

Chapter 42

Pharmacotherapy of Eating Disorders

B. Timothy Walsh

ANOREXIA NERVOSA

Anorexia nervosa is a potentially life-threatening illness that, in some cases, seems resistant to all forms of therapeutic intervention. For this reason, virtually every type of somatic treatment known to psychiatry has been employed at some time in the treatment of patients with anorexia nervosa and has been described as being effective. Currently, the limited number of controlled studies of pharmacological treatment in this illness suggest that medication can play, at most, a secondary role as one component of a comprehensive treatment strategy. In this chapter, I will briefly review the major approaches that have been taken in the use of medication in anorexia nervosa, and what the outcome of such attempts has been.

Antipsychotic Medication

Twenty-five years ago, soon after the introduction of antipsychotic medication into psychiatry, British clinicians described a treatment program using chlorpromazine and insulin to produce rapid weight gain among patients with anorexia nervosa [1]. Chlorpromazine was given in doses of 150 to 1000 mg daily combined with subcutaneous insulin, which was used to induce hypoglycemia in an attempt to increase appetite. In a follow-up report in 1966, these authors reviewed their experience [2]. They compared the outcome of 48 women with anorexia nervosa who had been hospitalized and treated with chlorpromazine and insulin to a series of 48

similar patients who had been admitted over the preceding 20 years and treated in the same hospital but without the use of chlorpromazine. They found that the patients treated with chlorpromazine gained weight substantially faster and left the hospital significantly sooner than the patients who had been treated without medication. However, on follow-up there was no advantage for the chlorpromazine-treated group. After two years, approximately 30% of each group required readmission, and 45% of the patients treated with chlorpromazine had developed bulimia compared with 12% of the patients treated without medication. Furthermore, the chlorpromazine treatment was associated with significant side effects, including grand mal seizures in 5 of the 48 patients. In a more recent review of the treatment of anorexia nervosa [47], Dally noted that although chlorpromazine had once been used for all patients with anorexia nervosa on his service, less than one third of such patients received chlorpromazine in 1981 and usually only for brief periods. The use of insulin had been abandoned entirely.

In recent years, Dutch investigators have explored the utility of antipsychotic medication in a controlled fashion. In 1982, Vandereycken and Pierloot reported a study of the antipsychotic drug pimozide in 18 patients with anorexia nervosa [3]. Patients were all hospitalized and were also treated in a behavior modification program. Patients received three weeks of either pimozide or placebo followed by three weeks of the alternative drug. Unfortunately, the group that was randomized to

begin pimozide was lower in weight at initiation of treatment than the group that started on placebo. The patients who began on pimozide gained weight faster than those who began on placebo, but this difference continued after drug crossover. Overall, there was a trend favoring weight gain on pimozide, but the difference from placebo was not statistically significant. The staff's rating of the patients' attitudes detected no significant effect of drug treatment.

In 1984, Vandereycken reported a study of similar design of the antipsychotic drug sulpiride [4]. Once again, there was a slight trend favoring the drug compared with placebo, but no statistically significant effect was demonstrated either on weight gain or on the staff's rating of the patients' attitudes or behavior.

In summary, although there was significant enthusiasm for the possible utility of antipsychotic medication in anorexia nervosa in the early 1960s, this enthusiasm has not been borne out by controlled study. In addition, the severity of potential side-effects of antipsychotic medication, including not only the grand mal seizures noted by Dally and his co-workers, but also hypotension and the long-term risk of tardive dyskinesia limit the utility of neuroleptics in this syndrome. Despite these limitations, a number of experienced clinicians believe that antipsychotic medications can be of assistance in the management of particularly physically active or particularly compulsive patients who may respond to the sedative properties of antipsychotic drugs. It is interesting to note that although the distortion of body image characteristic of anorexia nervosa at times approaches delusional proportions, there is no indication that antipsychotic medications reduce these cognitive disturbances.

Antidepressant Medication

Although some degree of mood disturbance has long been noted among patients with anorexia nervosa, the possibility that there is a link between anorexia nervosa and major affective illness has attracted significant attention only in recent years and has led to a number of attempts to explore the utility of antidepressant medication. Several case reports suggest that some patients with anorexia nervosa obtain impressive benefit from antidepressants [5-7]. However, other reports imply that substantial benefit is uncommon [8,9].

Several recent controlled studies have attempted to assess the utility of antidepressant medication in anorexia nervosa. In 1980 Lacey and Crisp reported a study of the tricyclic antidepressant clomipramine in 16 hospitalized patients [10]. Clomipramine is widely used in Europe for the treatment of depression and, in addition, has been found in several studies to be of significant

benefit in the treatment of patients with obsessive compulsive disorder. For these reasons, clomipramine would appear to be a particularly attractive medication for patients with anorexia nervosa who frequently exhibit both mood disturbance and compulsive symptoms. In this trial, however, the clomipramine-treated patients did no better than the patients receiving placebo. The implications of this study are limited by the small patient sample (a total of 16) and by a low dose of drug (50 mg/day) even allowing for the low weight of the patients.

The two other controlled studies of antidepressant medication in anorexia nervosa have used amitriptyline. Beiderman et al examined the usefulness of amitriptyline in a sample of 43 patients [11]. Twenty-five of these 43 patients elected to participate in a double-blind, placebo-controlled trial, and 11 were randomized to receive amitriptyline and 14 to receive placebo. The 18 patients who refused participation in the trial were used as a comparison group. Patients were treated at one of two institutions for five weeks; five were outpatients and 38 were inpatients. The mean dose of medication was 115 mg/day (2.8 mg/kg). This study was unable to detect any advantage for the amitriptyline-treated group compared with the placebo group or with the group of patients who had refused to participate in the study. Like the previous study of Lacey and Crisp, this study is limited by its small sample size and also by the fact that two of the patients taking amitriptyline had very low plasma levels of drug, suggesting poor compliance.

The third double-blind, placebo-controlled trial of antidepressant medication in anorexia nervosa was conducted by Halmi et al [12]. In this study, 72 female inpatients with anorexia nervosa were treated at one of two centers and, in addition, were randomly assigned to receive one of three medications for up to six weeks: placebo, amitriptyline, or cyproheptadine. There were indications of a marginal advantage for the amitriptyline-treated group in reaching target weight faster than patients treated with placebo. This study is notable for its large sample size and the rigorous analysis of the treatment response; however, plasma level data have not yet been reported, so there is some question about the adequacy of the pharmacological intervention.

