

Chapter 38

Learning and Behavioral Approaches to the Treatment of Anorexia Nervosa and Bulimia

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INTRODUCTION

Many signs and symptoms observed in patients with anorexia nervosa resemble those seen in non-self-induced starvation states; namely, personality changes, difficulty with concentration, memory disturbances, irritability, and apathy [1,2]. Indeed, actual cerebral atrophy may occur in the anorexia nervosa patient [3].

Accordingly, many anorexia nervosa patients in the initial stages of treatment do not respond or appreciate the traditional forms of insight-oriented psychotherapeutic interventions [4]. Active treatment is necessary. While parenteral nutrition (or enteral intubation techniques) may be indicated in life-threatening circumstances, more commonly (structured) behavioral methods may be useful when applied in a hospital setting where observation, measurement, and stepwise informal feedback is possible. The purpose of this active treatment is to physically prepare the patient for a more comprehensive psychiatric treatment program, which might include individual dynamic psychotherapy, group therapy, and family therapy.

HISTORICAL BACKGROUND AND DEFINITIONS

The behavioral approach to psychiatry has its origin in experimental animal studies exemplified by the works of Thorndike and Pavlov [5]. Emphasis is placed on ob-

servable behavioral events and related antecedent and consequent conditions and responses.

In the strictest sense, behavior can be defined as an individual's skeletal muscle activity, which includes verbal expression [5]. Overt behavior is considered to be events that are directly observable. Relevant examples might include vomiting, amount of time spent exercising, time spent eating, food choices, weight change, etc. This is to be distinguished from covert behaviors, which, while still observable, require indirect techniques. This includes affective and cognitive processes that could be indirectly observed by body posture, limb movements, or facial expressions. For example, clenched fists might indicate the cognitive (covert) behavior of tension or anxiety, and crying could similarly imply the affective behavior of sadness. Inferring the behavior by indirect observations, of course, would increase the possibility of error. Observed crying, while often signifying sadness, could also imply tears of joy—for example, at a wedding. Thus, a degree of caution is advised to discourage overgeneralization.

The concept of "behavior therapy" is more difficult to define, because different investigators have used it in a variety of ways. Some feel there is no universal definition of this term [6], but in general it can be considered as a group of treatment techniques based to some extent on learning theory, conditioning techniques, and empirical evaluation of results. We will describe four dis-

tinct subsections of behavior therapy, although the distinction often blurs in clinical application.

1. Classical conditioning, also referred to as respondent conditioning, involves the pairing of previously neutral stimuli with an innate response evoked by an unconditioned stimulus. The subject eventually "learns" to react to the neutral stimulus with the same response (now termed conditioned response) in the absence of the unconditioned stimulus. Clinical applications include systematic desensitization, flooding, and graded exposure.

2. Operant (instrumental) conditioning is based on work by Thorndike and B.F. Skinner [5]. It essentially consists of selectively rewarding and/or punishing specified "target behavior" to increase or decrease, respectively, their frequency of emission by the organism. This "shaping" of desired behaviors is often referred to as "behavior modification," although the authors feel this definition is too restrictive.

3. Cognitive behavioral conditioning was described by Beck [7,8] and operates under the premise that cognitions (ie, thoughts, belief systems) precede and are causally related to feelings, mood, and behavior. Therapeutic intervention would be aimed at modifying the antecedent thought to effect clinical change.

4. Social learning theory is based on vicarious experiences causing change in an individual's behavior. This concept, also referred to as "modeling" [5], suggests that a subject learns a behavior by observing another person engaged in that behavior and seeing whether the observed person is rewarded (or punished).

Behavior therapy in the mental health setting is generally recognized as having begun with Ayllon et al in 1964 [9]. They used attention by staff to modify eating behavior in chronic schizophrenic inpatients. Ward observers had noted that patients who were unwilling or unable to eat received increased staff attention. It was hypothesized that this increased attention was a positive social reinforcer. By changing the contingencies (ie, staff was subsequently instructed to simply ignore problem eaters), problem eating became unrewarding and decreased significantly. In other words, the target behavior (poor eating) was extinguished by withdrawal of reinforcement (social attention).

Note should be taken, however, that when Leitenberg et al (1968) tried this strategy in 1968 in patients with anorexia nervosa, it was ineffective [10].

SPECIFIC STUDIES

Behavioral techniques applied to eating disorders began in the 1960s. Hallsten and Lang in 1965 reported single case studies of female anorexics using systematic

desensitization to decrease both fears of weight gain and feelings of self-consciousness [11,12]. Although there were numerous methodological flaws, both authors reported some success.

Also in 1965, Bachrach et al used socialization (ie, attention and praise by staff) as a reward in an operant conditioning paradigm to reinforce improved eating behavior in a 37-year-old anorectic patient [13]. The patient gained weight initially, but this soon leveled off. At this point the contingency was changed to weight gain (as opposed to eating) when the possibility of secretive vomiting became apparent. The patient then resumed increasing her weight and was discharged after a two-month period, having gained approximately 17 lbs.

