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Eating disorders: Recognize early to prevent complications

■ ABSTRACT

Management of eating disorders can include psychotherapy, counseling, and medical therapy. Early diagnosis is critical. Medical therapy includes refeeding, educating, and encouraging the patient; identifying and treating any complications; prescribing an antidepressant agent when indicated; and scheduling frequent follow-up visits to monitor progress.

■ KEY POINTS

Eating disorders often remit or persist as a chronic condition, and patients with coexisting psychiatric diagnoses fare less well. The treatment plan needs to take this into account.

The diagnosis "eating disorder not otherwise specified" should not be downplayed, as recognizing these symptoms represents a chance to intervene and possibly to prevent the progression to full-blown anorexia nervosa or bulimia nervosa.

Bulimia often responds best to a combination of antidepressants and psychotherapy, rather than to one or the other alone.

EATING DISORDERS continue to surprise us. Although the classic patient used to be a white adolescent girl from an affluent family, eating disorders are now seen in both sexes and in all ages and socioeconomic groups.

Furthermore, although we physicians view eating disorders as psychiatric illnesses with potentially serious physical consequences, including death, patients may view them as "lifestyle choices," a view promoted by the Internet-based "pro-ana" movement, whose advocates are often devoted not to recovering from these conditions, but to promoting them with helpful hints on how to lose weight more effectively!

Awareness of the warning signs, especially in adolescent girls, can lead to earlier diagnosis, head off major problems, and start the patient on the long road to recovery.

In this article we review the key clinical features of the spectrum of eating disorders, the health consequences, and the principles of management.

■ EQUAL-OPPORTUNITY DISEASES

Eating disorders primarily affect adolescent girls and young women, but 5% to 10% of cases are in men.¹ Many patients are not overweight at the outset. Cases have been found in children as young as 5 years and in people well into their geriatric years.

Nevertheless, anorexia nervosa has two peak ages of onset: 13 to 14 years, the time of great pubertal change, and 17 to 18 years, the time of independence and transition from high school to college.²

*The author has indicated that she is on the speakers' bureaus of the Wyeth-Ayerst and Pfizer corporations.

How common are eating disorders?

The prevalence of anorexia nervosa has traditionally been cited as 1%, and that of bulimia as 1% to 9%.^{1,3} However, more than 10% of adolescent girls may have either a full or partial syndrome.⁴ In the Eating Among Teens (Project EAT) study of more than 4,000 Minnesota high school students, 45% of adolescent girls and 21% of adolescent boys reported that they were currently trying to lose weight, and an additional 26% of girls and 23% of boys were working on maintaining their weight.^{5,6}

An alarming number of children are using unhealthy dieting strategies. In one study,⁵ 57% of girls and 33% of boys said they use food substitutes (powders, special drinks), skip meals, fast, or smoke cigarettes to control their weight, and 12% of girls and 5% of boys reported using diet pills, laxatives, diuretics, or vomiting. For adolescent girls especially, these practices carry a high risk of serious health consequences (see below).⁷

Furthermore, the numbers seem to be increasing, although this trend may represent a greater awareness of the disorders on the part of physicians.⁸

What causes disordered eating?

The causes of eating disorders have not been clearly elucidated.^{1,2} Proposed culprits include:

- Genetics.^{9,10}
- Neuropeptide dysregulation.¹¹ New research is helping to elucidate the role of peptides including leptin, ghrelin, neuropeptide Y, and serotonin and their role in eating disorders.
- The media. As women in industrialized societies get heavier, the “ideal” body image promoted on television and in magazines continues to get thinner.^{1,8} Daily inundation with sleek, air-brushed images can affect how people perceive themselves. When television was introduced in Fiji, the prevalence of eating disorders increased exponentially.¹²
- Environmental stressors. Eating disorders are not just about food and weight. They may represent an attempt to cope with a perceived problem. The clinical team’s job is to help identify what problem the patient is trying to solve and to help her or him develop healthier coping strategies.