In summary, despite the indications of significant mood disturbances associated with the syndrome of anorexia nervosa, there are few rigorous data to suggest a major role for antidepressant medication in this syndrome. There are several potential explanations for this finding. First, it is possible that the depression associated with anorexia nervosa is distinct from those forms of major depressive illness that respond to antidepressant medication [13]. Second, the physiological disruptions

induced by starvation might conceivably make patients refractory who might otherwise respond to antidepressant treatment. Third, it is possible that only a subset of patients with anorexia nervosa are drug-responsive, and the positive drug effect in these patients is obscured by the results of other patients who are not drug-responsive. Finally, it should be noted that only three controlled studies involving only two antidepressant agents have been reported.

From a clinical perspective, the role of antidepressant medication in anorexia nervosa must, at this point, be viewed as an adjunct to a comprehensive and multifaceted treatment approach. The relatively limited benefits of antidepressant medication must be weighed against the potential side-effects in this medically ill population, including orthostatic hypotension and cardiac conduction disturbances.

Cyproheptadine

Cyproheptadine is a serotonin and histamine antagonist that is primarily used to treat allergic conditions. It was noted that patients receiving cyproheptadine for such problems sometimes gained weight, and several studies have explored whether this effect might be usefully applied to underweight patients with anorexia nervosa.

The first double-blind, placebo-controlled trial of cyproheptadine in anorexia nervosa was reported by Vigersky and Loriaux in 1977 [14]. They treated 24 outpatients with cyproheptadine, 12 mg daily, or placebo for eight weeks and were unable to detect a significant drug/placebo differences. They noted difficulties in interpreting this negative result because of uncertainty about patient compliance with medication and recommended further assessment of cyproheptadine.

In 1979 Goldberg et al reported the results of a multicenter trial of cyproheptadine in hospitalized patients with anorexia nervosa [15]. Cyproheptadine was given in doses of 12 to 32 mg/day for 35 days. The cyproheptadine-treated patients gained slightly more weight than the placebo-treated patients, but this difference was not statistically significant.

In the study of Halmi et al previously described, cyproheptadine was compared with amitriptyline and placebo in 72 patients hospitalized for anorexia nervosa. Cyproheptadine was slightly more effective than placebo in inducing weight gain and in relieving depression. It was of interest that there was a differential drug effect related to the presence of bulimia, so that cyproheptadine significantly increased treatment efficiency in the nonbulimic patients and impaired treatment efficiency in the bulimic patients. Although there have been consistent reports of differences between bulimic and nonbulimic subgroups of patients with anorexia ner-

vosa, it is unclear why cyproheptadine should affect these two subgroups differentially.

In sum, there are indications that cyproheptadine in relatively large doses may have some mild effect in promoting weight gain and relieving depression in anorexia nervosa. However, these therapeutic effects appear to be modest. One major advantage of cyproheptadine is that it appears to have few side-effects, even in the relatively large doses used.

Other Pharmacological Agents

A variety of other pharmacological agents have been used in the treatment of patients with anorexia nervosa, and some have been subjected to double-blind, placebo-controlled trials. A few open trials suggested that lithium might be of benefit [16,17]. In 1981 Gross et al reported a double-blind, placebo-controlled trial of lithium in the treatment of 16 women hospitalized for anorexia nervosa [18]. Over the four-week trial, the lithium-treated patients gained slightly more weight than the placebo-treated patients. However, the difference was not statistically significant. Because of baseline differences between the drug- and placebo-treated groups, because both groups did improve over this brief trial, and because of the small sample size, this study cannot be taken to exclude definitively the use of lithium in patients with anorexia nervosa. However, at present, except for the rare patient who has both bipolar affective illness and anorexia nervosa, there is no clear indication for the use of lithium in anorexia nervosa.

Gross et al also conducted a controlled trial of delta-9-tetrahydrocannabinol (THC), which is the active ingredient of marijuana [19]. The rationale was that since smoking marijuana is known to increase appetite, THC might be useful in assisting patients with anorexia nervosa to gain weight. In fact, the THC had a negative therapeutic impact; it produced a significant amount of dysphoria and had no detectable effect on weight.

Another avenue for the treatment of anorexia nervosa with medication has focused on a known physiological abnormality of patients with this illness, namely that gastric emptying is slowed [20]. Because many patients with anorexia nervosa complain of feeling bloated after meals, it has been suggested that drugs that increase gastric emptying may relieve symptoms and assist in the normalization of eating. Several authors have reported that gastric emptying is increased by the administration of drugs such as metoclopramide, bethanechol, and domperidon [20-22]. In these uncontrolled trials, patients have had fewer complaints of bloating after meals, but the overall utility of the drugs was not clear. Furthermore, the use of metoclopramide was associated with significant depression and with hormonal changes, potentially limiting its use in anorexia

nervosa.

Finally, a number of other drugs and hormonal preparations have been described in case reports as being of benefit to patients with anorexia nervosa. The drugs used include glycerol, phenoxybenzamine, L-DOPA, and anabolic steroids [23-26]. None of these drugs has been evaluated by controlled trial, and there is little evidence for their utility in anorexia nervosa.

Summary

In short, despite the multitude of biological abnormalities described in patients with anorexia nervosa, there is no compelling evidence that somatic treatments other than weight restoration are of dramatic benefit in this syndrome. As noted above, occasional patients, particularly agitated ones, may benefit from low doses of antipsychotic medications, and there are suggestions that some patients may benefit from antidepressant medication or from the serotonin antagonist cyproheptadine. However, at the moment, medication must be viewed as playing only a secondary role in the treatment of anorexia nervosa.

BULIMIA

Although the syndrome of bulimia in normal-weight individuals has only recently gained the attention of the mental health professions, an impressive amount of effort has already been devoted to the evaluation of pharmacological methods of treatment. These pharmacological interventions have been based on one of two conceptual models. The first model is that bulimia is a manifestation of a seizure disorder and could be treated effectively with anticonvulsant medication. The second model that has been explored more recently is that bulimia is in some way linked to disturbances of mood and can be approached through the use of antidepressant medication.