Leitenberg et al essentially repeated this procedure using two patients and reported similar success [10]. They attempted to refine the methodology by having the patients individually be their own controls, measuring weight gain in reinforcement versus non-reinforcement phases, but results were not clear-cut. Although both patients gained significant amounts of weight during reinforcement, one patient continued to show weight increase after reinforcement was stopped—thus the predicted extinction effect did not occur.

In 1970, Blinder et al studied six hospitalized anorectic patients and applied operant conditioning using access to activity contingent on weight gain [14]. Noting the patients' proclivity toward motor hyperactivity during the initial observation period, they were given six hours of unrestricted access to physical activity on any day that weight increased 1/2 lb over the previous day. The first three patients gained a mean of 4.8 lbs. per week during the initial three weeks after behavior therapy was started. As they approached more normal proportions, their rate of increase decreased and averaged 3.9 lbs. per week for the whole treatment period. Medication and limited psychotherapy were also used during this period.

A fourth patient did not respond to limited activity as a reinforcer but did complain about the sedative side-effects of her medication (chlorpromazine). This suggested a new source of reinforcement—decreasing dosage contingent on weight gain—and this was successfully used. The fifth and sixth patients were treated with the different contingencies (an initial 6-lbs. weight gain and then a further 10-lbs. increase before hospital discharge) while using the same reinforcements as patients one, two, and three (activity). This paradigm was also successful for short-term weight gain.

The significance of this study is that reinforcers were specifically tailored to individual patients.

In 1975, Brady and Rieger reported a 1972 University of Pennsylvania study involving 16 anorectic

patients treated with operant conditioning combined with other treatment modalities (medication, group therapy, occupational therapy, etc) [15]. Behavior analysis was conducted during an initial four- to five-day observation period, and reinforcers were individualized for each patient. These included increased activity privileges, access to textbooks, personal hygiene materials, other patients for socialization, and reduction of medication dosage. Reinforcement was given daily, contingent on a weight increase of 1/2 lb. All patients gained weight, averaging 4.07 lbs. per week. This study is significant for more refined diagnostic criteria, including greater than a 30% weight loss and amenorrhea greater than six months in female patients. However, the authors note the absence of exclusion criteria and the possibility that two schizophrenics were part of the sample. The study is also notable for a relatively long-term follow-up in a fairly large sample, ranging from four to five months after discharge. The results were extremely variable and included two deaths and several other patients who "did poorly after discharge."

Pertschuk, continuing the work of Brady and Rieger, reported a follow-up study of 29 patients, 27 of whom were treated with operant conditioning for weight gain in addition to medications [16]. Behavior therapy was shown effective in 25 of the 27 patients. The study, ranging from 3 to 45 months after discharge, was also significant for the finding that bulimia developed in ten patients who did not report this as a problem on admission.

Agras et al reported a 1974 study using five single case studies to investigate the relative importance of three variables on weight gain using an A-B-A design [17]. They were reinforcement, size of meals, and informational feedback.

Reinforcement of daily weight gain was proved effective as in prior studies, but they also noted that weight continued to increase during nonreinforcement phases—although at a decreased rate. At this point, the hypothesis of hospitalization as a negative reinforcer was introduced. This was tested by having the patient remain for a 12-week period, regardless of weight change, to remove the "discharge contingent on weight gain" effect. As predicted, the rate of weight increase declined, and food intake greatly declined.

Informational feedback consisted of the patient receiving exact information regarding the number of calories consumed and mouthfuls of food taken after each meal plus exact body weight every morning. Under conditions of informational feedback plus reinforcement versus reinforcement alone, weight clearly improved under the former.

Size of meal presented versus weight gain was also tested using a 3,000-calorie-per-day diet versus 6,000

calories, but only a weak treatment effect was noted.

While not demonstrated definitively, the authors felt that informational feedback was more important than positive reinforcement, but that combining all three variables was the most efficacious.

Agras and Werne published a subsequent report in 1977 on the first 25 patients treated at Stanford University [18]. They used a similar protocol and noted that only three of the 25 patients did not gain weight at discharge.

Bhanji and Thompson [19] studied 11 anorectic patients using operant conditioning often combined with medications. Patients were allowed to specify their own hierarchy of reinforcers, and these were made contingent on progressively more stringent criteria—initially a complete meal but later modified to account for actual weight gain. All but one patient gained significant weight at discharge. Long-term (2 to 27 months) follow-up was attempted using mailed questionnaires, but only seven were returned, and only three contained a weight update. The authors concluded that, based on limited follow-up, operant techniques were useful for initial weight gain, but were inadequate for long-term maintenance of normal eating and weight. However, it is unclear whether operant techniques were adequately maintained after discharge.