EATING DISORDERS DEFINED

Anorexia nervosa

According to the American Psychiatric Association’s Diagnostic and Statistical Manual, 4th edition (DSM-IV),³ all four of the following criteria must be met for a diagnosis of anorexia nervosa:

- Refusal to maintain a minimal body weight (85% of expected weight for age and height) or failure to grow appropriately during childhood and adolescence
- Fear of gaining weight even though underweight
- Disturbance in the way body weight is perceived; undue influence of body weight on self-evaluation; denial of the seriousness of the problem
- Absence of at least three consecutive menstrual cycles in a postmenarchal or developmentally appropriate girl or woman who is not receiving hormone therapy (more on this topic below).

Anorexia is considered “restrictive” if the patient avoids eating or exercises excessively, but many patients with anorexia go through stages of bulimia, ie, they binge and purge. Regardless of the subtype, most women with anorexia have a significantly distorted body image and feel fat despite actually being emaciated or undernourished.

Many patients move back and forth within the spectrum of eating disorders, most often starting with a typical restrictive anorexic pattern and then progressing towards bingeing with compensatory purging or restricting, or both (see below).⁹

Bulimia nervosa

The DSM-IV³ criteria for the diagnosis of bulimia nervosa are:

- Binge eating that occurs on the average of twice a week for at least 3 months
- Recurrent inappropriate behavior to control weight gain, ie, vomiting, abuse of laxatives or diuretics, excessive exercise
- Self-image that is unduly influenced by body shape and weight
- Disturbances that are not exclusively grouped with anorexia nervosa.

As with anorexia nervosa, patients with bulimia have a range of features and problems.

Eating disorders affect all ages and socioeconomic, racial, and ethnic groups

TABLE 1

Typical signs and symptoms of anorexia nervosa and bulimia nervosa

ANOREXIA NERVOSA

Gastrointestinal

Constipation, bloating, early satiety due to delayed gastric emptying and slowed metabolism

Endocrine

Dried skin, cold intolerance, fatigue, decreased energy, scalp hair loss, primary or secondary amenorrhea, bone pain

Cardiovascular

Blue hands and feet, fainting, palpitations, acrocyanosis

Hematologic

Easy bruisability

Neurologic

Neuropathy, sciatica

BULIMIA NERVOSA

Dental

Caries, mouth sores, tooth enamel loss, sialadenosis

Gastrointestinal

Heartburn, bloody diarrhea (in laxative abusers)

Endocrine

Oligomenorrhea, amenorrhea, weakness

Cardiovascular

Chest pain, fainting

Hematologic

Easy bruisability

Patients who abuse laxatives often choose ones with stimulant properties, eg, bisacodyl, cascara, or senna

Some purge and abuse laxatives, diuretics, or other medicines (eg, syrup of ipecac); others do not purge but exercise excessively or restrict their eating between binges. With time, they may vomit reflexively. Patients who abuse laxatives often choose ones with stimulant properties, including bisacodyl, cascara, or senna.¹³ Use of diuretics and diet pills is less common.

'Eating disorder not otherwise specified'

Patients whose eating disorder cannot be cat-

egorized precisely as anorexia nervosa or bulimia nervosa may fall under "eating disorder not otherwise specified," a diagnosis that includes the following criteria¹:

- For women or girls, all of the criteria for anorexia nervosa except for amenorrhea (partial-syndrome anorexia nervosa)
- All of the criteria for bulimia nervosa except that the frequency of binges or purges is less than twice a week for less than 3 months
- Inappropriate compensatory measures after eating a small amount of food, eg, running 7 miles merely for eating a cookie
- Repeatedly chewing and spitting out, but not swallowing, large amounts of food
- Binges without compensatory behaviors for anorexia nervosa, except that weight is maintained within normal limits.

This diagnosis should not be downplayed, as recognizing these symptoms represents a chance to intervene before these behavior patterns become "hard-wired" in an addictive pattern. The most common subtype of eating disorder not otherwise specified, which most internists and gynecologists will encounter, is binge-eating disorder. This often secretive disorder is characterized by obesity and its sequelae.⁹

■ EARLY DIAGNOSIS IS CRITICAL

Early recognition of eating disorders helps prevent more serious problems.^{1,14} Health care providers need to recognize the warning signs, address them aggressively, and screen patients they consider to be at risk.