Anticonvulsant treatment

In the mid-1970s Green and Rau suggested a relationship between binge eating and seizure disorders [27-30]. They noted that patients with binge eating typically described the binges as episodic, uncontrollable, and ego-dystonic. The episodes were also frequently preceded by a change in mental state that could be interpreted as an aura. Green and Rau were struck by the similarity of these features to those of patients with seizure disorders. Over several years they obtained EEGs in patients with "compulsive eating" and found that a majority had abnormal EEGs, most frequently the occurrence of 14- and 6-per-second spikes. These findings led them to propose that "compulsive eaters

have a *primary* neurologic disorder similar to epilepsy." On the basis of this formulation they treated patients with phenytoin and described impressive results in a series of uncontrolled trials.

There has been a limited amount of additional work pursuing this hypothesis. However, the work that has been done generally does not suggest that bulimia is a form of seizure disorder. In 1977 Wermuth et al reported a controlled study of phenytoin in bulimia [31]. As was true of the patients described by Green and Rau, the patients in the study of Wermuth et al were more heterogeneous in terms of body weight and eating behavior than patients who have been studied with bulimia in the last five years. Wermuth et al obtained EEGs from 20 patients and found definite abnormalities in only three, a far smaller fraction than that reported in the original studies of Green and Rau. One reason for this discrepancy is that electroencephalographers became more skeptical that the 14- and 6-per-second spike pattern was of any significance. Wermuth et al compared phenytoin to placebo in a double-blind crossover experiment in which patients received either phenytoin for three weeks followed by placebo for three weeks or the opposite sequence. The ten patients who began on phenytoin improved in comparison to their baseline, but when they were switched to placebo, there was no deterioration. The nine patients who received the placebo/phenytoin sequence did not improve on placebo, but reduced their bingeing somewhat on phenytoin. Overall, the difference between placebo and phenytoin treatment was not statistically significant. Contrary to what one would predict if bulimia were a form of seizure disorder, there was no relationship between pretreatment EEG abnormality and the response to phenytoin, or between the plasma level of phenytoin and the clinical response. In addition, the efficacy of treatment with phenytoin was modest even in those patients who did obtain some benefit.

Greenway et al obtained EEGs in seven patients with obesity associated with compulsive overeating [32]. No significant abnormalities were detected. Four patients were treated with phenytoin in a controlled fashion and no evidence of therapeutic efficacy was obtained.

There is only one other study that may bear on the idea that bulimia is a form of seizure disorder. Kaplan et al reported that one of six normal-weight patients with bulimia responded impressively to treatment with carbamazepine in a double-blind, placebo-controlled crossover trial [33]. None of the other five patients had significant therapeutic responses. The patient who did respond had, in addition to bulimia, a mood disturbance that may have been a mild form of bipolar disorder. Carbamazepine is an anticonvulsant drug that is widely used

in the treatment of seizure disorders but that also appears to be effective in the treatment of some patients with bipolar mood disturbance. Therefore, the efficacy of carbamazepine in this single patient with bulimia cannot be clearly interpreted as evidence of a seizure disturbance.

In sum, little compelling evidence has been gathered to support the hypothesis of Green and Rau that bulimia is a form of seizure disorder. There are hints that phenytoin may be of use for the treatment of some patients with bulimia, but additional controlled studies of phenytoin in eating disorders will be required to clarify its potential role.

Antidepressant Treatment

The relationship between bulimia in normal-weight individuals and mood disturbance has generated a great deal of interest and some controversy. There is general agreement that patients with bulimia are more anxious, depressed, and irritable than control subjects. Most studies that have used structured rating instruments have also found an elevated lifetime frequency of major depression among patients with bulimia. However, it is far from clear whether bulimia should be viewed as an unusual manifestation of an underlying affective illness or whether the mood disturbances of bulimia are best regarded as a secondary consequence of the eating disorder.

While this controversy concerning the significance of the mood disturbance in bulimia remains unsettled, the recognition of depression and anxiety among patients with bulimia has led to a series of trials of antidepressant medications. The first clear description is that of Rich, who in 1978 described a 21-year-old woman with bulimia and depression who failed to respond to a tricyclic antidepressant but had an impressive response to the monoamine oxidase inhibitor (MAOI) phenelzine [34]. In 1982 Pope et al described a series of normal-weight patients with bulimia who responded to open treatment with tricyclic antidepressants, and our own group published similar results using MAOIs [35,36]. These series of patients treated openly were followed by reports of seven double-blind, placebo-controlled trials of antidepressants in normal-weight patients with bulimia.

The first study reported was that of Sabine et al, who examined the use of mianserin, an antidepressant drug available in Europe [37]. Twenty patients were randomized to receive mianserin, 60 mg per day, and 30 to receive placebo. Both groups improved somewhat over the eight-week trial, but there was no significant difference between the drug- and placebo-treated groups. A major limitation of the study is that the dose of mianserin used was low compared with that sometimes re-

quired for treatment of depression. In addition, this study may have treated a group of patients who are less severely ill than those who were treated in subsequent studies.

Pope et al followed their initial report with a double-blind, placebo-controlled trial of imipramine in 22 normal-weight patients with bulimia [38]. The placebo-treated group improved minimally, while the group receiving imipramine reduced their binge frequency about 75%, a statistically significant result. Hughes et al conducted a similar study of patients with bulimia using the tricyclic antidepressant desipramine [39]. In a dose of 200 mg daily, desipramine was strikingly superior to placebo, and 15 of the 22 patients (68%) who received desipramine either during the study or later during follow-up achieved symptomatic remission.

Mitchell and Groat reported a controlled study of amitriptyline in which both the drug- and placebo-treated groups improved [40]. It is interesting that the therapeutic effect of amitriptyline in this study was similar to the effect of the active drug in the controlled studies of Pope et al and Hughes et al. The major difference between the studies is that in the amitriptyline study the placebo-treated patients improved substantially, while the placebo-treated groups did not improve in the other two studies. The procedures used to treat the patients receiving placebo were similar in all three studies, and the reason for the discrepant outcome of placebo treatment is not clear. While the patients in these studies were similar in simple clinical terms such as age, percent of ideal weight, and frequency of bulimic behavior, it is possible that these similarities camouflage important differences between patient groups treated at different centers.