Parker et al (1977) reported on ten anorectic females treated with a combination of psychotherapy and operant conditioning, and in some cases medication [20]. Reinforcement consisted of giving the patient a variable number of chips contingent on both cooperation with staff (which was individualized) and weight gain. For example, one chip was given for simply coming to weigh-in, one for maintaining weight, and ten for each 1/2 lb gained. These could be used to "purchase" passes, social activities, and other reinforcements that the patient selected. Additionally, the patients received novelty gifts at 1-lb weight-gain intervals. All patients are reported to have gained weight during hospitalization. The authors also emphasized the need to educate the staff that (1) a behavioral approach to therapy did not imply a totalitarian ideology, and (2) any comment on a patient's eating behavior (as opposed to weight) was unproductive.

In 1977 Garfinkel et al studied 42 inpatients and outpatients [21]. Of the 26 inpatients, 17 received operant conditioning with other therapies and nine received only other therapies. No outpatient received behavior therapy. All patients were followed a minimum of one year after discharge. Results showed that patients treated with behavioral methods were statistically similar in final weight to the remainder of the group. The authors also attempted to design a scale, the "Global Clinical Score," to measure parameters other than weight. They concluded that behavior therapy, while not superior to

other methods, was also not harmful, as had been implied [22].

In 1979 Eckert et al [23] did a randomized prospective study involving 81 patients assigned to either behavioral or nonbehavioral treatment. The behavioral program was similar to prior protocols using privileges to reinforce weight gain. However, reinforcement was not individualized and was given in five-day increments. Both groups received individual psychotherapy, and half the patients in each group also received cyproheptadine. Results showed no difference in weight gain over a 35-day period between the behavior versus nonbehavior groups. In our opinion, this would tend to underscore the need for individualizing reinforcement and suggests there may be a more optimal frequency of reinforcement. The authors also attempted to identify specific subgroups of patients who would benefit more from behavioral treatment by using psychiatric and social history pretreatment variables. Although no variable was statistically significant, it appeared that patients with little or no prior outpatient treatment gained more weight with behavioral treatment. Possibly more naive patients may be more responsive and less resistant to structure.

A large study was reported by Pierloot et al [24] using a group of 145 patients with a minimum one-year follow-up on 88 of them during 1967 to 1969. Patients were generally treated with operant conditioning for weight gain along with other treatment modalities. An unspecified number of "early patients" received "enforced feeding" and medications—a protocol later discontinued.

Of the follow-up group, 11.4% died, including four suicides and four deaths secondary to malnutrition. Using the "Global Clinical Score" of Garfinkel et al [21], the authors described only one third as having fully recovered. Because of the 49% drop-out rate, it is difficult to assess from this study the effectiveness or dangers of behavioral treatment.

Reese and Gross published a 1982 report in support of operant conditioning with positive reinforcement to facilitate weight gain [25]. Rewards were individualized and both patient and staff agreed on a behavior contract. An interesting variation of informational feedback was used. The patient's daily weight was visually displayed on a graph, along with a second line indicating weight expectations to gain privileges. This was designed to decrease confrontation with staff and give the patient an increased sense of control, and it also appeared to be effective.

Halmi [26] proposed another variation in treating anorectic patients with operant methods, using a general hospital instead of a psychiatric setting. The stated

advantages included immediate access to medical treatment—which is often necessary for patients severely emaciated and/or engaged in self-induced vomiting or laxative abuse. Additionally, there was a postulated increase in willingness of patients to enter "medical treatment" as opposed to "psychiatric" due to a presumed decrease in denial regarding a "medical condition." The author's protocol included restriction to bed until a target weight is achieved and use of bedpans (instead of bathroom to reduce surreptitious food disposal) until normal weight is achieved along with other less-severe deprivation. The goal is immediate restoration of weight to enable the patient to be more available to more definitive psychiatric treatment at a later date. Results of this variation are not clear-cut at the present time.

In addition to standard operant techniques aimed at promoting weight gain, some authors have suggested additional behavioral strategies to deal with the cognitive distortions and irrational beliefs in anorexia nervosa and bulimia [11,27,28].

For example, fear of weight gain or eating certain foods has been conceptualized as similar to a phobia and vomiting as similar to an obsessive-compulsive ritual.

In 1976, Williams treated six anorectic patients with a multifaceted behavioral approach [29]. In addition to reinforcement of weight gain, he also attempted to systematically desensitize patients to fears of putting on weight, disapproval by others, eating, and sexual activities. He also used aversion techniques (mild electrical shocks) to decrease pleasure at being thin and engaging in abnormal eating, along with other "miscellaneous techniques." No patient gained weight and the author described the overall results as a "resounding failure."

Perhaps trying to desensitize so many variables resulted in desensitization to the whole program. The study is, however, significant for its novelty, and it probably served as a stimulus for refinement of the technique by subsequent investigators.