Most patients do not present with a complaint of an eating disorder but rather with physical symptoms such as amenorrhea, heartburn, bloating, abdominal distention, fatigue, dizziness, fainting, or a stress fracture.¹ Therefore, a thoughtful and detailed evaluation is critical to the diagnosis.

Physical examination and laboratory clues

TABLE 1 lists typical signs and symptoms of anorexia and bulimia, and TABLE 2 lists useful laboratory studies. Although the physical findings and laboratory values are usually remarkably normal in eating disorders, possible abnormalities are:



- If the patient is vomiting, serum bicarbonate may be elevated, with or without metabolic alkalosis, and potassium may be low.
- A patient abusing laxatives may have non-anion-gap acidosis, which may be difficult to detect in the setting of hypokalemia.
- If the patient has consumed a lot of water just before the examination to boost her weight, the specific gravity of the urine may be low.
- Patients who restrict fluids and calories may be ketotic and dehydrated.
- Patients with anorexia nervosa may have euthyroid sick syndrome.¹⁴
- In severe anorexia, the hypothalamic-pituitary-ovarian axis is also affected, with decreases in both follicle-stimulating hormone (FSH) and luteinizing hormone (LH).⁸

Differential diagnosis

Most patients who present with the typical features of anorexia nervosa or bulimia nervosa really do have one of these two conditions. However, many other conditions should also be considered, particularly in a patient who appears concerned about her low weight and overall health. These are, in order of frequency:

- Hypothalamic disorders
- Regional enteritis (Crohn disease)
- Other chronic diseases
- Malabsorption syndrome (celiac sprue)
- Pituitary tumor
- Malignancy
- Tuberculosis of the gastrointestinal tract
- Sheehan syndrome (postpartum pituitary necrosis)
- Simmonds disease (panhypopituitarism).

■ COMPLICATIONS CAN BE SERIOUS

Complications of eating disorders range from minor to fatal. Several studies reported the death rate associated with anorexia to be as high as 14% to 21%,^{8,15} although more recent estimates put it at 4% to 10%.¹⁴ This substantial risk highlights the need for aggressive treatment as soon as the diagnosis is made.¹⁶ Those at greatest risk include anorectic patients who also purge, who are at increased risk of cardiac arrhythmias and suicide.⁹

Every organ system can be affected, depending on the length and severity of the

TABLE 2

Laboratory tests recommended for the evaluation of eating disorders

| |
|--|
| Complete blood cell count |
| Chemistry panel |
| Sedimentation rate |
| Urinalysis |
| Purified protein derivative (tuberculin) |
| Thyroid-stimulating hormone |
| Prolactin, follicle-stimulating hormone, luteinizing hormone |
| Computed tomography of the head in all male patients and in select women |
| Electrocardiography |

restrictive episodes, with problems ranging from pancytopenia to diffuse myalgias with muscle breakdown.

Cardiac dysfunction

Sinus bradycardia is common¹⁷ and is likely the heart's way of adapting to decreased body weight and negative energy balance.^{17,18} It may also be due to exercise,¹⁹ but heart rates of 30 to 40 beats per minute in a malnourished patient should not be passed off as "athlete's heart." More likely, electrocardiography will show low voltage, reflecting less muscle to depolarize and repolarize. A prolonged corrected QT interval is particularly worrisome because it can lead to life-threatening arrhythmias.^{18,19}

Mitral valve prolapse, another consequence of prolonged inadequate nutrition, can occur in one third of patients with anorexia. It is thought to be due to redundancy in the valvular apparatus, which occurs when the chamber size decreases as a result of large weight loss.^{18,19} When weight is regained, the chamber size may increase again,²⁰ and thus the mitral valve prolapse may remit.

Osteoporosis

Eating disorders can lead to endocrine complications, most notably osteoporosis or osteope-

Patients tend to present with the symptoms, not the complaint, of an eating disorder

nia. Osteoporosis in patients with eating disorders manifests as fractures, kyphosis, and bone pain.

