

Eating Disorders

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EATING DISORDERS - gross disturbances in eating behavior that usually begin during adolescence.

ANOREXIA NERVOSA

- **obsessional weight loss** without identifiable organic cause.
 Anorexia is misnomer - appetite remains until patient becomes cachectic!
- Richard Morton first described anorexia nervosa in 1689 as condition of "Nervous Consumption" caused by "sadness, and anxious Cares."

CLINICAL FEATURES

- 1) **refusal to maintain** minimally normal **body weight** (weight is at least 15% less than expected for given height)
 - 2) intense **fear of gaining weight** (even when significantly underweight).
 - 3) significant **disturbance in perception of body** shape or size - appropriate body weight is perceived as excessive or low body weight is perceived as appropriate (in some cases, may border on delusional).
 - 4) **amenorrhea** (absence of at least 3 consecutive menstrual cycles) - required for diagnosis in females!
- onset - early adolescence ÷ early adulthood (average 13-14 yrs); onset often preceded by period of mild obesity or mild dieting.
 - illness denial is common (anorectic persons are notoriously unreliable informants!).
 - self-report questionnaires provide more detailed information for diagnosis and degree evaluation.

SUBTYPES

1. **Restricting type** (≈ 50%) – weight loss accomplished primarily through **dieting, fasting, excessive exercise**;
 - patients are more likely to be more *obsessional*, more *socially awkward*, and more *isolated*.
2. **Binge-eating / purging type** (≈ 50%) – patients regularly engage in **binge-eating** → **purging** behavior (self-induced vomiting, laxatives, diuretics, enemas; most individuals engage in these behaviors at least weekly).
 - patients are more likely to have *problems with impulse control* (e.g. substance use disorder, emotional lability, sexual activity), have had *illness longer*, and are somewhat *heavier*.

ASSOCIATED FEATURES

- 1) many anorectics are **high achievers** with above average intelligence.
 - 2) **overactivity** (even in cachectic patients!), obsessive-compulsive features* (related and unrelated to food; e.g. obsessions and rituals connected with food and food preparation).
 - 3) organic brain symptoms (e.g. cognitive slowing, apathy, dysphoria); depressed mood*, irritability, insomnia
 - 4) secretiveness, social withdrawal, concerns about eating in public, limited social spontaneity
 - 5) diminished interest in sex.
 - 6) extreme behavioral rigidity and inflexibility, feelings of ineffectiveness, strong need to control one's environment, overly restrained initiative and emotional expression.
- *most common in cases with early-onset (i.e. age 7-12 yrs)

Comorbid psychiatric disorders:

- 1) substance abuse/dependence
- 2) mood disorders (unipolar and bipolar; → suicide)
- 3) social phobia
- 4) obsessive-compulsive disorder
- 5) personality disorders (borderline personality is predominant axis II pathology associated with binge-eating/purging type; also cluster C has been associated with anorexia nervosa)

Three **personality types** are classically cited as preceding onset of anorexia nervosa:

- a) **obsessive-compulsive** (perfectionistic).
- b) **histrionic** (individuals overly sexualize relationships).
- c) **schizoid** or **schizotypal** (individuals are prone to odd behavior).

PHYSICAL EXAMINATION

- findings reflect metabolic slowing, fluid and electrolyte disturbances, alterations in multiple endocrinologic axes, and organic brain symptoms:

Physical complications are often presenting feature!

1. Significantly reduced **weight** (up to point of **emaciation**).
2. **Dehydration** (→ prerenal failure, renal stones), rebound **peripheral edema**.
3. **Lowered metabolic rate** - (profound) **bradycardia**, low **BP**, **hypothermia**; predisposition to arrhythmias (hypokalemia, QT prolongation).
4. Brittle **nails** and dry yellowish* **skin**; skin may develop **lanugo** (downy soft body hair on face, volar forearms, and other surfaces) accompanied by loss of scalp hair.
 *due to carotenemia
5. **Teeth** in patients who engage in vomiting - decalcification of lingual, palatal, and posterior occlusal surfaces; amalgams (resistant to acid) end up projecting above surface of teeth.
6. **GI** - reduced taste, parotid gland inflammation, esophageal trauma / dysfunction, delayed gastric emptying (gastric dilatation → rupture), peptic ulcers, superior mesenteric artery syndrome, malabsorption, duodenal dilation, jejunal dilation, damage to myenteric plexus due to chronic laxative use, pancreatitis, hepatitis.
7. **Endocrine**: abnormal **hypothalamic-pituitary-gonadal axis** (prepubertal or early pubertal patterns of LH secretion → abnormal menses, delayed puberty), abnormal **hypothalamic-pituitary-adrenal axis** (cortisol secretion↑).

