Chapter 24

The Diagnosis and Clinical Characteristics of Bulimia

James E. Mitchell and Richard L. Pyle

The DSM-III-R category of bulimia nervosa describes an eating disorder characterized by binge-eating episodes as well as other abnormal eating-related behaviors. The choice of the term bulimia to label this syndrome in the DSM-III was in a sense unfortunate, since the term bulimia is also used simply to indicate binge eating episodes. However, this syndrome encompasses far more than binge eating episodes. Patients with this disorder usually demonstrate markedly abnormal eating patterns and eat little or no food when they are not binge eating. They also frequently self-induce vomiting or abuse laxatives in an attempt to control their weight.

This chapter will begin by discussing and contrasting the diagnostic systems for bulimia and bulimia nervosa that are in widespread clinical use. We will then turn to a discussion of the behavioral characteristics of the bulimic eating pattern, the psychological and family characteristics of this group of patients, and the medical complications and laboratory abnormalities associated with bulimia nervosa.

DIAGNOSTIC ISSUES

Several different systems have been suggested to diagnose the clinical syndrome of bulimia nervosa or clinical syndromes that appear to closely resemble it. The two systems most commonly used are the DSM-III-R and a system originally proposed by Russell in 1979, which describes a syndrome with considerable clinical overlap [1]. The former system is used commonly in the United States and the latter system in England. Several other terms have been suggested to describe similar syndromes, including "bulimarexia" [2] and "the dietary chaos syndrome" [3]. However, most researchers and clinicians currently seem to favor bulimia nervosa.

The DSM-III criteria were recently revised, as follows:

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than two hours).
2. At least three of the following:
    i.   consumption of high calorie, easily ingested food during a binge;
    ii. inconspicuous eating during a binge;
    iii. termination of such eating episodes by abdominal pain, sleep, social interruption, or self-induced vomiting;
    iv. repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics or diuretics; and
    v.   frequent weight fluctuations greater than 10 lb because of alternating binges and fasts.
3. Awareness that the eating pattern is abnormal and fear of not being able to stop eating voluntarily.
4. Depressed mood and self-deprecating thoughts following eating binges.
5. The bulimic episodes are not due to anorexia nervosa or any known physical disorder.

An examination of the DSM-III criteria indicates quite clearly that the emphasis was on the binge-eating

behavior itself. Such related problems as self-induced vomiting and laxative abuse are mentioned, but were not required for the diagnosis.

The syndrome of bulimia nervosa as originally described by Russell is diagnosed using the following criteria [1]:

1. The patient suffers from powerful and intractable urges to overeat.
2. The patient seeks to avoid "fattening" affects of food by inducing vomiting or abusing purgatives or both.
3. The patient has a morbid fear of becoming obese.

The criteria differ significantly from the DSM-III bulimia criteria in several ways, the most important of which may be that the presence of self-induced vomiting or laxative abuse is required for the diagnosis. The inclusion of the requirement for self-induced vomiting and/or laxative abuse in the bulimia nervosa criteria indicates that a more selective sample of patients will be identified. This is particularly important in view of research that has demonstrated that binge eating is a fairly common behavior in the general population [4-11]. The DSM-III bulimia criteria, when broadly interpreted, may therefore identify many individuals in the general population. The bulimia nervosa criteria of Russell probably are less likely to do so. Another essential point is that neither of these sets of criteria include frequency parameters. Since is is known that experimentation with bulimic behavior is common in young women [5-7], the lack of a frequency criterion suggests that people without clinically significant eating problems could be identified by either set of criteria. In defense of these criteria, it should be noted that both sets were proposed when little clinical work had been published concerning bulimic patients. Despite this, the authors showed considerable clinical accumen in delineating these syndromes.