Damage to bones, especially during adolescence, may be permanent,^{1,2} and for this reason osteoporosis is one of the most significant complications of amenorrhea and weight loss.^{18,21} From 40% to 60% of a young woman's bone mass accrues during adolescence,²¹ and peak bone mass is attained by age 28 to 29.²² An eating disorder during this period may therefore permanently disrupt the integrity of a patient's bones and puts her at substantial risk for later fractures.

Osteoporosis, once thought to be exclusively a disease of the elderly, has since been recognized to begin much earlier, and it may in fact be prevented at an early age.²¹

Young women with amenorrhea and women with irregular menses, with their accompanying hypoestrogenism, are prime candidates for poor bone health. Low-estrogen states in this condition appear to affect bone formation and bone resorption, with the lack of anticipated bone formation in adolescence playing a significant role in subsequent osteopenia.²¹

The female-athlete triad of disordered eating, osteoporosis, and amenorrhea—seen in women who participate in sports that emphasize a thin physique, such as gymnastics, skating, or ballet—is also common in adolescent girls.^{19,21} Energy input is less than their energy expenditure, resulting in a net energy loss that suppresses nonessential functions, such as reproduction. This results in both hypothalamic amenorrhea and hypoestrogenism. Stress fractures are a bothersome consequence.

Ongoing studies of biochemical markers of bone turnover suggest that resorption is the primary mechanism for osteopenia in older women, while lack of formation may play a bigger role in future osteopenia in younger patients with eating disorders. In healthy young adults, bone mass is maintained in a steady state in which bone formation equals bone resorption. In anorexia, this balance is disrupted: bone resorption increases and bone formation decreases, resulting in a net loss of bone.²¹

Modifiable risk factors for osteoporosis are low intake of calcium and vitamin D, lack

of weight-bearing exercise, low body weight or body mass index, and tobacco use.^{3,21,23} Modifying these risk factors directly affects peak bone mass and helps to prevent overt osteoporosis.

Dual-energy x-ray absorptiometry (DEXA) is the most widely used method to screen and follow bone mass. Osteoporosis usually presents with a fracture in women who were not even aware that they were at risk for this disease in the first place; DEXA provides a way to screen and intervene in girls and women who have been amenorrheic for at least 6 months.

Treatment. Weight gain and resumption of menses are the focus in the treatment of bone loss and the prevention of osteoporosis.^{19,21} In addition, calcium and vitamin D supplementation are warranted. Adequate calcium intake in this population may be difficult to obtain from food sources, given patients' tendency to restrict caloric intake.

Exogenous estrogen augmentation is controversial. Data from postmenopausal women support exogenous estrogen for the prevention of osteoporosis. However, in younger women, most studies show additional benefit in bone mineral density after taking exogenous hormones only in a subcategory of amenorrheic women of very low body weight with anorexia nervosa.²⁴

Amenorrhea

The average American girl has her first period by age 12.5 years, approximately 2 years after the start of puberty (defined as the onset of breast buds and pubic hair). A girl with Tanner stage 4 breast development²⁵ and with normal anatomy should be evaluated if menarche has not occurred more than 2 years since the start of puberty.

Moreover, if a girl has been taking oral contraceptives, withdrawal bleeding might be interpreted as "normal menses" when in fact the patient may be amenorrheic when not taking the pill. Lack of withdrawal bleeding after a progestin challenge (medroxyprogesterone acetate 10 mg orally once a day for 10 days or twice a day for 5 days) in an anatomically normal premenopausal woman is a useful screen for absence of menses. Use of ongoing cyclic medroxyprogesterone acetate diagnosti-

Amenorrhea is a red flag for an eating disorder

cally and therapeutically is suboptimal for those women also in need of contraception.

Secondary amenorrhea is defined as not having a period for 6 months. The causes are many and include pregnancy, excess exercise or other chronic stress, and ovarian and pituitary causes.²⁶ In patients with eating disorders, amenorrhea usually occurs before substantial weight loss.⁸ This condition is especially prevalent in the adolescent and younger female population, and most of these individuals tend not to seek medical attention.