Agras et al recently reported the results of a controlled trial of imipramine in 20 bulimic women [48]. This study differs from the preceding tricyclic antidepressant trials in its longer length (16 weeks vs. 6 to 8 weeks) and in its rigorous attempt at restricting therapeutic interventions other than medication. The imipramine-treated group obtained greater reductions in the frequency of binge eating and of purging than the placebo-treated group, but the differences between drug and placebo were not as impressive as in the studies of Pope et al [38] and Hughes et al [39]. For example, in the Agras et al study, there was a 72% reduction in binge frequency in the imipramine group after 16 weeks, similar to the reduction at 6 weeks reported by Pope et al in their imipramine group. However, at 16 weeks, the placebo-treated group of Agras et al obtained a reduction of 43% in binge eating frequency compared to virtually no change in the placebo-group in the study of Pope et al. In short, the study of Agras et al supports the efficacy of tricyclic antidepressant treat-

ment compared to placebo, but, like the study of Mitchell & Groat, raises questions about the reasons for differences in outcome in different centers.

We have conducted a controlled study of the MAOI phenelzine in the treatment of normal-weight patients with bulimia. We embarked on this investigation several years ago because of our impression that many bulimic patients presented symptoms of anxiety and depression similar to those of patients with "atypical" depression, which has been thought to respond particularly well to treatment with MAOIs. An analysis of the first 30 patients completing our double-blind, placebo-controlled trial indicated a significant advantage for phenelzine. Six of 14 phenelzine-treated patients were in remission at the completion of the study, compared with none of the 16 placebo-treated patients ($p < .01$) [41]. Our data, like those of three of the four studies using tricyclic antidepressants, suggest that antidepressant medications are of benefit to some patients with bulimia.

We were initially concerned about the potential risks of giving bulimic patients, who by definition cannot control their eating, MAOIs, which demand that patients avoid certain foods or risk a hypertensive reaction. We have carefully screened patients for their ability to comply with a tyramine-free diet and have not had a serious hypertensive reaction because of patient noncompliance. However, we have been impressed that, in this population, other side-effects of phenelzine such as postural hypotension and sleep disturbance caused significant difficulty and are a major impediment to the use of MAOIs in many patients.

Kennedy et al have reported the preliminary results of a controlled trial of another MAOI in bulimia [49]. They examined the utility of isocarboxazid in a double-blind, placebo-controlled crossover trial in 18 patients. As in our study of phenelzine, there was a significant advantage for the MAOI compared to placebo.

In addition to these controlled trials, therapeutic response has been reported on the basis of open trial experience with a variety of tricyclic antidepressants, MAOIs, trazodone, bupropion, nomifensine, and lithium carbonate [42-46]. The data are now reasonably convincing that, at least for some patients of normal body weight with bulimia, antidepressant medications have significant therapeutic effect. However, some important questions about the use of medication in such patients remain unanswered. First, it is unclear which patients are most likely to respond. Most of the controlled trials of antidepressant medications have studied patients who are chronically and moderately-to-severely ill. It is not clear if the same drug-placebo difference would be found in less severely ill patients. Although one could anticipate that depressed bulimic patients

would be particularly likely to respond to antidepressant medication, the study of Hughes et al [39] explicitly excluded patients with major depressive disorder and yet found impressive results with desipramine. Similarly, in a preliminary analysis of our patients treated with MAOIs, nondepressed patients appear to derive benefit as well as depressed patients. Thus it appears that the clinician cannot rely on the presence of depression to indicate which patients with bulimia will respond to antidepressant medication. Of more concern is the fact that we know very little about the long-term outcome of the drug treatment of bulimia or how best to combine drug treatment with other forms of therapy. It should be noted that all of the controlled studies of antidepressant medication in bulimia are of relatively short duration and there is no knowledge of how long patients who respond to medication need to remain on it or of what the relapse rate is when the drug is discontinued. Answers to these questions will demand the attention of investigators in this field for the next several years.

ACKNOWLEDGEMENTS

This work was supported in part by NIH grants AM-28150, MH-30906, and MH-00383, and by the Communities Foundation of Texas, Inc.

REFERENCES

1. Dally PJ, Sargent W. A new treatment of anorexia nervosa. *Br Med J* 1960; 1:1770-3.
2. Dally P, Sargent W. Treatment and outcome of anorexia nervosa. *Br Med J* 1966; 2:793-5.
3. Vandereycken W, Pierloot R. Pimozide combined with behavior therapy in the short-term treatment of anorexia nervosa. A double-blind, placebo-controlled, cross-over study. *Acta Psychiatr Scand* 1982; 66:445-50.
4. Vandereycken W. Neuroleptics in the short-term treatment of anorexia nervosa. A double-blind placebo-controlled study with sulphiride. *Br J Psychiatry* 1984; 144:288-92.
5. Needleman HL, Waber D. Amitriptyline therapy in patients with anorexia nervosa (letter). *Lancet* 1976; 2:580.
6. White JH, Schnaultz NL. Successful treatment of anorexia nervosa with imipramine. *Dis Nerv Sys* 1977; 38:567-8.
7. Hudson JI, Pope HG, Jonas JM, et al. Treatment of anorexia nervosa with antidepressants. *J Clin Psychopharmacol* 1985; 5:17-23.
8. Mills IH. Amitriptyline therapy in anorexia nervosa (letter). *Lancet* 1976; 2:687.
9. Kendler K. Amitriptyline-induced obesity in anorexia nervosa. *Am J Psychiatry* 1978; 135:1107-8.
10. Lacey JH, Crisp AH. Hunger, food intake and weight:

- the impact of clomipramine on a refeeding anorexia nervosa population. *Postgraduate Medical Journal* 1980; 56(Supplement);79-85.
11. Biederman J, Herzog DB, Rivinus TM, et al. Amitriptyline in the treatment of anorexia nervosa: a double-blind, placebo-controlled study. *J Clin Psychopharmacol* 1985; 5:10-6.
 12. Halmi KA, Eckert E, LaDu TJ, et al. Anorexia nervosa: treatment efficacy of cyproheptadine and amitriptyline. *Arch Gen Psychiatry* 1986; 43:177-81.
 13. Altshuler KZ, Weiner MF. Anorexia nervosa and depression: a dissenting view. *Am J Psychiatry* 1985; 142:328-32.
 14. Vigersky RA, Loriaux DL. The effect of cyproheptadine in anorexia nervosa. A double-blind trial. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977: 349-56.
 15. Goldberg SC, Halmi KA, Eckert ED, et al. Cyproheptadine in anorexia nervosa. *Br J Psychiatry* 1979; 134:67-70.
 16. Barcai A. Lithium in adult anorexia nervosa. A pilot report on two patients. *Acta Psychiatr Scand* 1977; 55:97-101.
 17. Stein GS, Hartshorn S, Jones J, et al. Lithium in a case of severe anorexia nervosa. *Br J Psychiatry* 1982; 140:526-8.
 18. Gross HA, Ebert MH, Faden VB, et al. A double-blind controlled trial of lithium carbonate in primary anorexia nervosa. *J Clin Psychopharmacol* 1981; 1:376-81.
 19. Gross H, Ebert MH, Faden VB, et al. A double-blind trial of delta-9-tetrahydrocannabinol in primary anorexia nervosa. *J Clin Psychopharmacol* 1983; 3:165-71.
 20. Dubois A, Gross HA, Richter JE, et al. Effect of bethanechol on gastric functions in primary anorexia nervosa. *Dig Dis Sc* 1981; 26:598-600.
 21. Moldofsky H, Jeuniewicz N, Garfinkel PE. Preliminary report on metoclopramide in anorexia nervosa. In Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:373-6.
 22. Saleh JW, Lebowl P. Metoclopramide-induced gastric emptying in patients with anorexia nervosa. *Am J Gastroenterology* 1980; 74:127-32.
 23. Caplin H, Ginsburg J, Beaconsfield P. Glycerol and treatment of anorexia. *Lancet* 1973; 1:319.
 24. Tec L. Nandrolone in anorexia nervosa (letter). *JAMA* 1974; 229:1423.
 25. Redmond DE, Swann A, Heninger GR. Phenoxybenzamine in anorexia nervosa. *Lancet* 1976; 2:307.
 26. Johanson AJ, Knorr NJ. L-dopa as treatment for anorexia nervosa. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:363-72.
 27. Green RS, Rau JH. Treatment of compulsive eating disturbances with anticonvulsant medication. *Am J Psychiatry* 1974; 131:428-32.
 28. Rau JH, Green RS. Compulsive eating: a neuropsychologic approach to certain eating disorders. *Comprehensive Psychiatry* 1975;16:223-31.
 29. Green RS, Rau JH. The use of diphenylhydantoin in compulsive eating disorders: Further studies. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:377-82.
 30. Rau JH, Struve FA, Green RS. Electroencephalographic correlates of compulsive eating. *Clinical Electroencephalography* 1979; 10:180-9.
 31. Wermuth BM, Davis KL, Hollister LE, et al. Phenytoin treatment of the binge-eating syndrome. *Am J Psychiatry* 1977; 134:1249-53.
 32. Greenway FL, Dahms WT, Bray GA. Phenytoin as a treatment of obesity associated with compulsive eating. *Curr Ther Res* 1977; 21:338-42.
 33. Kaplan AS, Garfinkel PE, Darby PL, et al. Carbamazepine in the treatment of bulimia. *Am J Psychiatry* 1983; 140:1225-6.
 34. Rich CL. Self-induced vomiting. *Psychiatric consideration*. *JAMA* 1978; 239:2688-9.
 35. Pope HG, Hudson JI. Treatment of bulimia with antidepressants. *Psychopharmacology* 1982; 78:176-9.
 36. Walsh BT, Stewart JW, Wright L, et al. Treatment of bulimia with monoamine oxidase inhibitors. *Am J Psychiatry* 1982; 139:1629-30.
 37. Sabine EJ, Yonace A, Farrington AJ, et al. Bulimia nervosa: a placebo-controlled, double blind therapeutic trial of mianserin. *Br J Clin Pharmacol* 1983; 15:195S-202S.
 38. Pope HG, Hudson JI, Jonas JM, et al. Bulimia treated with imipramine: a placebo-controlled, double-blind study. *Am J Psychiatry* 1983; 140:554-8.
 39. Hughes PL, Wells LA, Cunningham CJ et al. Treating bulimia with desipramine. A double-blind, placebo-controlled study. *Arch Gen Psychiatry* 1986; 43:182-6.
 40. Mitchell JE, Groat R. A placebo-controlled, double-blind trial of amitriptyline in bulimia. *J Clin Psychopharmacol* 1984; 4:186-93.
 41. Walsh BT, Stewart JW, Roose SP, et al. A double-blind trial of phenelzine in bulimia. *J of Psychiatric Research* 1985; 19:485-9.
 42. Pope HG, Hudson JI, Jonas JM. Antidepressant treatment of bulimia: preliminary experience and practical recommendations. *J Clin Psychopharmacol* 1983; 3:274-81.
 43. Hsu LKG. Treatment of bulimia with lithium. *Am J Psychiatry* 1984; 141:1260-2.
 44. Horne RL. Bupropion in the treatment of bulimia. Paper presented at the 138th Meeting of the American Psychiatric Association, Dallas, Texas, 1985.
 45. Brotman A, Herzog DB, Woods SW. Antidepressant treatment of bulimia: The relationship between bingeing and depressive symptomatology. *J Clin Psychiatry* 1984; 45:7-9.
 46. Pope HG, Heridge PL, Hudson JI, Fontaine R, Yurgelun-Todd D. Treatment of bulimia with nomifensine. Presented in the New Research Program. 138th Meeting of the American Psychiatric Association, Dallas, Texas, 1985.
 47. Dally P. Treatment of Anorexia nervosa. *Br J Hosp Med* 1981; 25:434-40.
 48. Agras WS, Dorian B, Kirkley BG et al. Imipramine in the treatment of bulimia: a double-blind controlled

- study. *Int J Eat Dis* 1987; 6:29-38.
49. Kennedy S, Piran N, Garfinkel PE. Isocarboxazid in the treatment of bulimia (letter). *Am J Psychiatry* 1986; 143:1495-6.