In the recent revision of the DSM-III (DSM-III-R), bulimia is renamed bulmia nervosa, and the diagnostic criteria have been tightened up significantly, now including the following requirements:

1. Recurrent episodes of binge-eating (rapid consumption of a large amount of food in a discrete period of time).
2. A feeling of lack of control over eating behavior during the eating binges.
3. The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.
4. A minimum average of two binge-eating episodes a week for at least three months.
5. Persistent over concern about body shape and weight.

These criteria continue to emphasize binge-eating but now include a minimum frequency and duration of symptoms criterion. The last criterion beings the DSM-III-R into a closer alignment with the British criteria, and it appears that the DSM-III-R criteria will offer improved diagnostic guidepoints for clinicians, although they may be too broad for research purposes.

CLINICAL CHARACTERISTICS

Several series of patients with bulimia or bulimia nervosa have been reported in the literature. These series suggest that a variety of problems occur in these patients, some involving eating behavior and some involving other areas [1,12-19].

Available studies indicate that the usual age of onset is between 16 and 19 years of age [13,15,19]. Most patients are ill for several years before seeking treatment; the average age of first treatment contact is about age 24 [14-19]. Although bulimia and bulimia nervosa are primarily problems of young women, we have seen women in their 40s or 50s with this disorder who have been actively bulimic for more than 20 years. Less than 10% of the cases reported in the literature have been male [1,19].

As indicated previously, various abnormal eating-related behaviors have been reported by patients with bulimia [14-17,19]. The hallmark of the illness is binge eating, which is required for the diagnosis. Published frequency data indicate that most patients with bulimia binge eat once a day or more often [14-17,19]. The details concerning binge-eating episodes will be discussed later. Self-induced vomiting is also a prevalent and frequent behavior in this patient population. Prevalence data indicate that 70% to 95% of bulimic patients practice self-induced vomiting on a regular basis, and frequency data indicate that most who self-induce vomiting do so once a day or more often [14,16,17,19]. Another abnormal eating-related behavior commonly reported by these patients is laxative abuse [14,16,17,19]. Although this problem has been less intensively studied, published reports indicate that between 15% and 60% of patients with bulimia abuse laxatives in an attempt to rid themselves of excess food or to lose weight. A significant minority, perhaps as many as 20%, abuse laxatives on a daily basis [13,17,19]. The amounts of laxatives ingested are usually in excess of recommended dosage. Some patients who have become tolerant to the effects use amounts several hundred times the recommended dose.

A variety of other abnormal eating-related behaviors have been described in patients with bulimia, including diuretic abuse (33.1%), excessive use of enemas (7%),

and chewing and splitting out food without swallowing it to prevent weight gain (64.5%). Although these behaviors have not been intensively studied, they are common enough to be considered as characteristics of the syndrome in some patients.

Meal intake patterns have also been studied in patients with bulimia. Most bulimic patients do not eat normally when they are not binge eating [14,15]. Their meal pattern is usually characterized by fasting or minimal food intake between binge-eating episodes. When these patients fast they become hungry, which may serve as a powerful stimulus for the next binge-eating episode. Therefore, in most cases the bulimic eating patterns should best be characterized as a pattern of alternating binge eating and fasting.