Amenorrhea in anorexia nervosa is related to a decrease in the levels of FSH and LH, which simulates a prepubertal state, with a blunting of FSH and LH secretion as a result of the loss of the pulsatile release of gonadotropin-releasing hormone. As a result, a progesterone challenge to induce menses in these patients usually fails, since there is no lining to shed due to the lack of estrogen.¹⁸

Bulimia, too, is often characterized by irregular menses.²⁶ Historically, amenorrhea associated with eating disorders was thought to be a consequence of severe weight restriction. However, recent studies indicate that body mass index may not be related to amenorrhea. In fact, some research has shown that body mass index is actually higher in women who binge and purge.²⁶ Regardless, amenorrhea in this population should be a red flag for physicians to query their patients about eating disorders.

Gastrointestinal effects

Many patients with anorexia experience constipation and bloating, which are symptoms of delayed gastric emptying.²⁷ The gastrointestinal tract works to absorb any available water and nutrients, but slowed movement allows more time for food to be broken down into gas, giving the sensation of bloating or feeling “big.”

Unfortunately, these symptoms may reinforce starvation behavior, encourage the abuse of laxatives and diuretics, and hinder refeeding attempts. Serious sequelae from constipation are rare, but since it often leads to feeling bloated, the patient may be more likely to resist treatment. These symptoms may be ameliorated with increased food intake and subsequent weight gain.

Patients with bulimia nervosa often experience an increased gastric capacity with concomitant decreased gastric relaxation, delayed gastric emptying, decreased release of cholecystokinin, and changes in enteric autonomic function.²⁷ These findings are often a consequence of repetitive bingeing and purging; however, as with anorexia, these disturbances may work to perpetuate the disease.

Medicines, though rarely used, may ameliorate these side effects and include prokinetic agents such as domperidone, metoclopramide, cisapride (available for compassionate use only at this time), and erythromycin.

Respiratory effects

Prolonged fasting, as in anorexia, weakens muscles. In particular, the muscles of respiration, ie, the diaphragmatic muscles, may be slower to recover,¹⁶ which may result in persistent exercise intolerance due to shortness of breath. Multiple studies have demonstrated pulmonary function test abnormalities in this population.

This finding can be particularly problematic in patients who use exercise to control their weight. They have trained themselves to feel no pain, or to persist in exertion despite pain—ie, to bear up and “push on despite the pain.”

Psychiatric effects

Changes in personality and behavior are apparent throughout the course of eating disorders.⁸ Patients with anorexia nervosa are often depressed, anxious, and irritable. Patients with bulimia nervosa experience guilt and a fear of gaining weight, which often motivates purging following a bingeing episode.

Some research has suggested an association between anorexia nervosa and depression, particularly with respect to starvation and protein malnutrition. Anxiety is another common psychiatric manifestation in patients with eating disorders. In anorexia nervosa, obsessive-compulsive disorder (OCD) has been found to be common.^{8,28} Some experts see eating disorders as “the tip of the iceberg,” the outward manifestation of a separate psychiatric diagnosis such as depression, anxiety disorder, or OCD.

Obsessive-compulsive disorder is common in anorexia

Other research has shown a relationship between substance abuse and bingeing and purging. There are many theories to explain this possible link, but it may in part depend on the coexistence of depression, drug dependence, and personality disorders.

■ GENERAL GOALS AND TYPES OF THERAPY

Overall, the aim of treatment for both anorexia nervosa and bulimia nervosa is to restore and stabilize weight and help modify the patient's eating and thinking.^{29,30} Treatment is difficult, given many patients' persistent denial and refusal to be treated.⁸ Treatment is most successful when it is individualized to the patient and carried out by a team including physicians, mental health professionals, and dietitians.^{1,31}

Both drug therapy and psychotherapy have a role.³ Treatment may be on an outpatient or inpatient basis, or it may involve partial hospitalization.³⁰ Inpatient management should be considered if the patient fails to reach outpatient therapy goals, has a body mass index less than 13, is medically unstable, or has suicidal ideation or other psychiatric issues.⁹

Especially in anorexia, younger patients appear to do better with family therapy or coaching from a parent. It is a long-term process, and restoration of weight is only the beginning. Subjective accounts of patients' causes and reasons for recovery have documented that nonfamilial relationships, either with a partner or a therapist, and maturation on the patient's part help in the recovery process.