INSTRUMENTAL EXAMINATION

- semistarvation, induced vomiting, abuse of laxatives, diuretics, and enemas can affect most major organ systems → abnormal laboratory findings.

- **body mass index** ↓, low body fat percentage (up to point of emaciation).
- **blood**: pancytopenia, Na ↓, K ↓, Mg ↓, phosphate ↓, hypochloremic metabolic alkalosis; protein and albumin concentrations are normal!; **serum carotene** ↑ (rare in other causes of weight loss!)
- **neuroimaging**: **ventricular enlargement**, **decreased gray and white matter** (generally normalize with weight gain).
- **bones**: osteoporosis.

EPIDEMIOLOGY

- 90-95% cases occur in **females**.
- PREVALENCE (fully meeting DSM-IV criteria) - **0.5-1% females** in late adolescence and early adulthood.
- INCIDENCE highest among **whites** (esp. Jewish, Italian) and increases.
- special risk group - **competitive athletes** (ballet, gymnastics, figure skating, distance running, bodybuilding, wrestling).
- **familial pattern** has been noted.
- far more prevalent in **industrialized societies** where food is abundant and thinness is measure of feminine attractiveness; many patients belong to **middle or upper socioeconomic classes**.

ETIOLOGY

- combination of **genetic**, **neuroendocrine**, **physiological**, and **psychosociological** influences.

Specific **genetic factors** appear to be important in etiology (disorder **runs in families**; concordance in identical twins > 50%).

Neuroendocrine - substantial number of abnormalities of hormone regulation have been described.

- **hypothalamic hypothesis** - hypothalamic abnormalities (e.g. neurotransmitter disturbances) are primary cause of dysfunctional eating and neuroendocrine dysregulation.
- **starvation hypothesis** - abnormal hormone and neurotransmitter regulation result from reduced caloric intake (i.e. adaptation to starvation state).

Physiological - act to sustain fasting: delayed gastric emptying.

Psychological:

- **psychodynamic theories - transactional theory**: anorexia is failure to separate, individuate, and develop autonomy from primary caregivers (i.e. child's refusal to eat is overridden by parent's need to feed child → eventually, child cannot regulate her own eating and becomes dependent on her environment).
- **psychoanalytic theory - fear of sexuality**: symptoms serve as defense in fear of and resistance to growing up (i.e. fear sexuality, menarche, and pregnancy, and starve themselves to remain prepubertal); **oral impregnation** - girl believes if semen is swallowed she will become pregnant + fear of fatness is rejection of any possible pregnancy.
- **family theories - dysfunctional family**: eating disorders are method used by female patient as cry for help for conflicted and dysfunctional family; adolescent's attempt to begin process of separation and emancipation in overinvolved family or to exert developmentally appropriate autonomy and self-control in rigid, autocratic family is seen as disrupting family system → therefore, regression of child from normal adolescent strivings to preadolescent developmental posture (through symptoms of anorexia nervosa) represents accommodation within family system.
 - **mothers** of anorectic girls often are controlling, allowing their daughters little autonomy; mothers may be fragile in terms of feminine identity and self-esteem, perceiving their pubescent daughters as competitive and threatening.
 - **fathers** of anorectic girls often are obsessive-compulsive - participate in quasi-weight-control activities, such as distance running ("obligate running" among males has been considered by some to be male equivalent to anorexia nervosa); fathers may be fearful of their own sexual impulses toward their daughters, which are heightened by girls' pubertal development.
- **cognitive-behavioral theories - learned behavior** maintained by positive reinforcement (low weight is reinforced by peers and society).
- **media influences**: multitude of social pressures promote dietary restraint (books and magazines touting keys to caloric counting, fashion industry promoting slimness, television and film industry's message that thinness is associated with sexual allure and professional success).

DIFFERENTIAL DIAGNOSIS

1. **Medical conditions**: Addison's disease, hyperthyroidism, any chronic illness (e.g. Crohn disease, ulcerative colitis, rheumatoid arthritis, tbc, diabetes), **neoplasms** (esp. CNS tumors), **superior mesenteric artery syndrome** (it may be cause and also complication of severe weight loss).
2. **Psychiatric conditions**: **schizophrenia** (delusions about food are more bizarre), **depression**, **hysterical noneating** (absence of morbid concern with weight and calories), **amphetamine abuse**.