In 1977 Monti et al reported a single case study of an anorectic female with bulimic traits [30]. The treatment consisted of standard positive reinforcement for weight gain using contingency contracting and feedback. In addition, a behavioral analysis revealed "maladaptive thoughts" (feeling she was a "bad person"), which often preceded bulimic episodes. These were treated with deep muscle relaxation and systematic desensitization. Both reinforcement and desensitization were continued on a weekly outpatient basis. Results showed that the patient gained a significant amount of weight during the inpatient phase, and this continued to a lesser extent during the six-month outpatient follow-up. Unfortunately, the results are confounded by development of edema early in treatment. Because of the nature of the

experimental design, it was not possible to assess the effect of adding desensitization to other treatment modalities.

In 1982 Mavissakalian reported a study of two anorectic patients [27]. Inpatient treatment used procedures specifically aimed at reducing the apparent "weight phobia" and "ritualistic exercising" components of the illness in addition to the more common behavioral methods of reinforcing weight gain based on Agras and Werne [18]. Compulsive exercise was decreased by using response prevention by requiring the patients to spend one hour resting after each 30-minute meal period. Thus, the patient was prevented from neutralizing (ie, "undoing" by exercise) the obsessively feared consequences of eating—weight gain. The investigator also noted that this had the effect of exposing the patient to phobic stimuli (food, weight gain, feelings of fullness) for 90 minutes per session. During a 30-day treatment period, this "...exceeds the 20-30 hours of programmed exposure required in the successful treatment of phobias and obsessive-compulsive behavior."

Both patients gained significant amounts of weight during hospitalization. Modified behavioral treatment continued by family members for the patients after discharge. Weight was not only maintained during the three- to five-month follow-up, but increased. Both patients became overweight without development of bulimia. The reasons for them becoming overweight are unclear. Perhaps this was an exaggerated response to behavior therapy. The author suggests chance and notes this occurs in an approximate 5% of anorectics. But he also calls for further study of behavioral treatment aimed at the special nature of the phobic and obsessive-compulsive components in anorexia nervosa. He states that patients with a simple phobic or obsessive-compulsive disorder are aware of the irrationality of their behavior, whereas anorectics usually deny this.

Cinciripini et al [28] reported on two anorectic patients with bulimic traits in a 1983 study designed to deal with cognitive aspects of the illness via behavioral methods in addition to operant conditioning for weight gain. After an initial four-day baseline period, patient #1 was put on a contingency management program for weight gain and continued to self-monitor urges and episodes of bingeing and emesis. After day 25, thought stopping was added to provide a cognitive control for these urges. This procedure used ten 60-minute sessions consisting of slides depicting binge behavior with the patient instructed to signal the therapist when the scenes became vivid and distressing. The therapist would then present a loud (distracting) noise with the word "stop" and instruct the patient to repeat "stop" to herself while imagining a relaxing scene after the binge urge declined.

After 28 days, flooding was added to the protocol for

a two-week period. This consisted of (1) having the patient taste, smell, and handle various "binge foods" while the therapist vividly described the bulimic act; (2) exposing the patient to binge items both before and after meals in the hospital; and (3) exposing the patient to binge items in the community (restaurants and supermarkets) both with and later without supervision.

Results showed that (1) patient #1 gained weight with reinforcement alone, and binge episodes stopped during this phase; (2) with addition of thought stopping, weight continued to increase, but both binge and vomiting episodes also increased while the frequency of these urges declined; and (3) toward the end of flooding and through discharge, binge and vomiting episodes were gradually eliminated, while some vomiting urges continued to be reported. Over a two-year follow-up period, weight was maintained. Although some binge activity was reported, brief outpatient therapy using flooding techniques "successfully suppressed immediate binge activity."

In patient #2, therapy consisted mainly of reinforcement for weight gain and response prevention for vomiting. Response prevention eliminated both emesis urges and episodes and these remained extinguished after this aspect of treatment was discontinued. The patient successfully achieved weight gain at discharge but required brief rehospitalization two months later after losing 10 lbs. and resuming emesis. She was discharged after two weeks and had maintained ideal weight at one-year follow-up. After two years, the patient reported by telephone a slight weight decrease but no vomiting and was "functioning well."

The authors concluded that contingency management, flooding, and response prevention but not thought stopping were effective.

Goldberg et al [31] studied a group of 105 female anorectic inpatients to evaluate treatment effects on attitudinal dimensions of the illness in addition to the more common weight gain/calorie intake criteria. Prior to assignment to a given treatment mode (eg, cyproheptadine, behavior modification, etc), patients responded to 63 attitude statements using a four-point scale ranging from agreement to disagreement. Initially hypothesized significant attitudinal dimensions included: denial of illness, loss of appetite, manipulativeness, ambivalence toward parents, fear of fatness, high achievement goals, cooking as a hobby, decreased sexual desire, and subjective feeling of hypothermia.