Chapter 43

Self-Help in Anorexia and Bulimia: Principles of Organization and Practice

Félix E.F. Larocca

INTRODUCTION

This chapter will cover the important aspects of organizing self-help groups (focusing on those that apply to treating eating disorders). Although literature abounds with theories and hypotheses on self-help in general [1-3], there are few theories on how self-help groups are formed and conducted in the treatment of anorectic and bulimic patients [4].

Bulimia Anorexia Self-Help, Inc., started in April of 1981. The idea for such a self-help organization began when more and more patients asked to meet with the therapist in a group rather than individually. Soon relatives and other members of the community became involved. A specific format was then designed to incorporate self-help groups with informational lectures aimed at educating patients and families alike. After several well-received meetings of this kind (dubbed BASH meetings), BASH began publishing a monthly newsletter. As BASH grew and the self-help format evolved, a facilitator's program was started and resulted in the publication of a formal training manual [5]. A summary of the evolution of BASH has been given elsewhere [6].

The self-help format of BASH was chosen by Rubel as a model self-help organization in structure and philosophy [4]. Additional literature on the effectiveness of the BASH format can be found in articles by Fauri and Larocca [6,7]. This chapter will outline the prin-

ciples of organization and the method of practice of self-help groups using the BASH format as a model.

HISTORY OF MODERN CONCEPTS OF THE SELF-HELP GROUP

Alcoholics Anonymous (AA), the first known modern health-related self-help group was founded in 1935 [8]. Other self-help groups, each with a different purpose and founder, soon followed. Recover, Inc., was started in 1937 by Abraham Low for former mental patients. In 1955 Integrity Groups were founded to help people realize a better psychological life [9]. An Australian self-help group, GROW, was founded to help ex-mental patients lead a more normal life [10]. Synanon, Compassionate Friends, Parents Anonymous, and Epilepsy Self-Help are only some of the many other health-related self-help groups founded between 1950 and 1975 [11-14]. The first eating disorder self-help group, Anorexia Aid Society, was formed in 1974 in England.

Self-help thrived throughout the 1970s and continued to grow in this decade. Gussow and Tracey estimated that self-help groups must grow at a rate of 3% a year to survive [15]. A successful self-help group must generate growth in order to keep operating. AA and Parents Anonymous (PA) both surpassed the 3% rate of growth between 1972 and 1978 when the number of

AA chapters doubled and PA chapters grew from 40 to 1,000 [16].

To keep pace with the necessary growth, other aspects of the self-help group must also change. Borman was correct when he suggested that the group's organizational structure will need to take diverse forms while continuing to reflect the stable ideology and philosophy of the group [17]. The group's energy may need to be redirected as it becomes more concerned with recruitment, organizational maintenance, chapter development, and public education [17,18].

The nature of group membership and leadership may also change [10]. Though many members of self-help groups join during a time of crisis, they can remain vital participants even after the crisis has subsided. Some can take on group leadership or administrative roles [4,6,19]. Others can work in community awareness [4]. For example, the efforts of GROW members to share their group with the community resulted in an increase in members who were not ex-mental patients but needed GROW's kind of help [17].

Most self-help groups have had to develop mutually beneficial relationships with affiliated professionals, institutions, and voluntary fund-raising associations. The Heart Association, for example, donates funds to Mended Hearts, a self-help group for heart patients. Hospitals and detoxification centers have special ties with AA groups. And some hospitals are now working in tandem with eating disorder self-help groups [4,7].

Although many self-help groups benefit financially from these relationships, a systematic means of acquiring financial support is found in all successful self-help groups [17,20].

CHARACTERISTICS OF THE MODERN SELF-HELP GROUP

Because self-help cannot be studied by the use of a control group, current studies of them often prove unreliable. There are other problems, too, that make the scientific study of self-help groups difficult: Membership cannot be denied to those in need of help; persons afflicted with the disorder who do not seek help are impossible to detect; most members are multiple-help seekers; and outcome studies fail to determine which type of help was the most significant in the patient's recovery [21]. Despite these obstacles, a number of studies have attempted to answer some questions about the characteristics of self-help groups [21-23].

Some researchers divide health-related self-help groups into two types [17,22]. The first type (like AA) is devoted to changing each member's behavior—group members view their membership as permanent. The

second type is a coping group (like Parents Without Partners), where members view membership as temporary. Members of this type of group usually join during a time of crisis. Frane et al [23] and Levy [24] concluded that in both types of groups the support and mutual disclosure offered by the group often encourages members' continued participation.

Membership in a self-help group extends the size and depth of the members' social network. Lieberman studied help-seeking behavior in members of self-help groups and in individuals who enter psychotherapy [25]. He hypothesized that social networks:

1. Buffer the experience of stress,
2. Decrease the need for professional help by replacing it with instrumental and affective support,
3. Act as screening and referral agents for professional services, and
4. Transmit positive attitudes, values, and norms about help-seeking.

Sidel and Sidel believe the growth of self-help groups is a response to inadequate and unequal distribution of professional resources [26]. The view self-help groups as products of "cop-out" by professionals who blame the victim instead of working to change the deeper problems in society that contribute to members' frustrations. They write, "The groups can exacerbate the very symptoms which caused members to seek help... [and] we now speak of *coping* to refer to what was once called *living*" [26].

Katz and Bender agree that proliferation of self-help groups is a response to inadequate resources, but they view this as a positive response [2]. They see the growth of self-help groups as a "grass-roots" consumer response consistent with the spirit of change and adaptation throughout American history. Reissman also agrees. He believes that on a micro-level self-help groups encourage members to live effectively, and on macro-level they shift members' attitudes to a "consumer-based approach." Reissman believes that participating as an equal member of a group instead of seeking help alone is a major benefit of self-help groups [27]. Levy is correct when he writes, "Self-help represents an effective and inexpensive alternative to the purchased friendship of psychotherapy" [28].

Mutual need, commitment, and sharing are often not enough, however, to sustain a self-help group. Strong leadership is required. Eating disorder self-help groups are no exception.

Rubel suggests the dynamics of anorexia and bulimia prevent long-term commitment to self-help groups and, unless some of those involved have above-average interpersonal skills, the groups usually disband [4]. What then are the mechanisms and interactions in self-

help groups that allow leaders to arise and members to be helped?