TREATMENT

Malnutrition correction – first step! (esp. fluid, electrolyte, cardiac status)

- indications for hospitalization: ≥ 20% weight loss, electrolyte, cardiovascular, or neurologic signs.
- removing patient from her home sometimes reverses downhill course.
- 30-40 kcal/kg/day - weight gain should be gradual (1.5 kg/wk during inpatient care and 0.5 kg/wk during outpatient care).
- **self-monitoring eating diary** is good for follow-ups; family members and roommates can check reliability.
- alimentation should be provided by **nasogastric tube** if patient steadfastly refuses to eat, and should be readministered if food is vomited; if these measures fail → hyperalimentation through **central intravenous line**.
- careful attention to **accuracy of weight measurements** - serial weights should be obtained at same time of day, on same scale, in same garb (preferably hospital gown only), and after voiding.
 - N.B. attempts to pad weight artificially by drinking large quantities of water or concealing objects on body are typical of anorectics!
- urine should be **monitored regularly for ketones** before each meal (information about starvation state).

Pharmacotherapy has limited value:

- 1) **SSRI** (esp. **FLUOXETINE**) can prevent relapse after weight gain.
- 2) **antianxiety agents** before meals - to reduce anticipatory anxiety concerning eating.
- 3) **promotility agents** (e.g. **METOCLOPRAMIDE**) - for bloating and abdominal pains due to gastroparesis and premature satiety.
- 4) **OLANZAPINE** may help with weight gain and relieve morbid fear of obesity.
- 5) **estrogen replacement** to reduce calcium loss in patients with chronic amenorrhea.
- 6) for osteoporosis - **CA** 1200-1500 mg/day, **VITAMIN D** 600-800 IU/day, and, if severe, **BISPHOSPHONATE**.
- 7) for *binge-eating type*, study (Shapiro, 2000) found **TOPIRAMATE** may have favorable effect.

Psychotherapy conducted over extended time frame.

- 2 weeks of monitoring is recommended before beginning psychotherapy (expect some initial resistance!).
- strong therapeutic relationship, based on trust and understanding, is crucial.
- **individual psychotherapy** (particularly *cognitive-behavioral* therapy): positive reinforcement (system of privileges) for eating and negative contingencies for refusing to eat.
- **family** should be involved in treatment (anorectics may need to be watched and instructed for most of day!).
- **group psychotherapy** sometimes can have negative therapeutic effects (e.g. patients may compete for who can be thinnest).

PROGNOSIS

Only 30-50% patients have relatively complete recovery.

Mortality is high!

6-20% patients eventually succumb to disorder – suicide (\approx 50% death cases), starvation, electrolyte issues, cardiac dysrhythmias, infections.

BULIMIA NERVOSA

- recurrent **binge eating (ravenous overeating)** followed by **guilt, depression, and anger at oneself** for doing so.

DSM-IV Criteria for Bulimia Nervosa:

1. Recurrent **episodes of bingeing**, characterized by:
 - 1) consumption of quantity of food that exceeds what normal person would eat during given time period, under similar circumstances.
 - 2) feeling of not having control over eating during episode.
2. Recurrent **inappropriate weight-controlling behavior** (e.g. self-induced vomiting, use of cathartics, excessive exercise)
3. Bingeing and inappropriate weight-controlling behavior, both **at least twice weekly for 3 months**.
4. Self-evaluation unduly influenced by body shape and weight (i.e. persistent **overconcern with weight and body shape**).
5. Disturbance does not occur exclusively during episodes of anorexia nervosa.
6. Two distinct types (how patients compensate for high-calorie binges):
 - purging type** – regular **vomiting** and use of **diuretics** or **cathartics**.
 - nonpurging type** – **caloric restriction** or **exercise**.

- onset - adolescent and young adult females (PREVALENCE 5%-10% college-age females).
- bingeing episodes occur **in secret** with ingestion of *high-calorie food*.
- during episode, **sense of lack of control over eating** exists.
- episode is **terminated** by sleep, abdominal pain, social interruption, or self-induced vomiting.
- **stealing** may be necessary to support expensive eating habit.
- fluctuations in weight with attempts to lose weight (through dieting, exercise, cathartics, diuretics, enemas).
 - N.B. no significant weight loss! (vs. anorexia with binge eating)
- lengthy phases of normal eating.

Associated psychiatric comorbidities: mood and anxiety symptoms, substance abuse, personality disorders.

Medical complications:

1. Metabolic abnormalities (e.g. hypokalemia, hypochloremic alkalosis), anemia
2. Sequelae of induced vomiting - scars on knuckles, dental erosion and caries, parotid gland swelling, chronic sore throats and esophagitis, esophageal rupture.
3. Gastric dilatation and rupture
4. Cardiomyopathy (from long-term ipecac abuse to induce vomiting).
5. Menstrual irregularities

ETIOLOGY

Psychological theories

- a) need to take in something orally (as substitution for maternal deprivation).
- b) short stature children fantasize that eating ravenously can help them grow.
- c) bulimia has been described in psychogenic dwarfism (retarded growth due to emotional neglect).
- d) disorder of self-regulation (high rates of coexistent substance abuse and stealing).