Our problem in discussing bulimia and bulimia nervosa concerns the definition, or the lack of definition, as to what actually constitutes a binge-eating episode or eating binge. DSM-III-R criteria suggest that binge eating involves the ingestion of a large amount of food, and available data indicates that patients usually do eat large amounts of food when binge eating [15,20]. Russell originally reported that some patients consumed between 5,000 and 20,000 calories during a binge-eating episode [1]. Other researchers have substantiated this finding [14,5,19,20]. However, not every patient with bulimia nervosa eats such large amounts of food during a binge-eating episode. A few patients, if asked to describe their behavior in detail, actually report an amount that many people would consider only slightly in excess of a normal meal. Patients tend to consume high-carbohydrate or high-fat foods during binge-eating episodes. Items such as ice cream, candy, bread, and doughnuts are commonly ingested [20,21]. These food choices are particularly interesting in view of the fact that these patients usually avoid such foods at other times because they consider them fattening. They eat them only when they are planning to "get rid" of the food through vomiting or laxative abuse. Binge-eating episodes are usually pursued in isolation. Although they can be temporally related to stressful life events, such as problems at work or interpersonal difficulties, for many patients binge eating eventually becomes an institutionalized part of the daily routine [13,15,21]. An example would be a bulimic individual who each day stops at a store on the way home from work, buys the foods necessary for binge eating, goes home, consumes the food over the period of an hour or so, and then self-induces vomiting. Although stress at work may have initially contributed to the development of this pattern, the pattern may continue even if the stress is no longer active. As this scenario suggests, binge-eating episodes tend to take place late in the day when individuals return home from work or school. Many patients state that they eat very rapidly when binge

eating and don't really taste the food. They may read, watch TV, or engage in other activities while eating.

How does this disorder begin? Little is known about the onset of bulimia nervosa. Some work has suggested a possible link between the onset of bulimia nervosa and traumatic events, but the retrospective nature of such data makes the validity of the finding open to question [14]. Several authors have noted that bulimia nervosa tends to begin during a period of dieting [14,17,21]. However, it must be remembered that most patients with bulimia nervosa are chronically concerned about their weight, and many may have been dieting off and on much of the time during adolescence. A history of anorexia nervosa is not uncommon in these patients, with various series reporting that 30% to 80% of patients with bulimia nervosa have a history of weight loss sufficient to qualify for such a diagnosis [1,13-19]. This finding, coupled with the finding that the symptom of bulimia is relatively common in patients with anorexia nervosa, suggest that these two disorders represent parts of a spectrum of eating problems.

Another important point when considering the pathogenesis of this disorder is the apparent increasing incidence of this disorder in our culture. Why does our culture seem to predispose to significant eating problems such as anorexia nervosa and bulimia nervosa? It is interesting to speculate that the abundance of food combined with the cultural preoccupation with thinness as a model of attractiveness may be involved. However, such a simple cultural model does not explain why certain individuals develop this problem and others do not. This is a particularly intriguing question when one considers that experimentation with bulimic behaviors appears to be common among young people. Why does this behavior escalate out of control in a few individuals, while most give it up before it becomes an ongoing pattern of behavior? This is the crucial question that cannot yet be answered.

Unfortunately, the problem of bulimia nervosa is not only a problem of eating. A variety of other problems have been described in association with this condition, depression being one of the most common [1,14,19,21]. Patients with bulimia nervosa frequently have been noted to be depressed at the time of evaluation [13,14,18]. The prevalence of depressive symptoms in these patients has led several clinicians to investigate the possible associations between bulimia nervosa and primary affective disorder. Several lines of evidence suggest a close relationship between the two disorders, including a high rate of nonsuppression on the dexamethasone suppression test [22-26], an unexpectedly high rate of affective disorders in relatives of patients with bulimia nervosa [25,27], and the apparent utility of antidepressant compounds in the treatment of patients

with bulimia [27-31]. Also, Hudson and associates found that the majority of patients with bulimia met DSM-III criteria for major affective disorders using the structured diagnostic interview schedule [23]. What has not been adequately studied is the course of depressive symptoms with improvement in eating behavior. Does depression improve if bulimic symptoms normalize? Or does the depressive component require separate treatment? These questions require further study.

Several other problems have been described in association with bulimia, including problems with impulse control [14], stealing behavior [32], and an unexpectedly high rate of chemical dependency problems [14,18]. Our group reported that 8 of 34 patients with bulimia previously had received chemical dependency treatment and that one additional patient was felt to have a diagnosis of alcoholism [14]. Herzog found that 10 of 30 bulimic patients reported alcoholism in at least one first-degree family member [18]. Hatsukami and associates discussed the similarities between bulimic behavior and the behavioral pattern associated with drug abuse, including loss of control over the use of the substance, intense preoccupation with the substance, social isolation accompanying the behavior, and the reinforcing nature of the behavior [33]. It would appear that for some patients there is a relationship between the abuse of food and the abuse of alcohol or other drugs. Certainly the presence of chemical abuse problems should be evaluated in every patient with bulimia.

