The Eating Disorders
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Chapter 22

Anorexia Nervosa

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INTRODUCTION

Anorexia nervosa as a clinical entity is an evolving concept [1]. Recognized as separate from pituitary failure for several decades, it is now also accepted to be distinct from other psychiatric disorders. Recently, bulimia has achieved recognition as a distinct nosologic category different from anorexia nervosa [2-4]. Refinements in classification will continue as pathophysiologic understanding expands. Additionally, social evolution continues to affect epidemiologic and clinical features. (See chapter by Gordon, this volume.)

Recognizing this continuous change, the aim of this chapter is to provide a description of the clinical features of anorexia nervosa at this point in its evolution. The patient population is currently defined by the diagnostic criteria in table 1 [5].

Following a historical overview, this chapter will consider premorbid factors, clinical features at presentation, cognitive and experiential aspects, differential diagnosis, and prognosis of anorexia nervosa. In conclusion, significant features in the assessment for treatment are discussed.

Many chapters in this volume relate to this one; those that expand on material specifically addressed in this chapter are cross-referenced in the text.

HISTORY

An early description of an anorexia-like syndrome, translated by Hajal [6], dates from the ninth century AD and describes the clinical history of a patient who is the teenaged son of the reigning Khalifah of the Islamic empire. An argument is made that he suffers from a primary refusal to eat or drink, with secondary medical complications. The royal physician, who has a psychological orientation, intervenes after the patient's resistant behavior has thwarted efforts by the family and local physicians to help. Behavioral techniques directed toward weight gain and increased eating are used, which begin the patient's recovery and cure.

Table 22.1 DSM-III-R Diagnosis of Anorexia Nervosa (5)

A.	Refusal to maintain body weight over a minimal normal weight for age and height, e.g., weight loss leading to maintenance of body weight 15% below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15% below that expected.
B.	Intense fear of gaining weight or becoming fat, even though underweight.
C.	Disturbance in the way in which one's body weight, size, or shape is experienced, e.g., the person claims to "feel fat" even when emaciated, believes that one area of the body is "too fat" even when obviously underweight.
D.	In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhea). (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration).

While not identified as such, a condition resembling anorexia nervosa may have been recognized in the 16th century. Loudon [7] contends that the diagnosis of chlorosis, made from then until the mid-20th century, at first consisted of a symptom complex of food avoidance, weight loss, amenorrhea, and depression during adolescence. Some physicians reported the incidence to be predominantly among upper-class females, and it was said to be cured by the beginning of sexual activity. There were often reports of pica and bulimia in chlorotic patients. With the development of microscopic hematology, chlorosis came to be considered to be primarily a form of anemia, and Loudon [7] believes that two diseases were thus serially associated with that diagnostic label.

In a review that follows the elucidation of anorexia nervosa, Lucas [8] distinguishes five eras in its study. These correspond to the approach to medicine during each epoch and reflect current theoretical positions toward psychosomatic illness. In approximate chronological order the eras are:

1. Descriptive era. This era ended in 1914. At first consisting of case reports, including Morton's [9] of 1689, the descriptive period produced the disease entity with Gull's [10] and Lasegue's [11] independent presentations of "hysteric apepsia" and "anorexia hysterique," respectively, in 1868. Further English [12], French [13], and American [14] reports followed, and anorexia nervosa was established as a clinical diagnosis with emotional components and divisible into primary and secondary varieties.
2. Pituitary era. With Simmons [15] description of pituitary cachexia in 1914 applying Virchow's [16] concept of cellular pathology, anorexia nervosa was subsumed under pituitary failure, a rare condition.
3. Rediscovery era. Anorexia's revival as a separate entity began in the 1930s, when more clinical and psychological formulations were made. Refutation of claims that pituitary extracts had helped more patients and better understanding of starvation in anorexic patients allowed for separation from pituitary cachexia [17].
4. Psychoanalytic era. Concurrent with the rediscovery era and continuing through 1960, this orientation reflected the importance of psychoanalytic thinking in America. Freudians formulated anorexia as a rejection of unconscious wishes for oral impregnation. This has been viewed by some as a supportable dynamic, but of limited clincial utility. The analytically- influenced psychosomatic movement brought to attention such features as personality and conflict situations as potential contributors to the disease [1,18].
5. Modern era. Opened by Bruch in 1961, the modern era shows an attempt to synthesize previously opposing viewpoints [19,20]. Bruch accepted anorexia nervosa as a disease entity with the clinical features described in the early reports. She also distinguished anorexia syndromes secondary to other illnesses. For the primary disorder, she elaborated a specific intrapsychic experience, which consisted of body image distortion, disorderd perception of somatic stimuli, and a pervasive sense of ineffectiveness. Current thinking acknowledges hypothalamic and other neuroendocrine abnormalities, while continuing to explore a comprehensive biopsychosocial model. Constitutional factors, developmental events, and prevalent societal values act in concert to produce pathologic dieting, where-upon the effects of starvation worsen the clinical situation. This scheme of a multidetermined disorder accounts for the considerable clinical variations and for the advantages of a multidimensional approach to treatment [21,22].