PROCESSES AT WORK IN SELF-HELP GROUPS

Levy divides the processes that take place in self-help groups into two types: behavior-oriented and cognitive-oriented [29]. Behavior of members of self-help groups is changed by group participation in various ways, such as social reinforcement of desirable behaviors, training in self-control, modeling of coping methods, and devising a program of action.

Cognitive processes serve to remove members' mystification of their experience, increase their expectancy of change, and provide a rationale for their problem. Normative and practical information is shared among participants. Most importantly, the "range of alternative perceptions of members' problems and circumstances and the actions they may take to cope with them broadens" [29]. Members essentially learn to discriminate for themselves between what works and what does not.

There are many similarities between self-help groups and psychotherapy groups. However, certain features distinguish them from each other. Hurvitz [30] concluded that mutual-aid groups use more subjective, peer-initiated approaches than orthodox psychotherapy. Instead of the private disclosures of psychotherapy, experiences are shared in group therapy. Where the psychotherapist is trained to use distance and objectivity, group participants are more judgemental with each other. In psychotherapy, objectivity is the goal. This objectivity has limitations—the patient may come to rely on the therapist instead of assuming more responsibility for himself or herself. Self-help groups can counterbalance the limitations of psychotherapy [30].

Thus, it can be seen that the professional is an important part of a successful self-help group, but is just that—a part. A balance is needed between professional and member responsibility. BASH is one example of a self-help organization with a balanced format. Along with psychotherapy, facilitator training and supervision is used to ensure that the quality of group interaction is high. Because the facilitators are chosen from among membership, the self-help premise of "equal status" relationships is preserved.

Health professionals also can contribute to the evaluative, educational, organizational, and community outreach components of self-help groups. Larocca and Kolodny write, "In the area of self-help such partnership and colleague complementarity can be particularly effective" [31].

RESEARCH ISSUES AND CURRENT HYPOTHESES

The use of self-help groups in the treatment of eating disorders is a recent innovation. The newness of this approach is reflected in the lack of significant study and research on the effectiveness of self-help in the treatment of eating disorders [13]. The use of self-help groups in general, however, has matured to the point of instigating serious research [17,31]. In this section four major issues in general self-help research will be discussed that could be applied to self-help groups for eating disorders.

The first and most important issue is evaluating the impact of self-help groups on group members. Devices must be developed to measure the effectiveness of a chosen self-help group. This is problematic, however, because certain factors inherent in the self-help group format make it difficult to study outcome. These problems can be summarized as follows:

1. Many participants in self-help groups are multiple help-seekers, making it difficult to determine the effect on their well-being of the self-help group experience alone [21,25].
2. It is difficult to establish an effective control group against which the self-help group can be measured. Should the controls be persons who chose another type of therapy for their problem: Or should the control group be composed of persons who have chosen no method of treatment [32]?
3. Many participants of self-help groups attend intermittently. When assessing the group's impact on a person's recovery, how should attendance be figured into the research protocol? A person may attend one of every three meetings but be more involved in those meetings than the person who attends every time [32].
4. The issue of participant involvement is complex and difficult to measure. One would hypothesize that the person who is more involved in the self-help process would receive more benefits, but factors that make up involvement are hard to pinpoint. Involvement could be measured by persistence of attendance, support of the organization, the volunteering of services, or any number of other outward signs of interest in and devotion to the group process.

Another issue in need of further research is how self-help groups are started. What set of circumstances in the initial organization of the group will give that group the best chance for survival? Leadership, group charter, recruitment of members, frequency of meetings and affiliation with other organizations are some of the specific

characteristics of the group that need to be researched and delineated.

And still another issue needing to be researched is who participates in self-help groups. By finding patterns in the types of people who are likely to be attracted to these groups, one might be able to better assess who would most benefit from this approach.

A final issue also needs to be researched: How do self-help groups work to aid their members? What are the specific change mechanisms in the group process? How important is an ideology in giving the group a character and personality that can help its members? What activities does the group support? What training and supervision should group leaders undergo? This research should try to pinpoint the major, dynamic factors that give the group its life and its salutary power [28]. If these factors can be determined and the other research issues settled, new and existing self-help groups would have a successful model after which to pattern their own organization.

FUTURE DIRECTIONS

After a successful campaign that was launched in the summer of 1984 by BASH in St. Louis, the St. Louis community was the beneficiary of the by-products of a \$300,000 significant grant for the purpose of community awareness and education and the formation of the Mary Anne Richardson Memorial BASH Assistance and Information Center.

The prediction was that with the community's greater awareness of the significance and importance of early detection and intervention on eating disorders a substantially greater number of individuals would seek assistance and find treatment resources early in the development of the eating disorder. Categorically, the BASH organization has moved in this direction and the number of its meetings have been augmented fivefold, reaching approximately 30,000 persons a year.

BASH has also developed a network of further community resources emphasizing transgenerational efforts, including the older (gentry) adult generation and children. In addition, BASH has instigated the development of groups for support of family members who do not necessarily suffer from an eating disorder.

The rising costs of medical treatment and hospitalization are forcing many people to consider alternative forms of therapy [7,33]. This cooperation should enhance the efficacy of self-help as an adjunct to medical treatment.

This efficacy would also enhance another trend that is developing: the greater emphasis on scientific and research material in self-help. Self-help organization,

when structured on the BASH format, can provide needed services to the general public by translating complex technical information into easily understood articles and presentations. Thus, self-help groups can educate the public and gain credibility in the eyes of the community [34].

Finally, a development yet to take place: a national registry of self-help groups, which should evolve in the next few years. Just as persons seeking medical or psychiatric care have the benefit of several types of referral services, those seeking self-help therapy deserve information on the kinds of help available. This registry should include evaluation of the type of care, perhaps by a national board yet to be created [35], numbers of years in existence, fees, curriculum for training group leaders, format of meetings, and other pertinent information.

However, as Larocca [36] lamented in his editorial on the Tower of Babel, the issues confronting the development of such a therapist registry are dwarfed by the obstacles arising from such undertaking. As in any new or growing field, the quality of services can vary drastically. A national referral service would help ensure, if properly monitored by supervising agencies, that every person who seeks self-help will find the best program for his or her needs.