Several lines of evidence suggest significant psychological impairment in patients with bulimia nervosa. Weiss and Ebert [32] compared a sample of normal-weight female bulimic patients with a sample of normal-weight female controls matched for age, socioeconomic status, and IQ. Bulimic patients demonstrated higher levels of psychopathology on all the instruments used and consistently rated themselves as sicker on all psychometric scales. They also indicated more impulsive behavior and a history of more suicide attempts, more psychiatric hospitalizations, more episodes of stealing, and more problems with drug abuse. Two MMPI studies also suggested psychological impairment in this group of patients. Hatsukami and associates demonstrated that the MMPI profile of patients with bulimia were similar to the MMPI profiles of women with alcohol and drug abuse problems, the composite profiles for both groups demonstrating elevations on the scales for depression, impulsivity, anger, anxiety, and social withdrawal [33]. Norman and Herzog [34] found similar results. Taken together, these studies suggest that many patients with bulimia nervosa have significant emotional disturbances, at least when they are actively bulimic. To what extent these represent stable ongoing personality

characteristics that antedate the bulimia nervosa is unknown. However, clinical experience with bulimic patients suggests that no single type of personality pattern is invariably present and that many patients appear to be reasonably stable and well-adjusted after their eating pattern normalizes.

When patients with bulimia nervosa are seen for evaluation, there is also often evidence of significant social impairment. Using structured self-rating scales, Johnson and Berndt [35] showed that individuals with bulimia report increased impairment in social adjustment compared with a community sample control group. The bulimic patients demonstrated a response pattern similar to the response pattern of a group of alcoholic patients.

One of the social parameters most commonly disrupted by bulimia nervosa is social relationships. Patients with this disorder frequently report that they have experienced difficulties with family, friends, or other significant people in their lives since the development of the problem. They find that they frequently have to lie to others about why food has disappeared, why they can't go out, or why they fail to show up for an appointment. Many also report that their work or school performance suffers, and some report financial problems because of the costs involved. Some of the individuals we have evaluated have taken second jobs or have had to declare bankruptcy because of the attendant financial problems. Overall, most patients with this disorder report that bulimia nervosa interferes a great deal with other aspects of their life [14,21,35].

Little is known about the longitudinal course of this illness. Many patients apparently experience an initial period of weight loss at the onset of bulimic symptoms. However, as the illness progresses, this pattern is reversed and patients may eventually gain weight [13]. This may reflect changes in the binge eating pattern itself in that patients may begin to binge eat more frequently and to consume more food during each binge-eating episode as the illness progresses.

SIGNS AND SYMPTOMS

Bulimia nervosa is associated with several potentially serious physical complications. However, there is usually nothing apparent on physical examination to indicate the severity of the problem. Complaints are frequently vague, including such things as lethargy, impaired concentration, and nonfocal abdominal pain. Commonly patients with bulimia may undergo physical evaluations for such complaints or because electrolyte abnormalities were found on screening laboratory work during a general physical examination. If the patients

never mention their bulimic symptoms, the evaluating physician will often not be able to uncover any etiology for the complaints or the laboratory results.

Of the medical complications, most commonly encountered are fluid and electrolyte abnormalities. Russell originally reported hypokalemia as a complicating factor of this disorder [1]. Our group [19] subsequently reported that nearly 50% of a series of 168 patients with bulimia or atypical eating disorder demonstrated fluid or electrolyte abnormalities. Most commonly encountered were metabolic alkalosis (27.4%), hypochloremia (23.8%), and hypokalemia (13.7%). This pattern of abnormalities results from the self-induced vomiting or laxative abuse pattern seen in these patients. Metabolic acidosis is less commonly seen and may result from prolonged fasting or the acute diarrhea that may follow laxative abuse [44].