Expectations for recovery

Estimates of how many patients recover from eating disorders have been as high as 75% to 85%.³² Yet eating disorders often persist as a chronic condition, and as many as one in four patients with anorexia nervosa displays bulimic tendencies at some point in life.¹⁶ In particular, patients with comorbid psychiatric diagnoses fare less well.³³ The treatment plan needs to take this into account.

■ TREATMENT OF EATING DISORDERS

Treatment of eating disorders is tailored to the patient and can include immediate medical

measures such as rehydration and dietary support, hospitalization for close monitoring, education, short-term and long-term psychotherapy, and sometimes drug therapy.

Refeeding

The cornerstone of medical therapy is to correct dehydration and electrolyte imbalances and to supply the necessary nutrients for the patient to gain weight. The goal is a slow, steady return to a normal weight. Whether or not hospitalization is required, rehydration and renourishment should be done orally whenever possible.

Refeeding syndrome. Patients who are rapidly refeed may go into congestive heart failure. The body becomes "phosphate-depleted" when suddenly asked to make adenosine triphosphate from adenosine diphosphate, a process slowed down when the body is faced with starvation.¹⁸ Other electrolyte imbalances can include hypokalemia and hyperkalemia. Giving supplemental phosphorus (500 mg orally twice a day for 5 days) can help avoid the expected nadir of total body phosphorus expected to occur 12 to 72 hours into refeeding, when the patient is striving to make adenosine triphosphate in the face of total body depletion.

Follow-up

Schedule the patient for frequent follow-up visits to monitor progress, identify any problems, and provide essential health education.¹ At each visit, weigh the patient with the patient wearing a hospital gown and after voiding. If you feel that telling the patient her weight at that time will impede rather than help recovery, ask her to stand on the scale facing backwards so she cannot see the reading.

Discuss any worrisome trends with the patient, such as excessive weight loss or weight gain since the last visit.

Take the blood pressure and pulse with the patient lying down and then sitting or standing, in that order, to assess for bradycardia and orthostatic hypotension. A fall of 10 mm Hg or more in the diastolic blood pressure—or an increase in the pulse by 20 beats per minute when going from horizontal to vertical—defines orthostatic hypotension.²⁵ Physicians who are supervising medical resi-

Treatment is difficult, given patients' persistent denial and refusal to be treated



dents or students should ensure that they use these methods and in the correct order, waiting 2 minutes between readings if the initial readings are abnormal, since normal individuals may show an initial jump that resolves within that time frame. Inaccurate measurement of blood pressure and pulse can easily mask problems of starvation or purging.

Treating bone loss due to anorexia

Estrogen therapy has not been proven to preserve bone in young women with anorexia as it has in postmenopausal women.^{18,34} Restoration of weight, calcium and vitamin D supplementation, and weight-bearing exercise if indicated may help to ameliorate some of the bone damage. Newer agents, such as dihydroepiandrosterone (DHEA), osteocalcin, and bisphosphonates are currently being evaluated. DHEA appears to make patients feel better even before bone gain has occurred, but its use is still experimental.³⁵

The bisphosphonates have a clear role in postmenopausal women; an ongoing study is evaluating their safety and efficacy in younger women (N. Golden, personal communication).

Antidepressants

Advances in drug therapy have been greater for bulimia nervosa than for anorexia nervosa, since anorectic patients may be more unwilling to take prescribed medications. Also, the anorectic body may not make enough serotonin, rendering selective serotonin reuptake inhibitors less effective.^{28,29}

Antidepressants are the only class of medications proven to have any benefit in the treatment of anorexia.²⁸ Selective serotonin reuptake inhibitors are currently the first-line agents, given how well they are tolerated and their safety profile.²⁸ Serotonin affects appetite control, sexual and social behavior, stress responses, and mood.¹ Selective serotonin reuptake inhibitors may increase or decrease food consumption, but because they help improve feelings of well-being, they may relieve obstacles to food intake.

