Clinical Review

Anorexia Nervosa and Bulimia

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Anorexia nervosa may occur in one of 200 white adolescent girls, and bulimia appears to be much more common, particularly in older adolescents and young women. These disorders are distinctly uncommon in the male population. Current opinion supports a psychological basis for these disorders, although there are some findings that suggest a primary hypothalamic defect. Early warning signs of anorexia nervosa include an arrest in weight gain during puberty, increasing social isolation, hyperathleticism, and increasing concern over academic performance. Bulimia may exist concomitantly with anorexia nervosa or as an entirely separate disorder characterized by a recurrent binge-purge cycle. The signs and symptoms of these eating disorders are mainly those associated with weight loss, dehydration, and electrolyte imbalance. Because of the complex psychological issues involved, an experienced psychotherapist should be involved while the primary care clinician provides anticipatory and supportive medical care.

Anorexia nervosa is currently viewed as having multiple interacting causes that include biological vulnerability, early experiences, and family influences creating a psychological predisposition, social climate and cultural obsession, possible endocrine changes, and psychological conflicts involving issues such as mastery and control, sexuality, inappropriate expectations, and abandonment.[1]

Bulimia may exist concomitantly with anorexia nervosa, as a subsequent clinical syndrome following anorexia, or as an apparent distinct entity with different psychological roots. The main clinical distinction between anorexia nervosa and bulimia involves starvation in the former and recurrent episodes of excessive food intake (binging) usually followed by vomiting or purging in the latter. Patients with anorexia nervosa usually become more cachectic than those with bulimia.[2] Patients with anorexia and bulimia both share an extreme and

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unrealistic concern regarding body image, food, and eating behavior.

In general, patients with anorexia nervosa are resistant to therapy and usually are brought to the health care provider by another concerned individual. Conversely, patients with bulimia unassociated with anorexia nervosa frequently are very concerned about their binge eating and vomiting and purging behavior and may present themselves to health care providers for assistance in gaining control over this compulsive activity. Bulimic patients are not easier to treat, however, since the pattern of binge eating and vomiting or purging usually proves extremely difficult to disrupt.

Because of the complex psychological aspects of these eating disorders, the involvement of an experienced mental health professional is strongly indicated. Nevertheless, the primary care clinician can play an important role in providing continuity of care through anticipatory and supportive medical management. This paper is intended to provide the clinician with information and guidelines useful in the medical management of anorexia nervosa and bulimia.

Etiology

Family dynamics appear to be paramount in the development of anorexia nervosa, although these factors have not been extensively evaluated in bulimia. Although many exceptions occur, and it is important to remain skeptical about the "typical" family dynamics involved in anorexia nervosa, several characteristics are quite common. At first glance the family may appear to be a model of success, with parents presenting a satisfactory and harmonious marriage. However, a general lack of fulfillment as a couple appears to lead the parents to overinvolvement and excessive expectation displaced upon the children. Typically, a daughter, who may be somehow vulnerable, becomes the primary figure in this subconscious family issue. She often feels she must be a model child in order to please her parents, earn their acceptance, meet their expectations, and make up for their personal disappointments and dissatisfactions with each other. Since she is never able to do this completely, she experiences a sense of failure and ineffectiveness, which may lead her to develop a symptom demonstrating superior personal control and expressing accumulated anger against her parents. The interpersonal relationships in these families tend to be intense, with evidence of excessive closeness among family members and a tendency toward overprotectiveness of one another. Bringing these family issues to the surface and facilitating alternative solutions requires the skills of an experienced psychotherapist.

Although current opinion supports a psychological basis for anorexia nervosa and bulimia, with endocrine and other physical changes produced mainly as a consequence of severe weight loss, there are some intriguing aspects that lend support to the hypothesis of a primary organic etiology. Several studies have suggested a primary hypothalamic defect in anorexia nervosa.[3,4] When exposed to increased environmental temperature, patients with anorexia nervosa do not exhibit the initial core temperature decrease seen in normal subjects or in those with simple starvation, and they develop a faster subsequent rise in temperature. Exposed to a cold environment, they do not show the usual initial core increase and develop a faster subsequent decrease in core temperature. Anorectic patients also do not exhibit shivering when exposed to a cold environment. In response to water deprivation, patients with anorexia nervosa do show an initial increased urine osmolality greater than that of plasma, but an additional rise is seen following the administration of vasopressin, suggesting the possibility of a partial diabetes insipidus.