Salivary gland swelling has been reported in patients with bulimia [36], the parotid glands being most commonly effected. The clinical picture is one of painless swelling at the lateral angle of the jaw and is sometimes referred to as "puffy cheeks" by patients. Biopsy studies have shown normal tissue or asymptomatic noninflammatory changes. The pathophysiology of this problem is unclear. In our experience this complication is fairly common. Interestingly, the problem may persist for several months beyond normalization of eating patterns.

A complication that is fortunately rare, but unfortunately quite serious when it occurs in association with bulimia, is gastric dilatation [37,38]. Although usually described in patients with anorexia nervosa who are undergoing refeeding, this condition can also be seen in patients of normal weight who binge eat. In a review of this problem, Saul and associates reported a case of a patient with a history of anorexia nervosa who excessively overate, developed gastric infarction, and subsequent perforation of the stomach [37]. Saul and associates reviewed 66 such cases of spontaneous rupture of the stomach and felt that approximately half of the cases were related to the ingestion of large amounts of food and/or gastric dilatation. Eleven of these patients had been diagnosed as having anorexia nervosa, and most of that subgroup were undergoing refeeding. We have also reported a case of gastric dilatation in a nonanorectic bulimic patient.

Dental complications are frequent in these patients. The pattern described most commonly is decalcification of the lingual, palatal, and posterior occlusal surfaces of the teeth, secondary to erosion of the enamel. This pattern is caused by exposure of the teeth to the acid gastric contents [39,45].

Several reports have suggested the possibility of underlying EEG abnormalities in patients with eating disorders, some of whom appear to have had bulimia nervosa. A series of studies reported by Rau, Green and their colleagues described certain EEG abnormalities in patients with compulsive eating disorders [40,41]. We subsequently examined EEGs in a series of patients diagnosed by DSM-III criteria as having bulimia and found a low rate of EEG abnormalities [42]. This area requires further research.

There has been considerable interest in recent years in the possibility of hypothalamic dysfunction in patients with eating disorders. Neuroendocrine regulatory systems that are under hypothalamic control have been studied to indirectly evaluate hypothalamic functioning. Most of the clinical work in this area on eating disorder patients has involved patients with anorexia nervosa or obesity. However, recent reports also suggest certain neuroendocrine abnormalities in patients with bulimia nervosa. The published studies have described pathological growth hormone responsiveness to TRH or glucose administration, elevated basal prolactin levels, and a high rate of nonsuppression on the dexamethasone suppression test but normal TRH responsiveness to TSH in most patients [24,25,46,47]. The clinical significance of these abnormalities is unclear. The medical complications of this disorder have recently been reviewed [48].

SUMMARY AND CONCLUSIONS

Bulimia nervosa, a disorder that is equated in the minds of many people with binge eating, actually is a syndrome characterized by a variety of abnormal eating-related behaviors as well as other problems. Patients with bulimia nervosa may demonstrate problems with self-induced vomiting, laxative abuse, diuretic abuse, excessive exercise, and abuse of diet pills and enemas. These patients rarely eat normal meals. Research indicates that this is a common problem and that it can be associated with serious psychological and medical consequences.

Unfortunately, we know little as to why certain people develop this disorder or about the longitudinal course of the illness once it is established. We do know that it usually begins late in adolescence. Stressful life events may precipitate the onset, as may a period of dieting behavior. There are several intriguing findings in the literature that suggest familial relationships between drug abuse problems, mood disorders, and bulimia. Clearly, further research is needed in this area.