PREMORBID FACTORS

An anorexic patient could be described as follows: She has been careful to please others, particularly authority figures in the family and at school. Her progress has been characterized by unquestioning adherence to age-appropriate expectations, and in transactions with others she quietly yields. She has sought and received praise, and her family finds her stubborn refusal to eat to be inconsistent with her previous behavior. At presentation she claims to have no control over most aspects of her life, and eating is the one area where she can have control.

While this description may apply to many patients with anorexia nervosa, there remains uncertainty as to the relative importance of constitution, developmental events, and social factors antecedent to the illness. Bruch [23] considers personality features and symptoms to have their roots in much earlier developmental challenges. Stating that the child's self-initiated cues weren't adequately confirmed, Bruch suggests that there is faulty discrimination of sensations such as hunger and other discomforts, which results in feelings of helplessness and discontrol. Throughout childhood, the future anorexic conforms excessively to rules, without exploring her own evolving capacities. This is met by uncritical praise, and the child remains at Piaget's stage of concrete operations. The incapacity for abstract thinking in patients is a cognitive deficit that re-

flects interference in mastery of prior developmental demands. In considering postpubertal personality features, Crisp [24] notes previous reports of obsessional and hysterical personalities in anorexic patients. Also, patients were described as egocentric, sensitive to rejection, timid, introspective, irritable, and hostile-dependent. While such traits might be used in developmental formulations of the patients, they were studied only near or at the time of hospitalization. Blinder [25] distinguishes three aspects of eating and psychopathology: (1) early feeding disturbances related to the mother-child interaction, (2) orality and its impact on personality organization and traits, and (3) specific eating disorders. With this distinction, developmental factors may have more of a role in the genesis of early feeding disturbances and personality traits than in eating disorders proper. These latter are distinguished by specific disturbances of appetite regulation, food preference, and consummatory pattern, with relatively more support for biological antecedents. Finally, Donohoe [26] presents a model for stress-induced anorexia, which acknowledges contributions from the developmental, constitutional, and social contexts, emphasizing intolerable stress experienced by the patient as the common pathway.

The families of anorxia patients have been repeatedly characterized. (See chapters by Liebman and Kramer, this volume.) Bruch [23] describes the family as materially successful, intact, and superficially happy. The family's communication style often minimizes parental and sibling problems, focusing almost exclusively on the anorexia patient's symptoms. There are both marital dissatisfaction and an underlying struggle for power. The father is older, somewhat passive, detached, and often overinvolved in his occupation. The anorexic's mother is omnipresent, allowing little room for individuation. She may show extreme concern for the patient while holding high, rigid expectations, with excessive concern for external appearances. Consistent with this family pathology orientation, Harper [27] revives the term "parentectomy" to emphasize a traditional protective-clinical approach. Citing cases where parents ignored severe deterioration of their daughter's health and examples of patients losing weight while on passes from the hospital, he states that certain parental behaviors actually sustain the illness. Using a family system approach, Liebman et al.[28], in reviewing work of Minuchin and others [29] from the Philadelphia Child Guidance Clinic, identify features of "psychosomatic families": enmeshment, overprotectiveness, rigidity, lack of conflict resolution, and involvement of the child in unresolved marital and family conflicts. The systems approach presumes the identified patient to belong to the family system and that family symptoms are expressed through the patient. Since the patient's illness represents a solution, albeit maladaptive, to family conflict, treatment can reactivate turmoil in the family. Experimental support for this is given by Crisp et al [30], who performed surveys of 44 anorexic patients and families both at evaluation and after combined nutritional-individual-family therapy. Mothers and fathers both scored higher than control parents in "somatic" and"hysteria" scales on the Middlesex Hospital Questionnaire. After restoration of weight, all scores increased, with significant rises in "anxiety" and "phobic" scales for both parents. In poor marriages, maternal "anxiety" and paternal "depression" scales rose significantly. This provides support for the "homeostatic" family model in which conflict is triangulated or routed through the patient.

