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In Response:

The Biological Basis and Treatment of Bulimia

Jeffrey M. Jonas, M.D.

The recent papers in the *Jefferson Journal of Psychiatry* by Drs. Levin ("Bulimia as a Masturbatory Equivalent," July, 1985) and Wilson ("A Discussion of 'Bulimia as a Masturbatory Equivalent'," Winter, 1986) contain a number of theories and recommendations about the etiology and treatment of bulimia which should be addressed. In large measure, both papers present a dynamic view of this disorder. However, psychodynamic formulations are but one of many theories put forward to explain this disorder. Other theories of etiology include notions that bulimia arises from cultural factors and an increased attention to body image, from disorders within the family as a whole, or from assorted behavioral factors which somehow reinforce binge-eating and purging.

One way to evaluate a theory of etiology is to ask whether treatments based upon a given theory benefit individuals with the disorder in question. For example, it would be of interest to know what evidence exists that individual psychotherapy is superior to group psychotherapy for the treatment of bulimia. Similarly it is important to know what empirical data underlie these theories. In the case of bulimia, as noted above, there are a host of ideas about what causes the disorder. And while a review of this literature is beyond the scope of this paper, at this time there is no systematic, controlled, non-anecdotal evidence that psychodynamic, behavioral, or cultural factors alone can lead to bulimia (1,2).

In terms of treatment, we have a similar situation, where controlled studies of individual psychotherapy, group therapy, and behavior therapy are also lacking. There are numerous uncontrolled reports of the efficacy of these modalities in bulimic patients, but it is important to note why controlled studies are important in assessing treatments of this disorder. First, bulimia may remit spontaneously (1), so that a placebo group is needed to evaluate if a given therapy is effective. Second, bulimic patients may have amelioration of symptoms in structured settings. In assessing the first 40 consecutive inpatients

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admitted to the Fair Oaks Hospital Eating Disorders program, 33 had complete cessation of bingeing within the first 10 days. Such a response is misleading. At the end of two weeks (before treatment is completed) nearly two-thirds of the 33 "responders" will binge and purge on their first pass, and nearly all of the 33 "responders" will report urges to binge during the first two weeks. This kind of phenomenon points out the need for a control group, as well as follow-up information, before a given treatment can be said to be effective.

Given the prevalence of bulimia, there are relatively few studies of psychological and group treatments. One difficulty often cited in designing studies is that response to therapy cannot be assessed in a systematic manner. In bulimia, this is not the case. Bulimic patients have a number of overt symptoms which can be followed readily, such as the number of binge and purge episodes each day, severity of binge and purge urges, and severity of depression. Thus, there seems little reason why studies of therapeutic efficacy of various modalities could not be undertaken. Yet data supporting the use of many popular therapies are scanty at best. There are at this time no controlled studies of individual psychotherapy in bulimia beyond uncontrolled case series. Cognitive behavioral therapy has been reported successful in two uncontrolled studies (3,4), whereas therapy has been reported useful in several uncontrolled reports (5-9).

We are not advocating that individuals with bulimia be deprived of group or individual therapy. Rather, we are pointing out that at this time there is little to commend one talking therapy from another. Recommendations for group and individual therapy should be made on an individual basis, rather than on the basis of diagnosis alone. In conjunction with such treatment, however, we must now address the use of a different therapeutic technique which appears applicable to this diagnostic category as a whole—the use of antidepressant medication.

The rationale for the use of antidepressants in bulimia comes from the body of evidence linking bulimia and anorexia nervosa to affective disorders. This evidence stems from three lines of research: studies of phenomenology, family history, and biological tests. Studies of phenomenology have examined groups of bulimic patients seeking treatment for their eating disorder. Such studies have consistently reported that bulimic patients have an increased lifetime prevalence of major depression and bipolar disorder, and that in up to 50 percent of cases the affective disorder antedates the onset of the eating disorder (10-12). Studies of family history also suggest a biological link between affective disorders and bulimia, in that most but not all groups have reported an increased incidence of affective disorders in the families of patients with bulimia (13-15).