A factor analysis of the 63 items yielded 19 factors accounting for 76.6% of the variance. Of these, 15 were considered interpretable and 11 were deemed useful as prognostic indicators. These included: feeling exploited by staff, fear of fatness, parents at fault, denial of illness, appetite loss, hypothermia, poor self-care, efforts re-

quired for achievement goals, the illness is due to mysterious physical causes, hobby cooking, and decreased sexual interest.

Attitudes useful as prognostic indicators of weight restoration included: less fear of fatness, less denial of illness, and a lesser feeling that food is sickening to the stomach. Behavior modification appeared to decrease fear of fatness, decrease interest in cooking, and make the patient feel that decreased effort is needed for achievement.

Although the identification of a given factor in an analysis does not automatically imply validity and reliability, this study is significant for its demonstration that attitudes can be prognostic indicators of weight gain and useful measurements of behavioral treatment effects. The evaluation of pretreatment variables may also be important in individualizing behavioral strategies in future studies.

In a 1982 study Fichter et al [32] examined attitude change (along with other variables) in 24 anorectic inpatients, 21 of which received behavior therapy consisting of reinforcement or systematic desensitization. Attitudes and "anorectic behavior" were measured by having patients use the "Anorexia Nervosa Inventory for Self-Rating" [32].

Weight increased steadily during the reinforcement phase to above 80% of ideal body weight on the average. Specific "anorectic attitudes" (figure consciousness, adverse effect of meals, and bulimia) seemed to change significantly, but not those of a "general neurotic" nature (feelings of insufficiency, obsessional traits, and sexual anxieties). While neither Fichter nor Goldberg attempted to actively change attitudes as part of their treatment program (ie, cognitive therapy), it would seem a reasonable course of action in future trials in view of the previously described beneficial changes in attitude secondary to "traditional" treatment methods.

In 1982 Garner and Bemis [33] published a basic set of guidelines for cognitive behavioral treatment of anorexia nervosa based on Beck's model for cognitive treatment of depression. They emphasized the finding of persistent "distorted attitudes and beliefs" even in weight-recovered anorexia nervosa patients and suggested a theoretical model involving the role of cognitions in the development of the illness.

Their treatment protocol emphasizes (1) the need to develop motivation for treatment in these patients (compared with patients with depression), (2) the need for patient education on both the illness and the theory of cognitive therapy, and (3) the need for early weight stabilization using behavioral methods that include role playing, scheduling pleasant events to enhance the set

of reinforcers beyond the pleasures of weight loss, diversion techniques (eg, response prevention to decrease bulimic behavior), social skills training, desensitization to phobic-like responses to eating and/or weight gain, and "standard operant technology" to reinforce desirable behavior.

Cognitive techniques are described separately but in reality are difficult to separate in an effective treatment program. The idea is to teach patients to examine the validity of their belief systems on a moment-to-moment basis. The therapist is then in a position to introduce the element of doubt regarding the basic (distorted) beliefs in a nonjudgemental fashion. To facilitate this process, the authors suggested several procedures including (1) operationalizing beliefs as precisely as possible, (2) de-centering (ie, ask, "Do your rules apply to others as well as yourself?"), (3) "What if" technique to reduce the imagined consequences of feared events, and (4) evaluation of automatic thoughts by teaching patients to ask themselves: What is the evidence, what are alternative explanations, and what are the realistic consequences if the analysis is correct? Patients could then test their conclusions and expectations in an ongoing manner. In addition, underlying assumptions (ie, the principles that organize irrational beliefs) need to be identified to enable modification using many of the above techniques.

The authors conclude that cognitive therapy is compatible with other treatment modalities, that the affective component of the illness must be addressed during treatment (but not necessarily at the beginning), and by stating the need for systematic studies to support or refute their treatment approach.

The behavioral treatment of bulimia as an isolated entity (ie, without anorexia nervosa or other psychopathology) has received much less attention in the literature. Factors related to this include a blurring of diagnostic criteria with anorexia nervosa in the past and the fact that the mortality rate for bulimia is much lower than that reported for anorexia nervosa.

In 1982 Rosen and Leitenberg reported on a single case of "bulimia nervosa" (defined as bingeing, self-induced vomiting, and fear of weight gain in a normal-weight person) using behavioral methods [34]. They hypothesized that the binge/purge cycle was linked by a "vicious circle of anxiety" regarding weight gain. Eating was seen as the cause of anxiety and purging was the anxiety reducer. Since reduction of anxiety is self-reinforcing, the treatment was aimed at reducing the purging behavior, which the author hypothesized would then lead to extinction of binge eating and fear of weight gain.