In reviewing family studies, Yager [31] notes the pitfalls of generalizations. Many assessments are made when the family is in crisis and not in its best-adapted state. Studies have often included only intact families with younger patients and have suffered observer bias. The systems observations are not specific to anorexia families. Finally, a family explanation alone is inadequate in fully explaining the anorexic syndrome, including altered self-perceptions, early-onset amenorrhea, hyperactivity, desire for thinness, and other seemingly stereotypic signs. Case detection of anorexia nervosa in a broader demographic base (age level, social class, ethnic groups) adds to increasing evidence [24,32,33] for more heterogeneity in premorbid characteristics of individuals and families involved.

Precipitants of anorexia nervosa have been described both theoretically and phenomenologically, and stressors can be viewed as developmental stage-specific or nonspecific. As an early observer using the framework of revised psychoanalytic theory, Bruch [23] began with the still-prevalent view of anorexia as a retreat from adulthood, including its attendant responsibilities and sexuality. Crisp et al [34] support this in emphasizing the anorexic (and bulimic) weight "instability" at ranges where reproductive function is normal. The patient strives for a prepubertal state, which explains self-imposed weight barriers. Stressing psychobiologic regression, Blinder [25] speculates that the effectiveness of behavioral treatment reflects a regression to a stage where operant learning was especially important. Bodily preoccupation is viewed by Romeo [35] as a more primitive communication than the verbal skills required of developing adolescents.

Precipitants that have been observed without assertions of etiology include recent weight loss, obesity, teasing by peers or adults, object loss, family disgrace, illness, and sexual challenges [36,37].

PRESENTING FEATURES

Symptoms

A significant clinical feature of patients with anorexia nervosa is the minimization of symptoms and medical complications. Professional attention is sought usually after pressure from family, spouse, school, or employer. Thus, initial complaints may be first expressed by others.

Unusual behavior in proximity to and contact with food, such as food hoarding, food stealing, changed food handling, or very slow eating may be noticed. (See related chapter by Densmore-John, this volume.) The patient may show increased interest in cooking, become an expert chef, and assume most of the family's cooking duties while abstaining from meals. She may be inadvertantly discovered while engaging in secretive binges, inducing vomiting or using laxatives, diuretics, or diet pills. Cuisine is usually restricted in range with progressive refusal of various types of foods, including carbohydrates, fats, and meats. Binge foods may be high-carbohydrate or fatty foods that the patient refuses publicly. Often, patients seek valid nutritional knowledge but use numerous rationalizations (including toxic fears, contemporary health concerns) for limiting their menu. There is increased exercise [38], sometimes amounting to hours each day. School studies may be pursued with increased intensity (but less efficiency), and social contacts may be progressively restricted.

The foregoing behavioral and attitudinal changes may be unnoticed, obscured, or actually praised by the family until the final common pathway of progressive weight loss becomes unavoidably apparent. All of the behaviors are then recognized as manifestations of the patient's perceived life's work, that is, an obesessional preoccupation with weight and appearance. When the marked incongruity between the patients' alleged concern about health and weight, and the reality of her cachetic state becomes painfully apparent, family and friends forcefully demand treatment.

Certain general features of patients at presentation deserve comment. It has been held that anorexics specifically avoid carbohydrate and that they usually have superior nutritional knowledge. Beumont et al [39], in studying diet composition in inpatient anorexics, found that they decreased carbohydrates only in proportion to total caloric reduction, and disproportionately reduced fat, while increasing protein. The mean nutritional knowledge was greater than controls, but 25% of patients knew less than controls. Halmi [40] found among 12 anorexic inpatients at the outset of treatment that the six "binge eaters" had a higher preference for very sweet solutions, while the "restrictors" showed more aversion to both sweets and fats, compared with controls. Huse and Lucas [41] assessed three parameters of diet in anorexics: regularity, quality, and quantity. Beyond noting that the largest such groups of patients (12 of 96) were disordered in all three parameters, no characteristic pattern of cuisine could be defined. There was considerable variety in the categories of vegetarianism, avoidance of red meats, and avoidance/preference of sweets and desserts. Similarly, the invariability of hyperactivity and its use as an indicator of poor prognosis has been questioned by Falk et al [42], who noted a quantitative increase in activity with weight gain in 20 improving hospitalized patients.