Patients with anorexia nervosa frequently appear to have a misperception of bodily sensations, particularly of enteroceptive stimuli. Distortions of satiety, sucrose aversion, and altered perception of body image have been reported.[5,6] In addition, these patients often appear to fail to recognize their own affective states (eg, anger, anxiety, guilt) and frequently will deny fatigue. Unlike starvation victims, who are not active, anorexia nervosa patients often are driven to hyperathleticism such as long-distance running and excessive calisthenic exercising. Since many patients with anorexia nervosa experience feelings of hunger that they pride themselves in resisting, the term *anorexia* is probably a misnomer. The German term *Pubertaetsmagersucht*, or "leanness passion

Table 1. DSM-III Diagnostic Criteria for Bulimia[11]

- Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than two hours)
- At least three of the following:
  - Consumption of high-caloric, easily ingested food during a binge
  - Inconspicuous eating during a binge
  - Termination of such eating episodes by abdominal pain, sleep, social interruption, or self-induced vomiting
  - Repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics or diuretics
  - Frequent weight fluctuations greater than ten pounds due to alternating binges and fasts
- Awareness that the eating pattern is abnormal and fear of not being able to stop eating voluntarily
- Depressed mood and thoughts of self-deprecation following eating binges
- Bulimic episodes not due to anorexia nervosa or any known physical disorder

Table 2. DSM-III Diagnostic Criteria for Anorexia Nervosa[11]

- Intense fear of becoming obese, which does not diminish as weight loss progresses
- Disturbance of body image, eg, claiming to "feel fat" even when emaciated
- Weight loss of at least 25 percent of original body weight or, if under 18 years of age, weight loss from original body weight plus projected weight gain expected from growth charts may be combined to make the 25 percent
- Refusal to maintain body weight over a minimal normal weight for age and height
- No known physical illness that would account for the weight loss

of puberty," may be more appropriate for this disorder.

Incidence

Since the eating disorders (particularly bulimia) are most certainly underreported, it is difficult to estimate their incidence. Although described for centuries, the prevalence of anorexia nervosa, and most likely bulimia, is apparently increasing. The incidence of anorexia nervosa in white adolescent girls in developed countries has been estimated to be approximately 1 in 200.[7] It appears that bulimia is much more common than anorexia nervosa, particularly during late adolescence and young adult years.[8] Anorexia nervosa is seen mainly in girls and women, with a female:male ratio estimated to be greater than 10:1.[7] Male patients with anorexia nervosa seem to have psychological characteristics similar to those of women with the disorder.[9] The incidence of anorexia nervosa peaks in early adolescence, whereas bulimia is more commonly a late adolescent phenomenon.[7,10]

Diagnosis

The *Diagnostic and Statistical Manual of Mental Disorders, Third Edition*[11] (DSM-III) criteria for bulimia listed in Table 1 are useful to the primary care clinician. The diagnostic criteria for anorexia nervosa listed in Table 2 are likewise helpful, although insisting upon a weight loss equaling 25 percent of premorbid body weight may result in underdiagnosis of milder or incipient cases that might benefit from early intervention. Typical development in the evolution of anorexia nervosa is depicted in Figure 1. Early warning signs that should alert the clinician to this diagnosis include arrest in weight gain during puberty, increasing social isolation, increased athleticism, and an increased concern for perfect performance in academic endeavors. Early intervention that