The protocol included obtaining a history of overeating behavior: types of food, amounts eaten, duration of

episodes, and also subjective feelings following an episode. Such feelings included feeling bloated, lightness around the mouth, worry over poor health, and "contamination" from food consumed. The next step consisted of exposing the patient to the "problem foods" under staff supervision and preventing the typical response (vomiting). The patient was given a detailed explanation of the treatment program and was instructed to eat an amount of food sufficient to cause vomiting urges during each session—knowing she would not be allowed to vomit. The therapist would help the patient focus on her subjective discomfort until the urge to vomit disappeared. The duration of the vomiting urge initially was greater than 90 minutes but decreased over time as did anxiety ratings. After this phase, the patient was then instructed to follow a schedule at home of gradually increasing the number of days per week with no vomiting. This part of the treatment lasted 44 days, and the rate of vomiting decreased from 3 to 1.25 times per day. During ten months of follow-up, there was only one episode of vomiting. Of interest, the patient also eventually stopped bingeing—without explicit instructions by the therapist to do so. It is also noted that the patient reported that the urge to vomit after eating was never eliminated but rather was described as much less distressing and of shorter duration.

Long and Cordle [35] reported on two patients with bulimia successfully treated with behavioral techniques on an outpatient basis. After a two-week baseline assessment, with patients keeping a daily record of food intake and vomiting, one-hour weekly sessions were started. Treatment concentrated on four main areas: (1) behavioral self-control, (2) dietary education, (3) cognitive restructuring, and (4) resocialization. The behavioral methods included self-monitoring of binge/purge episodes, self-targeting of frequency of binge/purge episodes, visual display of progress on a graph, positive verbal reinforcement by therapist, relaxation training, and response prevention (ie, increasing intervals after eating without vomiting). The cognitive restructuring included "cognitive modeling of covert assertion and thought management exercises."

Patient "A" required 40 sessions over ten months, and patient "B" required 12 sessions over three months. At the end of treatment, both patients were able to eat "normal" meals without vomiting or laxative use and were less preoccupied with their own body weight. Follow-up at nine months for patient "A" showed improvement was maintained. Patient "B" at 12 months described only two episodes of induced vomiting. The length of illness may be a crucial factor—the longer vomiting exists and the increasing frequency over years may make it a secondary autonomous behavior, which is reinforcing and will not disappear without special ef-

fort.

Fairburn [36] reported on a group of 11 patients with "bulimia nervosa." This included six patients with a history suggestive of anorexia nervosa, which complicates interpretation of the study. Patients were treated on an outpatient basis in two phases. The first part included self-monitoring, restricting eating to meal times only, and setting limited goals at the end of each session. This generally was accomplished in eight weeks or less. In the second phase (with bingeing and vomiting now decreased to an intermittent frequency) antecedent conditions to loss of control over food were identified, with the therapist suggesting more adaptive coping mechanisms. Systematic desensitization was also employed to decrease anxiety over intake of "forbidden" (ie, high-calorie) foods.

Results showed nine patients decreased the frequency of bingeing and vomiting to less than one per month without significant change in body weight. The mean duration of treatment was seven months.

The authors noted (in contrast to Rosen and Leitenberg) that control over food intake seemed to be the important factor in the binge/purge cycle and that once this was managed it was not necessary to focus on the vomiting.

CRITICISMS AND CONTROVERSIES

There have been many critics of behavioral techniques applied to eating disorders since its inception. Bruch has long been a critic of behavior therapy and considers it damaging [22]. Indeed, death has been reported following an inpatient behavioral regimen [14]. But even Bruch feels that psychotherapy is ineffective until the malnourished patient can assimilate and process new information [37]. Blinder pointed out [14] that weight gain achieved in behavioral therapy should not be treated as equivalent to comprehensive treatment in the eating disorders; in fact, the very potency of behavioral techniques may be a hazard in leading to change in one clinical feature (weight) dissociated from other clinical improvement. Thus, while it is true that sometimes behaviorally oriented therapists focus on weight gain as the sole criterion for improvement, it need not necessarily be so, nor is it desirable.

Recently Touyz et al [38] compared a strict and lenient operant conditioning protocol for weight gain in 65 inpatients with anorexia nervosa and found no significant difference between them. However, they did find that in the lenient program the patients were better motivated and accessible to psychotherapy and other aspects of treatment. Behavior therapy should be viewed as but one aspect of the total treatment plan and is consistent with human concern [16].

Specific criticisms of studies in behavioral and cognitive therapy of eating disorders are shared by those in the basic sciences and medicine as a whole: (1) Do all the investigators use the same diagnostic criteria? (2) Is the selection of the sample and its size sufficient to discount bias? (3) Are other variables held constant so as not to contaminate the variable(s) being studied? (4) How is treatment effectiveness to be evaluated? (5) What is appropriate follow-up time? (6) Are the results reproducible?