Recent reviews [36,43,44] have studied the prevalence of presenting symptoms and signs in anorexia patients by reviewing these in series of patients admitted to hospitals. While diagnostic criteria have been equivalent, the identification of symptoms is impaired, both by patient selection and by patients being unwilling or unable to report symptoms. This is reflected in wide ranges of prevalence, such as 10% to 45% for binge-eating, 31% to 62% for hyperactivity, and 11% to 43% for emesis. Within these limitations, common symptoms in anorexics are shown in table 2.

Table 22.2 Symptoms

Amenorrhea
Abdominal discomfort/constipation
Cold intolerance
Agitation/hyperactivity
Lethargy
Emesis
Binge eating
Insomnia
Dry skin
Brittle hair/nails
Musculoskeletal pain

As discussed in the next section, the patient's perceptions may be distorted, but there is considerable corroboration for many of the common symptoms. Amenorrhea, usually a sine qua non for the diagnosis, has varying onset in relation to severe dieting. Halmi [44] found 73% of patients had amenorrhea at the time of or before food refusal. This compares with 39% in the UCLA series [36] and 50% to 55% in previous studies [44]. Also noted in one follow-up study [45] was a correlation of disturbed psychological states with persistent amenorrhea in normal-weight patients. Hypothermia and cold intolerance have been attributed [46] to reduced tri-iodothyronine commonly found in anorexic patients, but hypothalamic thermoregulatory dysfunction has been proposed as well. Mecklenburg et al [47] found five anorexic patients unable to correct core temperatures to hot and cold challenges, and they

could distinguish them from controls on this basis. Also, the patients failed to show normal autonomic responses of vasoconstriction and shivering. Luck and Wakeling [48] supported the hypothalamic etiology by showing reproducible elevations of preferred temperatures by patients compared with controls. Finally, there is a case report [49] of glucose- and thiamine-dependent hypothermia in an anorexic patient. Gastrointestinal complaints were studied by McCallum et al [50], who identified bloating, nausea, vomiting, heartburn, belching, bulimia, pain, and constipation in 16 anorexia patients. Employing the diagnostic technique of radionuclide gastrography, they demonstrated a significantly slowed gastric emptying time as measured by percent of food retained in the stomach (80% retained at 120 minutes versus 50% for controls, with significantly more retained continuously from 60 minutes onward). This difference was abolished with administration of metoclopramide. Dystrophic changes in the skin and nails, in addition to having a hypothyroid etiology, can be caused by zinc deficiency, as demonstrated by Casper [51] who found decreased serum zinc, copper and iron-binding capacity levels in 30 anorexia patients. Musculoskeletal pains should alert the clinician to the possibility of stress fractures secondary to the osteroporosis prevalent in anorexics [52].

Physical Findings

While many of the physical findings have been attributable to starvation, further elaboration is possible (table 3). The symptom complex of hypothermia, bradycardia, hypotension, skin changes, slowed relaxation phase of deep tendon reflexes, and hypercarotenemia correspond to the "euthyroid sick" state in anorexic and starving patients. In such cases, peripheral conversion of T4 to T3 is decreased without hypothalamic compensation, probably as an adaptation to starvation [36]. Hypotension and arrhythmias can result from decreased cardiac muscle mass and hypokalemia, the latter due to vomiting and laxative abuse [55]. Similarly, tetany, salivary gland enlargement, and dorsal hand scars are indirect complications in the subgroups of patients who induce vomiting [54,57]. Patients in this subgroup may be first seen by dentists, where they may present with irreversible erosion, decalcification, and severe caries [58]. (See chapter by Levinson, this volume).