The third line of evidence linking eating disorders to affective disorders arises from studies of the dexamethasone suppression test (DST) and thyrotropin releasing hormone stimulation test (TRHST). Both the DST (14,16,17) and TRHST (14) yield positive results in bulimia at rates comparable to those observed among individuals with depression. These results should be interpreted with caution, since metabolic stresses attributable to bingeing, purging,

admitted to the Fair Oaks Hospital Eating Disorders program, 33 had complete cessation of bingeing within the first 10 days. Such a response is misleading. At the end of two weeks (before treatment is completed) nearly two-thirds of the 33 "responders" will binge and purge on their first pass, and nearly all of the 33 "responders" will report urges to binge during the first two weeks. This kind of phenomenon points out the need for a control group, as well as follow-up information, before a given treatment can be said to be effective.

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and weight loss could produce false positive results (18). However, studies of inpatient bulimics have shown that the rate of dexamethasone non-suppression is not affected by hospitalization, even after bingeing and purging are controlled through milieu treatment (16,19).

The evidence summarized above suggests a biological link between bulimia and the affective disorders. It should be noted that there are aspects of the eating disorders not explained by this linkage, such as the relationship of eating disorders and substance abuse (20). However, the affective model of eating disorders has led to an important treatment of bulimia, the use of antidepressant medication.

Numerous open-studies of antidepressants in bulimia have appeared in the literature since 1977 (1,2), describing the use of heterocyclic antidepressants and monoamine oxidase inhibitors (MAOI's). Since then, five placebo-controlled, double-blind studies of heterocyclic antidepressants or MAOI's have been completed (21-25). One study using low dosages of mianserin had negative results (21), and one study using amitriptyline had weakly positive results (22). Both studies appeared to suffer from the use of inadequate dosages of antidepressant. The other three studies of antidepressants reported strongly positive findings. Pope et al. (23) observed a 70 percent reduction of binge-eating in patients taking imipramine in dosages up to 200 mg each day, as compared to virtually no change in a placebo-control group. Significant reductions in depression, food preoccupation, and subjective global improvement were also noted. Walsh et al. had similarly positive results using phenelzine (23), as did Hughes et al. using desipramine (24). The study by Hughes and colleagues was of particular interest because they selected bulimic patients who did not display major depression. Despite this fact, Hughes' group found that 15 (68%) of 22 subjects experienced a remission of their bulimic symptoms within ten weeks. Another important finding of the Hughes study was that plasma levels of desipramine comparable to those required in depression were required for response, and that dosages up to 350 mg of desipramine were required to obtain adequate serum levels (125-275 ug/ml in their laboratory).

Overall, a growing body of evidence supports the use of antidepressant medication in the treatment of bulimia, whether or not the patient suffers from concomitant major depression. Follow-up data from the study of Pope et al. further support the utility of antidepressants (26). After one to two years on medication, 50 percent of patients were free of bulimic symptoms, 45 percent had a greater than 50 percent reduction of symptoms, and only one patient, who had discontinued medication, was unchanged. These promising data are tempered by the fact that complicated pharmacologic treatments were required in some cases in order to achieve maximal results. Nevertheless, a majority of these patients were able to return to full function, and did so without the aid of other ancillary therapies.

A number of caveats should be raised at this point. First, agents other than

antidepressants have been useful in bulimia, including lithium (27), and methylamphetamine (28). Naltrexone has also been reported to be of use in these patients (29), but only on an open-label basis. In general, treatment with thymoleptics must be systematic and careful utilizing medication in adequate dosages for adequate periods of time, carefully monitoring serum levels (30).

Second, medication does not interfere with psychotherapy. If anything the cognitive improvement which patients experience when depression and food preoccupation lessen make them more amenable to therapy.

Third, the notion that patients should be allowed to "experience" their eating disorder in order to achieve insight should be considered as archaic and cruel as notions that antidepressants should be withheld from depressed individuals for like reasons. Such an approach is also medically dangerous, in light of the physical damage which may attend constant bingeing and purging. The role of treatment is to hasten recovery, and antidepressant therapy can help in this regard.

Finally, clinicians often misinterpret the use of antidepressants as a de facto statement against the use of psychotherapy. This is untrue. Many if not most patients with bulimia will have individual and family problems which require individual, group, and family psychotherapy. In our own treatment program, patients are engaged in all of these therapies, in addition to milieu, nutritional, and exercise therapy. In treating the whole patient, one needs to address biological as well as psychological needs. In so doing, the use of thymoleptics plays an important role. Given the evidence for the efficacy of antidepressants in bulimia, there is little reason to withhold them from patients with this disorder. [Ed.: For Dr. Wilson's reply, see the Letters to the Editor section.]

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