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Eating Disorders

Eating disorders are grouped into 3 categories: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified (EDNOS). Within EDNOS, provisional diagnostic criteria are provided for binge eating disorder.

Anorexia Nervosa

http://healerpoint.com/anorexia-nervosa.html

Anorexia nervosa is characterized by a relentless pursuit of thinness, a morbid fear of obesity, a refusal to maintain a minimally normal body weight, resulting in body weight below the normal range and, in women, amenorrhea. Diagnosis is clinical. Most treatment is with some form of psychologic therapy. Olanzapine may help with weight gain.

Anorexia nervosa occurs predominantly in girls and young women. Onset is usually during adolescence.

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The exact etiology is unknown. Other than being female, few risk factors have been identified. In Western society, obesity is considered unattractive and unhealthy, and the desire to be thin is pervasive, even among children. More than 50% of pre-pubertal girls diet or take other measures to control their weight. Excessive concern about weight or a history of dieting appears to indicate increased risk, and some genetic predisposition probably exists. Studies of identical twins have shown a concordance of < 50%. Family and social factors probably play a role. Many patients belong to middle or upper socioeconomic classes; are meticulous, compulsive, and intelligent; and have very high standards for achievement and success.

Two types of anorexia nervosa are recognized:

- Restricting type: Patients restrict food intake but do not regularly engage in binge eating or purging behavior.
- Binge-eating/purging type: Patients regularly binge, then induce vomiting, misuse laxatives, diuretics, or enemas, or a combination.

Binges are defined as consumption of a much larger amount of food than most people would eat in a similar time period under similar circumstances with loss of control, ie, perceived inability to resist or stop eating.

Pathophysiology

Endocrine abnormalities are common; they include low levels of luteinizing hormone (decreased secretion), low levels of thyroxine (T_4) and triiodothyronine (T_3), and increased cortisol secretion. Menses usually cease. Bone mass declines. In severely undernourished patients, virtually every major organ system may malfunction.

Dehydration and metabolic alkalosis may occur, and serum K may be low; all are aggravated by induced vomiting and laxative or diuretic use.

Cardiac muscle mass, chamber size, and output decrease; mitral valve prolapse is commonly detected. Some patients have prolonged QT intervals (even when corrected for heart rate), which, with the risks imposed by electrolyte disturbances, may predispose to tachyarrhythmias. Sudden death, most likely due to ventricular tachyarrhythmias, may occur.

Symptoms and Signs

Anorexia nervosa may be mild and transient or severe and long-standing. Most patients are lean yet are concerned about body weight and restrict food intake. Preoccupation and anxiety about weight increase, even as emaciation develops.

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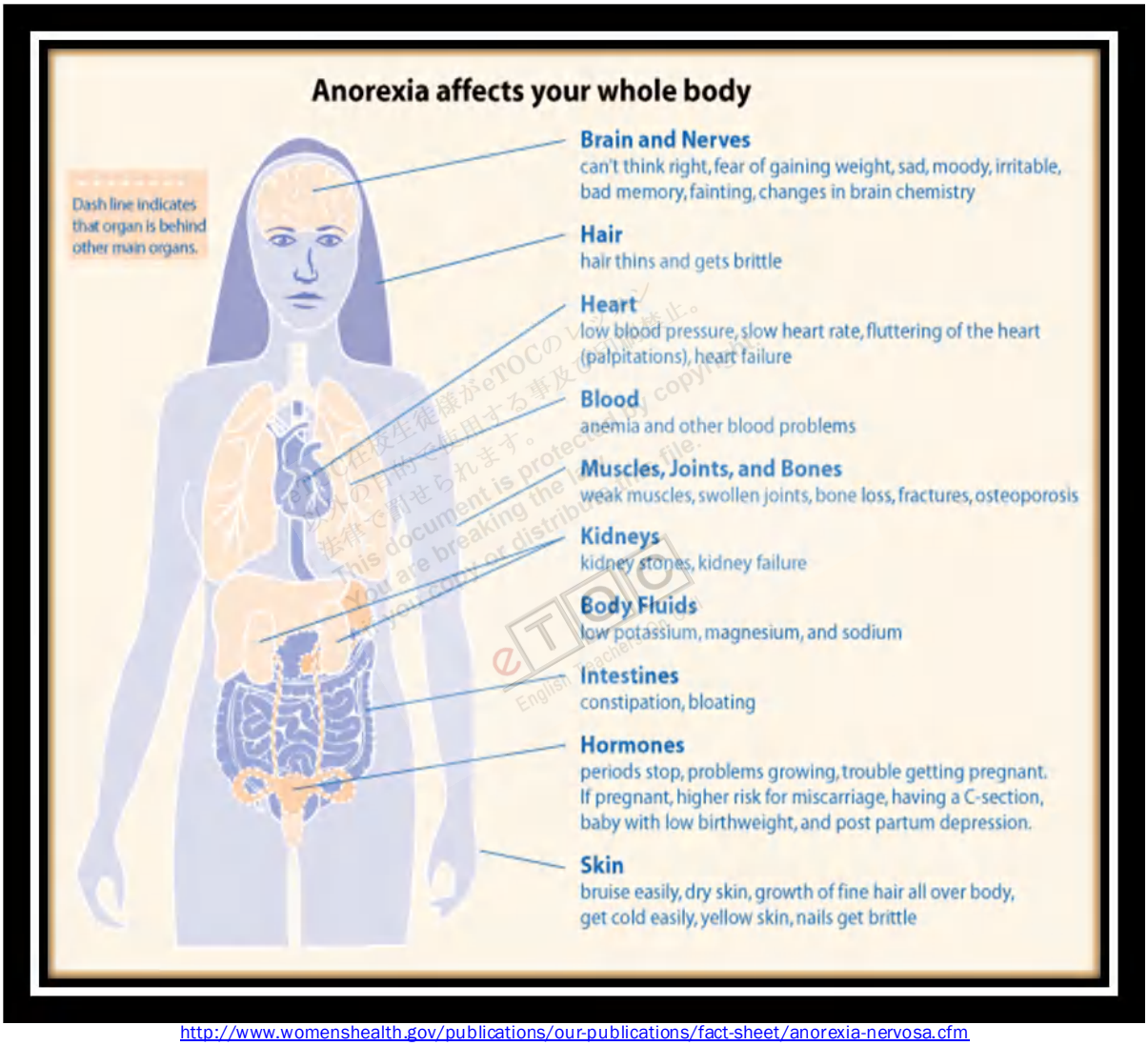
Anorexia is a misnomer because appetite remains until patients become cachectic. Patients are preoccupied with food:

- They study diets and calories.
- They hoard, conceal, and waste food.
- They collect recipes.
- They prepare elaborate meals for other people.



http://www.womenshealth.gov/publications/our-publications/fact-sheet/anorexia-nervosa.cfm

Patients are often manipulative, lying about food intake and concealing behavior, such as induced vomiting. Binge-eating/purging occurs in 30 to 50% of patients. The others simply restrict their food intake.

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Many anorectics also exercise excessively to control weight. Even patients who appear cachectic tend to remain very active (including pursuing vigorous exercise programs), are free of symptoms of nutritional deficiencies, and have no unusual susceptibility to infections.

Reports of bloating, abdominal distress, and constipation are common. Patients usually lose interest in sex. Depression occurs frequently.

Common physical findings include bradycardia, low BP, hypothermia, lanugo hair or slight hirsutism, and edema. Body fat is usually greatly reduced. Patients who vomit frequently may have eroded dental enamel, painless salivary gland enlargement, and an inflamed esophagus.

Diagnosis

- Clinical criteria

Denial is a prominent feature, and patients resist evaluation and treatment. They are usually brought to the physician's attention by family members or by intercurrent illness.

Clinical characteristics include the following:

- Body weight ≤ 85% of expected weight (with a BMI of < 17.5 kg/m²)
- Fear of obesity
- Denial of illness (body image disturbance)
- Amenorrhea in females

Patients should otherwise appear well. The key to diagnosis is eliciting the central fear of fatness, which is not diminished by weight loss.

Differential diagnosis: Another mental disorder, such as schizophrenia or primary depression, may cause similar findings.

Rarely, a severe physical disorder may cause substantial weight loss. Disorders to consider include malabsorption syndromes (eg, due to inflammatory bowel disease or celiac sprue), new-onset type 1 diabetes, adrenal insufficiency, and CNS tumors. Amphetamine abuse may cause similar symptoms.

Prognosis

Without treatment, mortality rates approach 10%; unrecognized mild disease probably rarely leads to death. With treatment, half of patients regain most or all of lost weight and reverse any endocrine and other complications. About one fourth have intermediate outcomes and

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may relapse. The remaining one fourth have a poor outcome, including relapses and persistent physical and mental complications.

Treatment

- Nutrition supplementation
- Psychologic therapy (eg, cognitive-behavioral treatment)
- For adolescents, family therapy

Treatment may require life-saving short-term intervention to restore body weight. When weight loss has been severe or rapid or when weight has fallen below about 75% of ideal, prompt restoration of weight becomes critical, and hospitalization should be considered. If any doubt exists, patients should be hospitalized. Removing patients from their home sometimes reverses a downhill course, but psychiatric treatment is also required.

Nutritional therapy, which begins by providing about 30 to 40 kcal/kg/day, can produce weight gains of up to 1.5 kg/wk during inpatient care and 0.5 kg/wk during outpatient care. Oral feedings are best, but very resistant, undernourished patients occasionally require nasogastric feedings. Loss of bone mass should be treated with elemental Ca 1200 to 1500 mg/day, vitamin D 600 to 800 IU/day, and, if severe, a bisphosphonate.

Once nutritional, fluid, and electrolyte status has been stabilized, long-term treatment begins. Outpatient psychologic therapy is the cornerstone of treatment. Cognitive-behavioral therapy is the modality of choice, done over a period of 1 yr for weight-restored patients and up to 2 yr for low-weight patients. Results are best in adolescents who have had the disorder < 6 mo. Family therapy, particularly using the Maudsley model, is useful for adolescents. This model has 3 phases:

- Family members are taught how to refeed the adolescent (eg, through a supervised family meal) and thus restore the adolescent's weight (in contrast to many approaches, this model does not assign blame to the family or the adolescent).
- Control over eating is gradually returned to the adolescent.
- After the adolescent is able to maintain the restored weight, therapy focuses on engendering a healthy adolescent identity.

Treatment is complicated by patients' abhorrence of weight gain, denial of illness, and manipulative behavior. The physician should attempt to provide a calm, concerned, stable relationship while encouraging a reasonable caloric intake.

Although psychologic therapy is primary, drugs are sometimes used. Second-generation antipsychotics (eg, olanzapine 10 mg po once/day) may help produce weight gain and

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relieve the morbid fear of obesity. Fluoxetine, beginning with 20 mg once/day, may help prevent relapse after weight has been restored.

Reference: http://www.merckmanuals.com

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