Biologic theories – hypothetical lesion in hypothalamic satiety center.

DIFFERENTIAL DIAGNOSIS

1. **Prader-Willi syndrome**
2. **Klüver-Bucy syndrome**
3. **Kleine-Levin syndrome**
4. **Hypothalamic lesions**
5. **Anorexia nervosa** (includes significant weight loss!)
6. **Binge eating in obesity** (bingeing is not terminated by purging)
7. **Epilepsy**
8. **CNS tumors**

TREATMENT

- treatment-resistant condition; use combination:

1. **Individual psychotherapies** of psychodynamic, behavioral, and cognitive orientations + supportive **group psychotherapy**.
2. **Medications** are not universally indicated: **antidepressants** (SSRI, esp. **FLUOXETINE**)!!!, lithium, carbamazepine, phenytoin.

N.B. antidepressants are more effective in bulimia than in anorexia!

BINGE EATING DISORDER

- **binge eating not followed by inappropriate compensatory behavior** (such as self-induced vomiting or laxative abuse).

- occurs most commonly in obese people (vs. bulimia nervosa).
- affects 2-4% general population (up to 30% among obese people); nearly 50% are men.
- \approx 50% obese binge eaters are **depressed** (especially if they are trying to lose weight) compared with $<$ 5% of obese non-binge eaters.

Most effective treatment - standard behavioral weight-loss program (not only produces weight loss but also controls binge eating).

- **cognitive-behavioral therapy** is effective in controlling binge eating but has little effect on body weight (because of compensatory nonbinge eating).
- **SSRIs** control both binge eating and weight, but discontinuation is frequently followed by relapse.
- **self-help groups** (Overeaters Anonymous, Food Addicts Anonymous) may help some patients.

FEEDING AND EATING DISORDERS OF INFANCY AND EARLY CHILDHOOD

- parents of 2-8-yr-olds are often concerned that child is not eating enough or eating too much, eating wrong foods, refusing to eat certain foods, or engaging in inappropriate mealtime behavior (e.g. sneaking food to pet, throwing or intentionally dropping food).
- if child appears well and growth is within acceptable range, parents should be reassured and encouraged to minimize conflict and coercion related to eating.
 - **Prolonged excessive parental concern** may in fact contribute to subsequent eating disorders!
 - attempts to **force-feed** are unlikely to increase intake; child may hold food in his mouth or vomit.
 - parents should offer meals while sitting at table with family, *without distractions* (such as television or pets), and show little emotion when putting food in front of child.
 - **food should be removed** in 20-30 min without comment about what is or is not eaten.
 - **child should participate in cleaning up** any food that is thrown or intentionally dropped on floor.
 - **restrict between-meal eating** to one morning and one afternoon snack.

PICA

- persistent (for ≥ 1 month) **eating of non-food products** (e.g. dirt, clay, paper, plaster).
- differentiate from practice of mouthing inanimate objects, which is normal at 6-12 months of age.
- etiology:
 - 1) **mental retardation**.
 - 2) **nutritional deficiencies** (esp. iron deficiency \rightarrow craving for ice and nonfood items).
 - 3) **parent-child problems** (esp. repeated, traumatic separations) - ingestion represents response to deprivation and unfulfilled oral needs.
 - 4) **rare neurologic conditions** (e.g. Klüver-Bucy syndrome).
 - 5) **cultural factors** - ingested substance is believed to have magical or medicinal properties.
- complications: **bezoars, lead poisoning**.
- treatment:
 - 1) any *nutritional deficiencies* should be corrected.
 - 2) *dangerous objects* should be removed from child's environment.
 - 3) increase amount of **child stimulation** (most cities offer infant-stimulation programs).
 - 4) **psychotherapy** for child and parents.

RUMINATION DISORDER

- **purposeful expulsion of previously ingested food followed by food re-chewing**.
- potentially fatal disorder.
- most common in **infancy** \div **early childhood** (extremely rarely - in adults, usually with coexistent bulimia nervosa or mental retardation).
- usually occurs when *child is alone* or is attended only peripherally - food and chewing soothe infant when alone, much like special doll or blanket.
- treatment:
 - in-depth **psychotherapy** of one or both parents.
 - infant's **nutritional status** should be closely followed.

BIBLIOGRAPHY for ch. "Psychiatry" \rightarrow follow this [LINK >>](#)