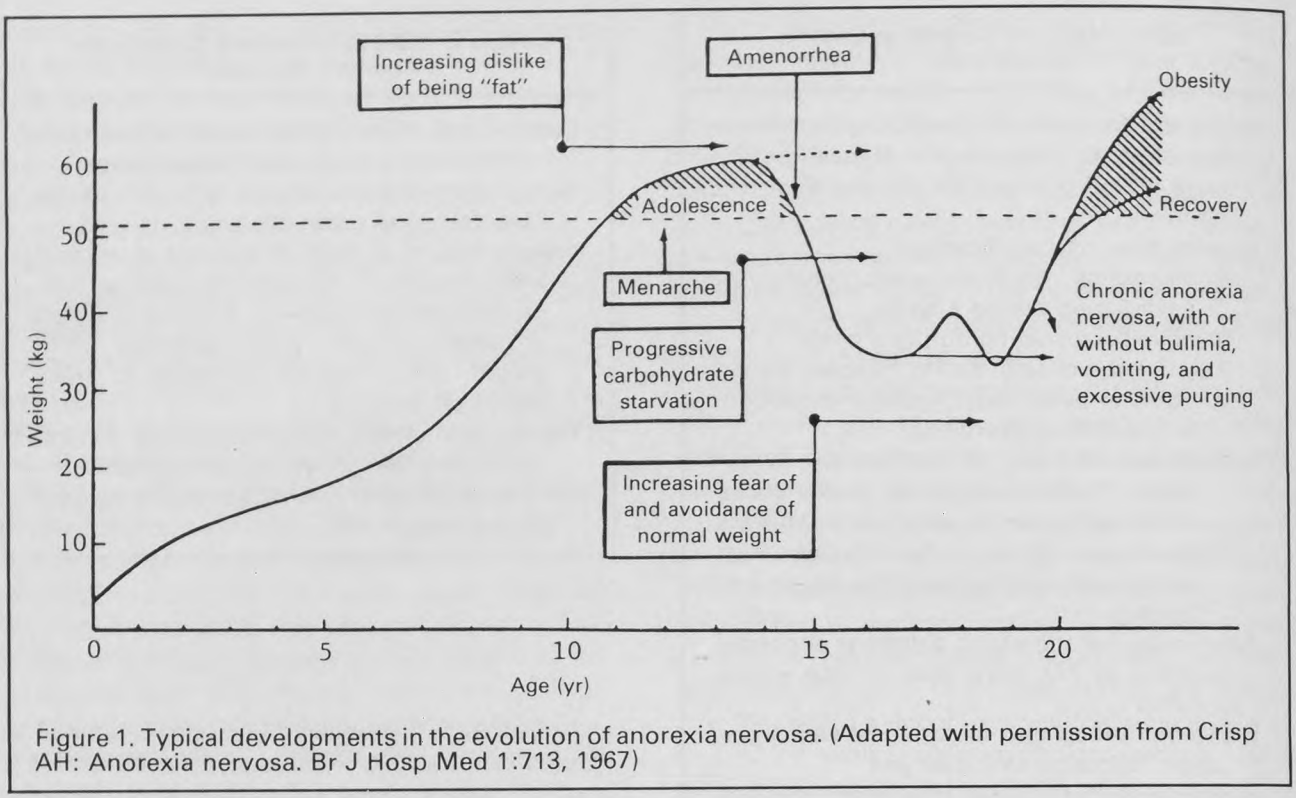

Figure 1. Typical developments in the evolution of anorexia nervosa. (Adapted with permission from Crisp AH: Anorexia nervosa. Br J Hosp Med 1:713, 1967)

mobilizes the assistance of a mental health professional, coupled with ongoing supportive guidance by the primary care clinician, may possibly avert the development of a full-blown anorexia nervosa syndrome.

Differential Diagnosis

It is unlikely that in an amenorrheic adolescent, the onset of weight loss with dieting, the desire for extreme thinness, and the fear of weight gain will be due to a disorder other than anorexia nervosa.[12] Although a rare case of a patient with an underlying organic disorder may simulate anorexia nervosa, most of these patients present with some clinical aspects that are inconsistent with the diagnosis of primary eating disorder.[13,14] Certainly the clinician must consider occult organic conditions such as hyperthyroidism, adrenal insufficiency, hypopituitarism, inflammatory gastrointestinal disease, and brain tumor in the differential diagnosis of anorexia nervosa; but a meticulous history and physical examination will usually suffice to rule out the likelihood of such disorders.

In hyperthyroidism weight loss occurs despite normal or increased food intake. Although hyperactivity is common, it does not take the form of vigorous exercise, and lethargy is a frequent complaint. Tachycardia, rather than bradycardia, is present and thyroid hormone levels are elevated. Adrenal insufficiency may be associated with poor appetite and hypotension, but lethargy and weakness are usually prominent symptoms. Increased skin pigmentation and a reduction of pubic and axillary hair may be found on physical examination. In contrast to anorexia nervosa, plasma cortisol levels are low but maintain normal diurnal variation, and the adrenal response to stimulatory tests is subnormal or absent. Pituitary

dysfunction also causes amenorrhea and must be considered in the differential diagnosis of anorexia nervosa. Craniopharyngioma is the most likely cause of pituitary dysfunction in this age group, although prolactin-secreting pituitary tumors must also be considered. Weight loss is not usually a prominent feature of either tumor.