Finally, with the instigation of National Eating Disorders Awareness Week by BASH in 1981, and with the mailings of many thousands of packages of free information to every state of the union, its possessions and other countries, BASH has helped insure the realization this year by the American Psychiatric Association, for the first time, of its observation of a Mental Illness Awareness Week, with all its important derivations.

CONCLUSION

In this chapter the present state of self-help has been summarized in relation to the study of a model self-help group. Historically, the self-help movement for anorexia and bulimia is relatively new. But because of its effectiveness in treating eating disorders, it has grown by quantum leaps. The time has come for a more systematic and scientific point of view to be taken in the study of self-help for the treatment of eating disorders. As Rubel asserts, the success and survival of self-help groups for eating disorders depends on systematic structuring and direction by a professional committed to the group's continuance [7]. BASH has been fortunate enough to have evolved in this manner, resulting in its current position as a thriving self-help organization [4].

REFERENCES

1. Evans G. The Family Circle guide to self-help. New York: Ballantine Books, 1979: 198-199, 224.
2. Katz AH, Bender EI. The strength in us: Self-help groups in the modern world. New York: New Viewpoints, 1976: 113-114.
3. Weber GH. Self-help and beliefs. In: Weber GH, Cohen LM, eds. Beliefs and self-help. New York: Human Sciences Press Inc, 1982: 13-30.
4. Rubel JR. The function of self-help groups in recovery for anorexia and bulimia. In: Larocca FEF, ed. Psychiatric clinics of North America: Symposium on eating disorders. 1984; 7:2.
5. Larocca FEF, Kolodny NJ. A facilitator's training manual: A primer: The BASH approach. St. Louis: Midwest Medical Publications, 1983.
6. Larocca FEF. The relevance of self-help in the management of anorexia and bulimia. Res Medica (medical magazine published by St. John's Mercy Medical Center, St. Louis, Missouri, USA) 1983; 1:16-9.
7. Fauri DP. The use of self-help groups with persons and family members facing anorexia nervosa. BASH Newsletter 1983; 2:4.
8. Powell TJ. Self-help organizations and professional practice. Silverspring, Maryland: National Association of Social Workers, 1987: 11.
9. Mowrer OH. Integrity groups: Basic principles and procedures. Couns Psych 1972; 2:7-33.
10. Wood M. The road to mental recovery. Aust Fam Phys 1981; 10:858-859.
11. Borman LD. Characteristics of development and growth. In: Lieberman MA, Borman LD, eds. Self-help groups for coping with crisis. San Francisco: Jossey-Bass, 1979: 22-23.
12. Pitch L. What a show. Health Visitor 1981; 8:21.
13. Tapia F. Self-help on an individual basis. In: Larocca FEF, editor. Eating disorders: Effective care and treatment. St. Louis: Isiyakyu EuroAmerica Inc, 1986: 247-257.
14. Lieber LL. Mothers anonymous: New directions against child abuse. Paper presented at the First Biennial Conference of the Society for Clinical Social Work, San Francisco, 1971.
15. Gussow A, Tracy GS. The role of self-help clubs in adaption to chronic illness and disability. Soc Science and Med 1976; 10:407-14.
16. Parents Anonymous Frontiers, Torrance, California, Parents Anonymous, 1979.
17. Borman LD. Characteristics of development and growth. In: Lieberman MA, Borman LD, eds. Self-help for coping with crisis. San Francisco: Jossey-Bass, 1979:13-42.
18. Gartner A. Self-help and mental health. Soc Pol 1976; 7:20-40.
19. Larocca FEF, Stern J. Eating disorders: Self-help and treatment in Missouri. Mo Med 1984; 81(12):764-73.
20. Gartner A, Reissman F. The self-help revolution, vol. X. New York: Human Services Inc., 1984: 243-248.
21. Rosenblatt A, Mayer JE. Help-seeking for family problems: A survey of utilization and satisfaction. Am J of Psych 1972; 28:126-30.
22. Wollert RW, Levy LH, Knight BG. Help giving in behavioral control and stress coping self-help groups. Sm Gr Beh 1982; 13:204-18.
23. Frane CC, Knight B, Levy LH, et al. Self-help groups: The members' perspectives Am J of Comm Psych 1980; 8:53-65.
24. Levy LH. Self-help groups viewed by mental health professionals: A survey and comments. Am J of Comm Psych 1973; 6:305-15.
25. Lieberman MA. Help seeking and self-help groups. In: Lieberman MA, Borman LP, eds. Self-help groups for coping with crisis. San Francisco: Jossey-Bass, 1979:116-150.
26. Sidel VW, Sidel R. Beyond coping. Soc Pol 1976; 7:67-70.
27. Riessman F. The helper therapy principle. Social Work, April 1965; 10:27.
28. Levy LH. Self-help groups: Types and psychological processes. J of App Beh Science 1976; 12:310-22.
29. Levy LH. Process and activities in groups. In: Lieberman MA, Borman LD, eds. Self-help groups for coping with crisis. San Francisco: Jossey-Bass, 1979: 234-271.
30. Hurvitz N. Characteristics of orthodox (professional) psychotherapy and self-help group therapy conference. Sixty-Sixth Annual Conference of the American Psychological Association, San Francisco, California, September 1968.
31. Larocca FEF, Kolodny NJ. Treating depression in adolescence: The psychiatric and social work connection. In: Munoz RA, ed. New directions for mental health services. San Francisco: Jossey-Bass, 1984: 51-8.
32. Lieberman MA, Bond GR. Self-help groups: Problems and measuring outcome. Sm Gr Beh 1978; 9:221-41.
33. Mantell JE, Alexander ES, Kleiman MA. Social work and self-help groups. Health and Soc Work 1976; 1:80-100.
34. Larocca FEF, Goodner SA. Eating disorders and self-help revisited. Clinical Psychiatric Quarterly 1987; 10(2):6-9.
35. Fink PJ. Mental illness awareness week (editorial). American Journal of Psychiatry 1987; 144(10):1298, 1300.
36. Larocca FEF. Tower of babel—a therapist registry (editorial). BASH Newsletter 1983; 2(1):3.

