effect; limited evidence
++ = Clear effect; good evidence from controlled trial(s)
+++ = Strongly documented effect; evidence from multiple controlled trials.

PSYCHOTHERAPY

Cognitive-behavioral therapy. CBT—given either individually or in groups—is the most effective psychotherapy for bulimia (Table 3). CBT typically involves 3 to 6 months of helping the patient focus on her bulimic behaviors and on specific attitudes—such as unrealistic preoccupations with being “too fat”—that perpetuate the behaviors.

In practice, unfortunately, few bulimic patients are offered CBT, perhaps because few clinicians are trained in the specific approach used for bulimia nervosa. If you are not trained in using CBT for bulimia and do not have access to colleagues who offer this treatment, you may begin with medication plus simple behavioral treatments, such as:

- offering supportive therapy in the office
- referring patients to self-help groups for persons with eating disorders.

If this strategy fails, encourage patients to consider CBT—even if they must travel some distance to obtain it.

Other specialized psychotherapies. Dialectical behavior therapy and interpersonal psychotherapy appear to be effective in bulimia. Again, however, clinicians who lack training in these techniques or access to local experts may be unable to offer them. Psychodynamic therapy does not appear to offer greater benefit in bulimia nervosa than ordinary supportive counseling.

Dubious therapies. One psychodynamic approach—regrettably still practiced—is “recovered memory therapy.” Therapists who use it claim that childhood sexual abuse or other trauma can cause bulimic symptoms but patients have repressed the memory of these events.

No methodologically sound evidence has shown that childhood sexual abuse can cause bulimia nervosa years or decades later. Nor is there acceptable evidence that people can repress the memory of a traumatic experience. Therapists administering recovered memory therapy have been subjected to malpractice judgments totaling tens of millions of dollars from suits filed by patients who eventually realized that so-called “recovered” memories were false.

continued
Another dubious therapy—eye movement desensitization and reprocessing (EMDR)—also may involve attempts to “recover” memories of putative traumatic events. No methodological sound evidence has shown that EMDR is effective in bulimic patients, and the technique’s theoretical basis is questionable.

References


Related resources

- Association for Advancement of Behavior Therapy. www.aabt.org

<table>
<thead>
<tr>
<th>DRUG BRAND NAMES</th>
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<tbody>
<tr>
<td>Bupropion • Wellbutrin</td>
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<tr>
<td>Citalopram • Celexa</td>
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<tr>
<td>Desipramine • Norpramin</td>
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<tr>
<td>Fluoxetine • Prozac</td>
</tr>
<tr>
<td>Lithium • Lithobid, Eskalith</td>
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<tr>
<td>Nortriptyline • Pamolor, Aventyl</td>
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<tr>
<td>Sertraline • Zoloft</td>
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<tr>
<td>Topiramate • Topamax</td>
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<tr>
<td>Trazodone • Desyrel</td>
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<tr>
<td>Lithostramine • Cytomel</td>
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<tr>
<td>Venlafaxine • Effexor</td>
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