Anecdotally, fluoxetine (Prozac) suppresses appetite while sertraline (Zoloft) may be associated with weight gain despite no obvious change in dietary intake. In addition, SSRIs in higher doses are useful in OCD, which often

coexists in these patients.²⁸

The US Food and Drug Administration has so far approved only fluoxetine to treat bulimia. Higher doses are used than when treating depression; it is common to start at 60 mg/day in adults.¹³

Psychotherapy

Cognitive behavioral therapy, a form of psychotherapy, seeks to correct false beliefs that patients harbor about themselves and, in this case, about food.²⁹ Insight-oriented therapy may be applicable later on in treatment but has less of a role in the initial management when the brain is starved. Family therapy is essential with the younger patient. Group work has a role but also comes with risks; some patients may actually use the time to compete to be “thinnest” or pick up tips on other aberrant behaviors.²⁹

Like other psychiatric conditions, bulimia often responds best to a combination of antidepressants and psychotherapy, rather than to one or another alone.¹³

Exercise

Exercise poses a dilemma in eating disorders. On one hand, it may offer psychological benefits, but many patients exercise excessively to control their weight, and it may contribute negatively to the overall bone health of these women.²¹

In patients with eating disorders with heart rates less than 50, exercise and caffeine should be prohibited.³ When exercise is added back, care must be taken to avoid letting the patient exercise compulsively or in excess of what his or her body can handle.

■ MONITOR INTERNET USE, EXPOSURE TO MEDIA

Mass media are a powerful tool: truly, a picture is worth a thousand words. Seeing repetitive images of too-thin models in magazines, on television, and in films can foster abnormal body-image thoughts in susceptible individuals.

Monitoring the patient's use of the Internet is critical. Almost half of teenagers worldwide have access to the Internet,²³ and the emergence of this powerful medium has exacerbated the situation. Web sites that pro-

Younger patients respond to family therapy or coaching from a parent

mote and encourage disordered eating (using words like “pro-ana” for pro-anorexia or “pro-mia” for pro-bulimia) have proliferated in the last several years.³⁶ These sites promote eating disorders as lifestyle choices, not diseases.²³

■ PROGNOSIS

Eating disorders are life-threatening illnesses with serious medical sequelae. With early

recognition and prompt and aggressive treatment, the patient can return to a normal life, without compromising future fertility. Patients who fall into the “chronic” category are at greater risk of osteopenia, of prolonged devotion of time and energy to eating disorders to the exclusion of other aspects of life, and of premature death due to their illness. ■