Gastrointestinal disease with malabsorption may result in weight loss, but should rarely be confused with anorexia nervosa. In inflammatory bowel disease there is usually a history of abdominal discomfort and diarrhea, often with blood and mucus. In celiac or other malabsorption syndromes, steatorrhea is a prominent feature. When diarrhea is present in anorexia nervosa, it is usually self-induced by laxatives and is frequently not volunteered as a symptom.

Poor appetite with weight loss may be a feature of severe depression or schizophrenia. The presence of diagnostic features of these disorders and the absence of abnormal desire to lose weight usually allow differentiation from anorexia nervosa. Although symptoms of depression frequently accompany anorexia nervosa, the characteristic features of the underlying syndrome are usually predominant.[15,16]

Severe physical illness, such as tuberculosis and malignancy, will usually be accompanied by obvious physical symptoms or signs other than weight loss and amenorrhea. Lethargy and malaise, rather than hyperactivity, are usually prominent, and there is not an abnormal desire for thinness.

In the differential diagnosis of bulimia, one must consider esophageal and gastrointestinal disorders, metabolic abnormalities, and tumors of the central nervous system. Although changes in personality and vomiting behavior may occur with tumors of the central nervous system, and a careful neurological examination with visual field assessment is mandatory, the binge-purge or -vomit cycle and an excessive concern about body image are not typical of these disorders.

Signs and Symptoms

The signs and symptoms of these eating disorders are those associated with weight loss, dehydration, and electrolyte imbalance. Amenorrhea may precede significant weight loss in up to 25 percent of patients with anorexia nervosa.[17] Although some authors have suggested that this finding lends support to the hypothesis of an organic etiology of anorexia nervosa, it is clear that most of these patients are experiencing significant emotional stress long before the onset of significant weight loss. Emotional distress is well established as a common cause of secondary amenorrhea in adolescent and adult women.[18] Patients may complain of abdominal pain and constipation. Decreased gastric motility has been documented in anorexia nervosa and may be associated with abdominal pain.[19] Acute gastric dilatation and pancreatitis may occur during periods of refeeding.[20] Constipation may be related to relative dehydration or simply scant stools due to sparse food intake mislabeled as constipation by the patient. In addition, many patients with eating disorders abuse laxatives and may become confused regarding normal bowel habits.[21] Other symptoms that may be volunteered include cold intolerance, dry skin, and brittle hair and nails, which are probably due to loss of subcutaneous fat and malnutrition, but decreased tri-iodothyronine levels (reflecting a compensatory decreased metabolic rate, not hypothyroidism) may also contribute to these symptoms.[22]

Physical Findings

The physical findings in anorexia nervosa are essentially the same as those found in any starvation state. Subcutaneous fat is lost and the skin may be dry and inelastic with a yellowish carotenemic hue. The occasional petechiae that may be noted are presumably due to increased capillary fragility and are not related to a coagulation disorder. Body temperature is low and bradycardia with decreased blood pressure may be noted. Postural hypotension is a frequent finding. Pubic and axillary hair is preserved and lanugo may appear, predominantly on the extremities. Peripheral edema may be noted, particularly during times of refeeding. Parotid swelling of unclear etiology[23] and increased dental caries possibly related to re-

current contact with acidic vomitus or excessive carbohydrate intake during binging[2] may be noted in bulimic patients. Anthropometric measurements using a tape measure and calipers will provide indices for estimation of chronic undernourished states. Patients with longstanding starvation may show significant decrease in both fat and muscle measurements.

Laboratory Evaluation

With the exception of values indicating fluid and electrolyte imbalance, the laboratory evaluation of these eating disorders is of minimal assistance in clinical management. Repetitive vomiting or use of laxatives may produce metabolic abnormalities, the most common of which is hypokalemic, hypochloremic alkalosis.[24] Occasionally diuretic abuse is also encountered, which may compound this metabolic state.[25] Electrocardiography may reflect these metabolic abnormalities as well as the hypometabolic state (bradycardia, low voltage).[26] Cerebral atrophy, probably secondary to malnutrition, has been noted by computed tomography in patients with anorexia nervosa.[27]

Glucose levels are generally maintained in a low normal range.[28,29] Anemia is not usual except in severely emaciated patients.[30] Leukopenia is frequently present but does not appear to be associated with an increased risk of infection.[31] Serum albumin levels are maintained in the normal range in all but the most severely malnourished cases.[30] Although low in other forms of weight loss, serum carotene levels are frequently elevated in anorexia nervosa.[32] The mechanism, which does not appear to be related solely to increased intake, is unclear.[33] Serum carotene levels have been reported to be elevated in patients with anorexia nervosa, but not in those with bulimia.[34] While these findings relating to serum carotene are interesting, they are rarely useful in the clinical management of these eating disorders.