REFERENCES

1. Russell G. Bulimia nervosa: an ominous variant of anorexia nervosa. Psychol Med, 1979; 9:429-48.

2. Boskind-Lodahl M, White WC. The definition and treatment of bulimarexia in college women: a pilot study. JACHA, 1978; 27:84-6.

3. Palmer RL. The dietary chaos syndrome: a useful new term? Br J Med Psychol, 1979; 52:187-190.

4. Hawkins II, Clement PF. Development and construct validation of a self-report measure of binge-eating tendencies. Addictive Behaviors, 1982; 7:435-9.

5. Halmi KA, Falk JR, Schwartz E. Binge-eating and vomiting: a survey of a college population. Psychol Med, 1981; 11:697-706.

6. Sinoway CG. The incidence and characteristics of bulimarexia in Penn State students. Paper presented at the American Psychological Association Meeting, Washington, DC, 1983:

7. Pyle RL, Mitchell JE, Eckert ED, et al. The incidence of bulimia in freshman college students. International Journal of Eating Disorders, 1983; 2:75-85.

8. Cooper PJ, Fairburn CG. Binge-eating and self-induced vomiting in the community—a preliminary study. Br J Psychiatry, 1983; 142:139-44.

9. Clark MG, Palmer RL. Eating attitudes and neurotic symptoms in university students. Br J Psychiatry, 1983; 142:299-304.

10. Johnson CL, Lewis C, Love S, et al. A descriptive survey of dieting and bulimic behavior in a female high school population. In Understanding Anorexia Nervosa and Bulimia. Columbus, Ohio: Ross Laboratories, 1983:

11. Pope HG Jr, Hudson JI, Yrgelun-Todd D. Anorexia nervosa and bulimi among 300 suburban women shoppers. Am J Psychiatry, 1984; 141:292-4.

12. Fairburn CG. Binge-eating and its management. Br J Psychiatry, 1982; 141:631-3.

13. Fairburn CG. Binge-eating and bulimia nervosa. SK and F Publications, 1982; 1:1-20.

14. Pyle RL, Mitchell JE, Eckert Ed. Bulimia: a report of 34 cases. J Clin Psychiatry, 1981; 42:60.

15. Abraham SF, Beumont PJV. How patients describe bulimia or binge- eating. Psychol Med, 1982; 12:625-35.

16. Fairburn CG, Cooper JP. Self-induced vomiting and bulimia nervosa: an undetected problem. Br Med J, 1982; 284:1153-5.

17. Johnson CL, Stuckey MK, Lewis LD, et al. Bulimia: a descriptive survey of 316 cases. International Journal of Eating Disorders, 1982; 2:3-16.

18. Herzog DB. Bulimia: the secretive syndrome. Psychosomatics, 1982; 23:481-7.

19. Mitchell JE, Pyle RL, Eckert ED, et al. Electrolyte and other physiological abnormalities in patients with bulimia. Psychol Med, 1983; 13:273-8.

20. Mitchell JE, Pyle RL, Eckert ED. Frequency and duration of binge-eating episodes in patients with bulimia. Am J Psychiatry, 1981; 138:835-6.

21. Johnson C, Larson R. Bulimia: an analysis of mood and behavior. Psychosom Med, 1982; 44:341-51.

22. Hudson JI, Laffer PS, Pope HG. Bulimia related to affective disorder by family history and response to the dexamethasone suppression test. Am J Psychiatry, 1982; 139:685-7.

23. Hudson JE, Pope HG, Jonas JM. Bulimia: a form of affective disorder? Paper presented at the American Psychiatric Association Meeting, New York, May 1983:

24. Hudson JI, Pope HG, Jonas JM, et al. Hypothalamic-pituitary- adrenal axia: Hyperactivity in bulimia. Psychiatry Research, 1983; 8:111-7.

25. Gwirtsman HE, Roy-Byrne P, Yager J, et al. Neuroendocrine abnormalities in bulimia. Am J Psychiatry, 1983; 140:559-63.