Generally, many of the early investigators suffered from not consistently addressing these questions, but as the field has evolved, later studies have improved.

Accounting for appropriate variables and holding them constant is an often difficult and subtle exercise, but necessary to avoid spurious conclusions. For example, Goldberg et al [31] initially designed a study to evaluate the effects of behavior modification and a drug on weight gain in 105 patients with a diagnosis of anorexia nervosa. Three different hospitals participated, using the same protocol, and at first there appeared to be a positive correlation between weight gain and increasing milieu structure. The investigators point out, however, that when patient prognostic characteristics were used in the analysis of the data, an entirely different conclusion was reached. Their results showed that differences in weight gain among the hospitals were due to the kinds of patients admitted to the various hospitals rather than any differences among the hospitals themselves.

Studies with a small sample size are common in the literature and have been criticized. Bemis [39] points out that many single case studies haven't used an "A-B-A-B" design, and therefore, specific interventions cannot be assigned to successful results. For example, both Rosen et al and Fairburn report positive results stopping or greatly reducing bingeing and purging in a small number of patients [34,36]. However, Fairburn focused on food intake while Rosen felt vomiting was more important and similar to an obsessive-compulsive neurosis.

While such disparity precludes generalization, it may obliquely address the question of homogeneity in eating disorders. How discreet are the entities of anorexia nervosa and bulimia? Some investigators describe them as being linked with other psychiatric disorders [20,33,40]. Such a finding may have strong implications for behavior therapy. It seems reasonable that once a patient's specific problems other than anorexia nervosa or bulimia are identified, a behavior therapy protocol could be designed for them. Thus, the behavior therapy for an eating disorder patient with a phobic component might involve desensitization or flooding, and a patient

with a depressive component might receive cognitive therapy [33,39]. Specific treatment tailored to the individual patient and systematic long-term follow-up to evaluate such treatment are both clearly needed.

FUTURE DIRECTIONS

Traditionally, treatment of eating disorders has focused on patients and their immediate environment (eg, the hospital, family, etc) with little effort going to the enormous cultural pressures for thinness. As exemplified by the "Venus de Milo" and the paintings of Rubens, it is clear that the ideal cultural shape is ephemeral.

In 1980 Garner et al [41] quantified a shift toward a thinner cultural ideal for females over the last 20 years using data from Miss America Pageant contestants and Playboy centerfolds. They noted an increase in diet articles in six women's magazines during the same period and also an increase in population weight norms for young women using statistics from the Society of Actuaries. The authors also felt that these trends were linked to "the apparent increasing prevalence of anorexia nervosa and related eating disorders."

Designing an effective behavioral therapy program that addresses these issues might include desensitization or cognitive components awaits future investigation.

REFERENCES

1. Keys A, Brozek J, Henschel A, Mickelson O, Taylor HL. The biology of human starvation, (2 vols). Minneapolis: University of Minnesota Press, 1950;
2. McLaren DS. Nutrition and its disorders. New York: Churchill Livingstone, 1981;
3. Nussbaum M, Shenker R, Marc J, et al. Cerebral atrophy in anorexia nervosa. *Journal of Pediatrics* 96(5):867-9.
4. Bruch H. Anorexia nervosa: Therapy and theory. *Am J Psychiatry* 1982; 139:1531-8.
5. Brady JP. Behavior therapy. In: Kaplan HI, Freedman AM, Sadock FJ, eds. *Comprehensive textbook of psychiatry/III*. Baltimore: Williams and Wilkins, 1980;
6. O'Neill GW, Gardner R. Behavior therapy: An overview. *Hospital and Community Psychiatry* 1983; 34:709-15.
7. Beck AT. Thinking and depression. 2. Theory and therapy. *Arch Gen Psychiatry* 1964; 10:561.
8. Beck AT. Cognitive therapy: Nature and relation to behavior therapy. *Behav Ther* 1970; 1:184.
9. Ayllon T, Haughton E, Osmond HO. Chronic anorexia: A behavioral problem. *Canadian Psychiatric Association Journal* 1964; 9:147-54.
10. Leitenberg H, Agrad WS, Thompson LE. A sequential analysis of the effect of selective positive reinforce-