The endocrine aspects of anorexia nervosa are intriguing but, with the exception of amenorrhea, do not appear to be clinically significant. Growth hormone and cortisol are frequently increased, and the latter may or may not lose its diurnal rhythm.[35,36] Dexamethasone suppression tests may yield inadequate responses.[37] Gonadotropins are decreased, frequently to prepubertal ranges.[38] Although thyroxine and thyroid-stimulating hormone are in the normal range, tri-iodothyronine levels are frequently decreased.[39] This decrease probably represents a basal metabolic rate normalized for body mass with decreased peripheral conversion of thyroxine to tri-iodothyronine and is not an indication for treatment with thyroid medication.

Management

The initial medical evaluation of the adolescent with an eating disorder should include a thorough history and physical examination coupled with the selective use of a few indicated laboratory studies. A "shotgun" approach with multiple laboratory and radiologic procedures is costly and has a low diagnostic yield. The amenorrheic patient with diet-induced weight loss, an expressed desire for extreme thinness, and inordinate fear of weight gain who has no signs or symptoms of other organic disease requires little laboratory investigation. A recommended initial medical evaluation of the adolescent with an eating disorder includes a urinalysis, complete blood count, erythrocyte sedimentation rate, and serum electrolytes. Clearly, the more atypical features there are in a patient with suspected anorexia nervosa, the more consideration must be given to alternative diagnoses. Laboratory studies should be selected accordingly.

Because anorexia nervosa and bulimia are complex and chronic disorders, a multidisciplinary approach to therapy is recommended. Although the efficacy of psychotherapy in these eating disorders has not been clearly established in well-controlled studies, the early involvement of an experienced mental health professional is strongly recommended. The natural course of anorexia nervosa, at least that portion of the syndrome reflected by severe weight loss, commonly extends over a three- to four-year period.[40,41] Although psychological problems, excessive concern over food and body image, and, not infrequently, obesity may follow this initial active period of anorexia nervosa,[42] the life-threatening aspect of

the disorder will usually subside with time. The natural course of bulimia is unclear, but it appears that it may persist unabated for many years, possibly becoming a lifetime problem.[43,44] With this perspective regarding the chronicity of these disorders in mind, it is obvious that continuity of therapy is desirable. In addition to mental health assistance, an experienced nutritionist who is comfortable working with adolescents with eating disorders can be helpful. Although these patients are clearly not suffering from a primary nutritional disorder, the practical advice and guidance of a nutritional authority can be helpful in reducing anxiety and guiding the patient toward more sound nutritional practices.

Clearly, all health professionals involved in the care of patients with eating disorders must be prepared to deal with their own feelings of frustration, impatience, and, occasionally, anger. In the interdisciplinary approach, it is important to establish the roles of each provider, since adolescent patients with eating disorders are frequently adept in the art of playing one member of the team against another. Regular communication among members of the team is important in this regard. Optimally, the role of the primary care clinician should focus upon the physical aspects of the disorder, while management of psychological issues such as family conflict and feelings of helplessness are deferred to the mental health professional. However, general supportive counseling and assistance in stress management may be appropriate roles for the primary care clinician.

The role of the primary care clinician is often difficult, since the adolescent with an eating disorder is typically unable to address rationally the issues of nutrition, food intake, and body image. The clinician must realize that it is not possible to assume personal responsibility for the nutritional status of the patient. Presenting ideal body weight charts, lecturing on nutrition, or performing caloric counts at clinic visits is not helpful and simply will not be heard by the patient. Acknowledging the difficulty the patient is having with weight management and expressing clinical concern over nutritional and metabolic status are strongly indicated.