26. Mitchell JE, Pyle RL, Hatsukami D, et al. The dexamethasone suppression test in patients with the bulimia syndrome. J Clin Psychiatry, 1984; 45:508-11.

27. Hudson JE, Pope HG, Jonas JM, et al. Family history study of anorexia nervosa and bulimia. Br J Psychiatry, 1983; 142:133-8.

28. Pope HG, Hudson JI. Treatment of bulimia with antidepressants. Psychopharmacology, 1982; 78:176-9.

29. Pope HG, Hudson JI, Jonas JM. Antidepressant treatment of bulimia: preliminary experience and practical recommendations. J Clin Psychopharmacology, 1984; 3:274-81.

30. Pope HG, Hudson JI, Jonas JM, et al. Bulimia treated with imipramine: a placebo-controlled double-blind study. Am J Psychiatry, 1983; 140:544-58.

31. Mitchell JE, Groat R. A double-blind placebo-controlled trial of amitriptyline in bulimia. J Clin Psychopharmacology, 1984; 4:186- 93.

32. Weiss SR, Ebert MH. Psychological and behavioral characteristics of normal-weight bulimics and normal-weight controls. Psychosom Med, 1983; 45:293-303.

33. Hatsukami D, Own P, Pyle R, et al. Similarities and differences on the MMPI between women with bulimia and women with alcohol or drug abuse problems. Addictive Behaviors, 1982; 7:435-9.

34. Norman DK, Herzog DB. Bulimia, anorexia nervosa, and anorexia nervosa with bulimia: a comparative analysis of MMPI profiles. International Journal of Eating Disorders, 1983; 2:43-52.

35. Johnson C, Berndt DJ. Preliminary investigation of bulimia and life adjustment. Am J Psychiatry, 1983; 140:774-7.

36. Levin PA, Falko JM, Dixon K, et al. Benign parotid enlargement in bulimia. Ann Intern Med, 1980; 93:827-9.

37. Saul SH, Dekker A, Watson CG. Acute gastric dilatation with infarction and perforation. Gut, 1981; 22:978-83.

38. Mitchell JE, Pyle RL, Miner RA. Gastric dilatation as a complication of bulimia. Psychosomatics, 1982; 23:96-7.

39. Stege P, Visco-Dangler L, Rye L. Anorexia nervosa: review including oral and dental manifestations. JADA, 1982; 104:648-52.

40. Rau JH, Green RS. Compulsive eating: a neuropsychologic approach to certain eating disorders. Compr Psychiatry, 1975; 16:223-231.

41. Rau JH, Struve FA, Green RS. Electroencephalographic correlates of compulsive eating. Clin Electroencephalogr, 1979; 10:180-9.

42. Mitchell JE, Hostfield W, Pyle RL. EEG findings in patients with the bulimia syndrome. International Journal of Eating Disorders, 1983; 2:17-23.

43. Mitchell JE, Bantle JP. Metabolic and endocrine investigations in women of normal weight with the bulimia syndrome. Biol Psychiatry, 1983; 18:355-65.

44. Mitchell JE, Hatsukami D, Pyle RL, et al. Metabolic acidosis as a marker for laxative abuse in patients with bulimia. International Journal of Eating Disorders, 1987; 6:557-560.

45. Simmons MS, Grayden SK, Mitchell JE. The need for psychiatric-dental liaison in the treatment of bulimia. Am J Psychiatry, 1986; 143:783-4.

46. Lindy DC, Walsh BT, Roose SP, et al. The dexamethasone suppression test in bulimia. Am J Psychiatry, 1985; 142:1375-6.

47. Norris PD, O'Malley BP, Palmer RL. the TRH test in bulimia and anorexia nervosa: a controlled study. J Psychiatr Res, 1985; 19:215-9.

48. Mitchell JE, Seim HC, Colon E, et al. Medical complications and medical management of bulimia. Ann Int Med, 1987; 107:71-7.