- ment in modifying anorexia nervosa. *Behaviour Research & Therapy* 1968; 6:211-8.
11. Hallsten EA. Adolescent anorexia nervosa treated by desensitization. *Behaviour Research & Therapy* 1965; 3:87-91.
 12. Lang PJ. Behavior therapy with a case of nervous anorexia. In: Ullmann LP, Krasner L, eds. *Case studies in behaviour modification*. New York: Holt, Rinehart & Winston, 1965:
 13. Bachrach AJ, Erwin SW, Mohr JP. The control of eating behavior in an anorexic by operant conditioning techniques. In: Ullmann LP, Krasner L, eds. *Case studies in behavior modification*. New York: Holt, Rinehart & Winston, 1965:
 14. Blinder BJ, Freeman DMA, Stunkard AJ. Behavior therapy of anorexia nervosa: Effectiveness of activity as a reinforcer of weight gain. *Amer J. Psychiatry* 1970; 126:1093-8.
 15. Brady JP, Rieger W. Behavioral treatment of anorexia nervosa. In: Thompson T, Dockens WS, eds. *Applications of behavior modification: International symposium on behavior and modification at Minneapolis, 1972*. New York: Academic Press, 1975:
 16. Pertschuk MJ. Behavior therapy: Extended follow-up in anorexia nervosa. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:
 17. Agras WS, Barlow DH, Chapin HN, et al. Behavior modification of anorexia nervosa. *Arch Gen Psychiatry* 1974; 30:279-86.
 18. Agras S, Werne J. Behavior modification in anorexia nervosa: Research foundations in anorexia nervosa. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:
 19. Bhanji S, Thomson J. Operant condition in the treatment of anorexia nervosa: A review & retrospective study of 11 cases. *Brit J Psychiatry* 1974; 124:166-72.
 20. Parker JB, Balzer D, Wyrick L. Anorexia nervosa: A combined therapeutic approach. *Southern Medical Journal* 1977; 70:448-52.
 21. Garfinkel PE, Moldofsky H, Garner DM. The outcome of anorexia nervosa: Significance of clinical features, body image & behavioral modification. In: Vigersky RA, ed. *Anorexia nervosa*. New York: Raven Press, 1977:
 22. Bruch H. Perils of behavior modification in treatment of anorexia nervosa. *JAMA* 1974; 230:1419-22.
 23. Eckert ED, Goldberg SC, Halmi KA, et al. Behavior therapy in anorexia. *Brit J Psychiatry* 1979; 134:55-9.
 24. Pierloot R, Vandereycken W, Verhaest S. An inpatient treatment program for anorexia nervosa patients. *Acta Psychiat Scand* 1982; 66:1-8.
 25. Reece BA, Gross M. A comprehensive milieu program for treatment In: Gross M, ed. *Anorexia nervosa, a comprehensive approach*. Collamore Press, 1982:
 26. Halmi KA. Anorexia nervosa: Treatment in the general hospital. *Current Psychiatric Therapies*, Vol 21, 1982: 181-184.
 27. Mavissakalian M. Anorexia nervosa treated with response prevention & prolonged exposure. *Behavior Research & Therapy*, 1982; 20:27-31.
 28. Cinciripini PM, Karnblith SJ, Turner SM et al. A behavioral program for the management of anorexia & bulimia. *The Journal of Nervous & Mental Disease* 1983; 171:186-9.
 29. Williams W. A comprehensive behavior modification programme for the treatment of anorexia nervosa: Results in six cases. *Australian & New Zealand Journal of Psychiatry* 1976; 10:321-4.
 30. Monti PM, McCrady BS, Barlow DH. Effect of positive reinforcement, informational feedback & contingency contracting on a bulimic anorexic female. *Behavior Therapy* 1977; 8:258-63,
 31. Goldberg SC, Halmi KA, Eckert ED, et al. Attitudinal dimensions in anorexia nervosa. *J Psychiat Res* 1980; 15:239-51.
 32. Fichter MM, Doerr P, Pirke KM, et al. Behavioral, attitude, nutrition & endocrinology in anorexia nervosa. *Acta Psychiat Scand* 1982; 66:429-44.
 33. Garner DM, Bemis KM. A cognitive-behavioral approach to anorexia nervosa. *Cognitive Therapy & Research* 1982; 6:123-50.
 34. Rosen JC, Leitenberg H. Bulimia nervosa: Treatment with exposure & response prevention. *Behavior Therapy* 1982; 13:117-24.
 35. Long CG, Cordle CJ. Psychological treatment of binge eating & self-induced vomiting. *British Journal of Medical Psychology* 1982; 55:139-55.
 36. Fairburn C. A cognitive behavioural approach to the treatment of bulimia. *Psychological Medicine* 1981; 11:707-11.
 37. Bruch H. Anorexia nervosa: Therapy & theory. *Am J Psychiatry* 1982; 139:1531-8.
 38. Touyz SW, Beumont PJV, Glaun D, et al. A comparison of lenient & strict operant conditioning programmes in refeeding patients with anorexia nervosa. *British Journal of Psychiatry* 1984; 144:517-20.
 39. Bemis KM. Current approaches to the etiology & treatment of anorexia nervosa. *Psychological Bulletin* 1984; 85:593-617.
 40. Herzog DB. Are anorexic & bulimic patients depressed? *Am J Psychiatry* 1984; 141:1594-7.
 41. Garner DM, Garfinkel PE, Schwartz D, et al. Cultural expectations of thinness in women. *Psychological Reports* 1980; 47:483-91.