Although frequently suggesting cooperation and a desire to comply with medical therapy, the patient with an eating disorder often employs deceit and subterfuge in the effort to avoid the frightening experience of gaining weight. The clinician, although often exasperated by this behavior, should avoid taking it as a personal affront and responding punitively.

The primary care clinician should attempt to develop a therapeutic relationship with the adolescent with an eating disorder based upon the same principles of concern, mutual respect, and trust that are applied to any adolescent patient. Obviously, the establishment of trust in a conventional sense frequently is impossible. However, the clinician can impart to the patient a sense of trust that ultimately all therapeutic efforts are for the patient's benefit and do not represent arbitrary efforts to impose external control. The clinician should not assume that the adolescent with an eating disorder will be noncompliant in all areas. Although resistant to food intake and weight gain, these patients frequently will comply with regimens such as potassium therapy when the treatment rationale is clearly presented.

The patient who is severely compromised in nutritional status should be seen weekly. Weight, blood pressure, and electrolyte determinations (if the patient is vomiting or purging) are indicated measures. The clinician should regularly ask about eating behavior, vomiting, and laxative and diuretic use. These questions should be presented in a concerned and noncritical manner. The clinician should also inquire regarding other specific concerns of the patient. Whether the clinician should relate to the patient a specific weight at which hospitalization would be mandatory is debatable. The clinician should have in mind criteria for hospitalization such as continued weight loss below a certain limit or accelerated weight loss or increased vomiting behavior. Life-threatening conditions that mandate immediate hospitalization include shock, arrhythmia, and infection. Significant dehydration with an orthostatic drop in blood pressure of greater than 15 mmHg, severe hypokalemia, and greater than 40 percent loss from normal body weight or 25 to 30 percent loss occurring within three months are also indications for hospital admission with fluid, electrolyte, and nutritional therapy.[45]

There is no clear evidence that any psychotropic medication changes the course of anorexia nervosa or bulimia. Antidepressant medication may ameliorate significant dysphoric states, but usually will not affect the underlying eating disor-

der. A few reports have suggested that certain drugs (eg, carbamazepine, monoamine oxidase inhibitors) might be useful in some individuals with eating disorders, but no well-designed large prospective study has supported their efficacy.[46,47] Appetite stimulants such as cyproheptadine have not been shown to be useful in anorexia nervosa.[48] Metoclopramide may alleviate symptoms with decreased gastric motility in anorexia nervosa.[49] Although vitamin deficiency has not been documented in anorexia nervosa, pragmatic vitamin supplementation may be appropriate. In patients who are vomiting or purging frequently, potassium supplementation is frequently necessary.

Common Management Problems

Decreased fluid intake, vomiting, and laxative and diuretic abuse may lead to significant problems with volume depletion and electrolyte imbalance. Orthostatic hypotension is diagnosed when a standing blood pressure taken two minutes after a supine measurement shows a profound drop in systolic blood pressure of 15 to 30 mmHg, and a diastolic drop of greater than or equal to 30 beats/min. In addition to volume depletion, fasting may be associated with decreased sympathetic nervous system tone and increased endogenous opiate activation, which further decrease blood pressure.[50] Frequent vomiting may be associated with chronic sore throat, parotid swelling, dental caries, and gastric dilatation.[2,43]

Laxative or diuretic abuse may lead to hypochloremic alkalosis with seriously depleted potassium stores.[21,25] Although phosphate may be reduced with inadequate intake and markedly so during refeeding following starvation states, catabolism results in the release of intracellular stores of phosphate. Moderate levels of hypophosphatemia seen in untreated eating disorders appear to be well tolerated. Severe hypophosphatemia (less than 1.0 mg/dL), which may result in serious consequences (eg, rhabdomyolysis, hemolysis, myocardial dysfunction), is rarely seen except in the most severe cases of malnutrition or during refeeding without adequate phosphorus intake.[51]

Hypokalemia, usually induced by repeated vomiting, is frequently associated with eating disorders. The primary clinician should regularly question the patient regarding vomiting, laxative abuse, or the use of diuretics. In contrast to sodium, the kidney is unable to eliminate potassium completely. Even with a serious deficit, 5 to 15 mEq of potassium are excreted per day.[52] Potassium excretion is enhanced by the following factors: (1) increased sodium delivered to the distal tubule (diuretics), (2) alkalosis increasing intracellular potassium, which is then available for exchange at the lumenal border (vomiting), (3) increased mineralocorticoid with increased exchange of potassium for sodium at the distal tubule (hypovolemia), (4) nonreabsorbable anion at the distal tubule (chloride is better reabsorbed than bicarbonate; in chloride deficiency associated with vomiting or diuretics, only cation is exchanged with potassium for sodium), and (5) increased urine flow (partial diabetes insipidus?).