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■ REFERENCES

1. Rome ES, Ammerman S, Rosen DS, et al. Children and adolescents with eating disorders: the state of the art. *Pediatrics* 2003; 111:e98–108.
2. Anorexia nervosa—part I. How the mind starves the body, and what can be done to prevent it. *Harv Ment Health Lett* 2003; 19:1–4.
3. Rome ES. Eating disorders in adolescents and young adults: what’s a primary care clinician to do? *Cleve Clin J Med* 1996; 63:387–395.
4. The McKnight Investigators. Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study. *Am J Psychiatry* 2003; 160:248–254.
5. Neumark-Sztainer D, Story M, Hannan PJ, Perry CL, Irving LM. Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders. *Arch Pediatr Adolesc Med* 2002; 156:171–178.
6. Neumark-Sztainer D, Croll J, Story M, Hannan PJ, French SA, Perry C. Ethnic/racial differences in weight-related concerns and behaviors among adolescent girls and boys: findings from Project EAT. *J Psychosom Res* 2002; 53:963–974.
7. Neumark-Sztainer D, Hannan PJ, Story M, Perry CL. Weight-control behaviors among adolescent girls and boys: implications for dietary intake. *J Am Diet Assoc* 2004; 104:913–920.
8. Gross M. Anorexia nervosa: an overview. *Cleve Clin Q* 1983; 50:371–376.
9. Guarda AS, Redgrave GM. Eating disorders: detection, assessment and treatment in primary care. *Adv S Med* 2004; 4: 468–475.
10. Bulik CM, Devlin B, Bacanu SA, et al. Significant linkage on chromosome 10p in families with bulimia nervosa. *Am J Hum Genet* 2003; 72:200–207.
11. Munoz MT, Argente J. New concepts in anorexia nervosa. *J Pediatr Endocrinol Metab* 2004; 17(suppl 3):473–480.
12. Becker AE, Burwell RA, Gilman SE, et al. Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *Br J Psychiatry* 2002; 180: 509–514.
13. Mehler P. Bulimia nervosa. *N Engl J Med* 2004; 349:875–881.
14. Pritts SD, Susman J. Diagnosis of eating disorders in primary care. *Am Fam Physician* 2003; 67:297–304.
15. Keel PK, Dorer DJ, Eddy KT, Franko D, Charatan DL, Herzog DB. Predictors of mortality in eating disorders. *Arch Gen Psychiatry* 2003; 60:179–183.
16. Birmingham CL, Tan AO. Respiratory muscle weakness and anorexia nervosa. *Int J Eat Disord* 2003; 33:230–233.
17. Galetta F, Franzoni F, Prattichizzo F, Rolla M, Santoro G, Pentimone F. Heart rate variability and left ventricular diastolic function in anorexia nervosa. *J Adolesc Health* 2003; 32:416–421.
18. Mehler PS, Krantz M. Anorexia nervosa medical issues. *J Womens Health (Larchmt)* 2003; 12:331–340.
19. Rome ES, Ammerman S. Medical complications of eating disorders: an update. *J Adolesc Health* 2003; 33:418–426.
20. Gottdiener JS, Gross HA, Henry WL, Borer JS, Ebert MH. Effects of self-induced starvation on cardiac size and function in anorexia nervosa. *Circulation* 1978; 58:425–433.
21. Golden NH. Osteopenia and osteoporosis in anorexia nervosa. *Adolesc Med* 2003; 14:97–108.
22. Recker RR, Davies KM, Henders SM, Heaney RP, Stegman MR, Kimmel DB. Bone gain in young adult women. *JAMA* 1992; 268:2403–2408.
23. Golden NH, Katzman DK, Kreipe RE, et al. Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. *J Adolesc Health* 2003; 33:496–503.
24. Klibanski A, Biller BM, Schoenfeld DA, Herzog DB, Saxe VC. The effects of estrogen administration on trabecular bone loss in young women with anorexia nervosa. *J Clin Endocrinol Metab* 1995; 80:898–904.
25. Swartz MH. *Textbook of Physical Diagnosis: History and Examination*. 2nd edition. Philadelphia: WB Saunders, 1994: 246.
26. Selzer R, Caust J, Hibbert M, Bowes G, Patton G. The association between secondary amenorrhea and common eating disordered weight control practices in an adolescent population. *J Adolesc Health* 1996; 19:56–61.
27. Bruce KR, Koerner NM, Steiger H, Young SN. Laxative misuse and behavioral disinhibition in bulimia nervosa. *Int J Eat Disord* 2003; 33:92–97.
28. Kim SS. Role of fluoxetine in anorexia nervosa. *Ann Pharmacother* 2003; 37:890–892.
29. Anorexia nervosa. Part II. *Harv Ment Health Lett* 2003; 19:5–7.
30. Clark S, Oxbrough J, Smith P, Rowan P. Anorexia nervosa and the efficacy of an eating disorder service. *Nurs Times* 2003; 99:34–36.
31. Chan Z. Partnership works: the complexity of anorexia demands multidisciplinary care. *Nurs Stand* 2003; 17:20–21.
32. Johnson CL, Lund BC, Yates WR. Recovery rates for anorexia nervosa. *Am J Psychiatry* 2003; 160:798.
33. Ward A, Campbell IC, Brown N, Treasure J. Anorexia nervosa subtypes: differences in recovery. *J Nerv Ment Dis* 2003; 191:197–201.
34. Jamieson MA. Hormone replacement in the adolescent with anorexia and hypothalamic amenorrhea—yes or no? *J Pediatr Adolesc Gynecol* 2001; 14:39.
35. Gordon CM, Grace E, Emans SJ, et al. Effects of oral dehydroepiandrosterone on bone density in young women with anorexia nervosa: a randomized trial. *J Clin Endocrinol Metab* 2002; 87:4935–4941.
36. Andrist LC. Media images, body dissatisfaction, and disordered eating in adolescent women. *MCN Am J Matern Child Nurs* 2003; 28:119–123.

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