Early symptoms of hypokalemia (muscle weakness, lethargy) may be ascribed to the starvation state. Progressive hypokalemia leads to nausea and vomiting, ileus and abdominal distention, muscular paralysis, and potentially fatal cardiac arrhythmias. Patients with persistent vomiting may need regular potassium supplementation. Congestive heart failure has been reported in physically active anorexia nervosa patients during volume depletion.[53] Weakened cardiac muscle, patient overactivity, and volume overload appear to be significant factors.[54,55] Fortunately, these patients usually respond to the simple measures of volume restriction and diuretic agents.

Mean levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are significantly lower in anorectic and many bulimic patients than in healthy women or girls of comparable developmental stage.[38] Twenty-five percent of anorectic patients become amenorrheic prior to significant weight loss.[17] At less than 75 percent of the initial body weight, the pubertal progression of gonadotropin release is reversed, and a correlation exists between the degree of weight loss and the lack of maturity of circadian LH secretion. With restoration of the initial body weight and remission of psychological symptoms, the adult gonadotropin pattern is resumed. It has been reported, however, that patients who regain their initial body weight but retain the psychological symp-

toms of anorexia may experience persistent immature patterns of gonadotropin secretion.[56] The mechanism of hypothalamic dysfunction and amenorrhea with weight loss is unclear. Estrogen balance may be related to the percentage of body weight composed of fat. Catecholestrogens are increased in anorexia nervosa and may produce an antiestrogen effect as well as inhibit gonadotropin release.[38]

Amenorrheic individuals, such as significantly malnourished patients with anorexia nervosa, will usually have low levels of gonadotropins. Patients with borderline nutritional states such as those with bulimia, however, may have enough gonadotropin secretion to produce an endometrial effect without sufficient levels of progesterone (analogous to secondary amenorrhea in otherwise healthy women). This unopposed estrogen effect may cause continued stimulation of the endometrium with possible long-term deleterious effect. In these individuals, periodic induction of menses with progesterone may be indicated. The ultimate prognosis for menstruation and subsequent fertility following weight gain appears to be good, even after years of amenorrhea. Once pregnant, gestational weight gain is usually normal, and pregnancy, labor, and delivery appear to be uncomplicated.[38]

Hospitalization

Given the long clinical course of anorexia nervosa and bulimia, hospitalization does not represent a cure. Short-term hospitalization may be necessary for severe nutritional and metabolic derangement. Longer term hospitalization may stabilize these patients through behavior modification, milieu therapy, or family and individual psychotherapy. Recent reviews have described several types of programs.[57-59] In determining criteria for discharge from prolonged hospitalization, both weight gain and psychological status should be considered. Recommended discharge criteria include a weight for height greater than or equal to the 25th percentile and evidence that the patient is motivated to maintain a minimal nutritionally acceptable weight and continue in a psychotherapeutic relationship as an outpatient.

When the anorectic patient is hospitalized, the clinician should monitor ward staff relationships with the patient. Anticipation of efforts at deception is indicated, and nonpunitive behavioral modification techniques are often helpful. Clear role definition among the health professionals (eg, nutritionist, nurse, psychotherapist, primary care clinician) is important, and regular communication through team meetings is necessary.

The most difficult issue for the clinician, implicit in all aspects of care of the patient with anorexia nervosa, is the right of the individual to act in a self-destructive manner and ultimately to die. Since the psychodynamics of this disorder suggest that control over his or her own body is the final perceived power the adolescent with anorexia nervosa maintains, therapeutic efforts toward correcting self-induced starvation are necessarily in opposition to this conviction. Fortunately, most patients respond to therapy well enough to prevent death, even though typically maintaining precariously low body weights for several years. Some patients, however, will resist all efforts toward nutritional support, subtly sabotaging intravenous lines and nasogastric feeding tubes while gaining no insight from psychotherapy. Although legal commitment and forced feeding may be instituted, there is no clear answer to this dilemma, and the truly resistant, determined patient with anorexia nervosa may ultimately die.

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