Laboratory Findings

A number of abnormal laboratory results can be anticipated with the multiplicity of involved systems in anorexia patients. Much of the data is nonspecific. Noting that one might find a laboratory profile including

> "...leukopenia, a relative lymphocytosis, a low serum potassium level, a low serum chloride level, a low fasting glucose level, an elevated SGOT, an elevated LDH, an elevated alkaline phosphatase, an elevated amylase, and an elevated serum cholesterol," [59]

in an anorexia patient, Halmi and Falk [59] reviewed laboratory results in 40 inpatient anorexics. When mean values were calculated, many of these findings were not prominent, but considerable ranges indicated some patients had abnormalities in nearly every category. Milner et al [60], in reviewing 47 inpatients, found a substantial incidence of abnormal laboratory values: decreased hematocrit (in 25% of patients), leukopenia (in 60%), increase SGOT and LDH (in 45% and 15%, respectively), increased total protein and albumin (in 33% and 25%), increased serum cholesterol (in 37%), decreased alkaline phosphatase (in 65%), and increased BUN and creatinine (in 33% and 25%).

Reviews [59,61] indicate that the hematologic/immunologic profile of leukopenia, anemia, mild thrombocytopenia, bone marrow hypoplasia, and decreased complement components (with *decreased* albumin and protein sometimes seen) are reversible responses to starvation and are not accompanied by an increased incidence of infection [62]. Increased liver enzymes seen on admission will sometimes become further increased during refeeding; this may represent fatty infiltration but may also mimic changes seen in kwashiorkor and marasmus [59]. Increased BUN and creatinine may be secondary to a combination of dehydration, a decreased glomerular filtration rate, and increased protein catabolism [55]. Sodium may be decreased or increased (due to polydipsia or dehydration/partial diabetes insipidus, respectively) [59,63] and increased sodium avidity accounts for refeeding-induced peripheral edema [55]. Hypokalemia results from vomiting, diarrhea, and renal losses; vomiting also causes the hypochloremic metabolic alkalosis [59]. ECGs reveal metabolic changes of hypokalemia and can also show low voltage, bradycardia, and nonspecific T-wave changes [46,53]. Finally, Lankenau et al [64] and Pirke and Ploog [65] note that both reversible and initially irreversible cerebral atrophy have been seen in some patients with anorexia nervosa.

COGNITIVE AND EXPERIENTIAL ASPECTS

Formal studies and case reports attempting to define characteristic personality profiles and cognitive deficits in patients with anorexia nervosa have not led to consensus in the interpretation of findings. Comparison of studies is difficult because of differences in stage of treatment, choice of controls, testing instruments, and self-reporting by patients (see below).

Table 22.3 Physical Findings

Halmi (44)	Provenzale (53)	Smith (46)	Andersen (54)	Herzog (55)	Schwabe (36)	Suematsu (56)
Weight loss	Weight loss	Weight loss	Weight loss	Weight loss	Weight loss	Weight loss
Bradycardia 28%	Bradycardia	Bradycardia	Bradycardia	Bradycardia	Bradycardia 36%	Bradycardia 49%
Lanugo 18%		Lanugo	Lanugo		Lanugo 28%	Lanugo 28%
Dependent edema 18%	Dependent edema					Dependent edema 26%
Hypothermia 18%	Hypothermia	Hypothermia	Hypothermia			Hypothermia 46%
	Hypotension		Hypotension		Hypotension 22%	Hypotension 40%
		Dry, inelastic skin			Dry skin 22%	
	Acrocyanosis	Loss of subcutaneous fat	Tetany seizures	Slowed reflex relaxation		Pubic hair loss 5%
		Petechiae Parotid swelling	Arrhythmias Breast atrophy	Hypercarotenemia		Breast atrophy 21%
		Increased caries	Dorsal hand scars			
			Decreased muscle mass			

In a study of 12 anorexics, Skoog et al [66] showed pretreatment MMPI profiles to have peaks in depression, social introversion, and hypochondriasis, which improved significantly after treatment. The pretreatment profile was similar to that of an earlier study [66]. Vandereycken and Vanderlinden [67] showed overall heterogeneity in the MMPI profiles to 40 severely ill inpatient anorexics, but were able to identify certain profiles based on the Eating Attitudes Test (EAT) scores of these patients. Patients with false-negative (normal scores <30) EAT results showed significantly more ego strength and lower psychasthenia, schizophrenia, hysteria, and hypochondriasis on MMPI than those with high (>55) EAT scores. It was concluded that self-reporting studies must consider the effects of denial and also that there may be a subgroup of anorexics who have more severe neuropsychological deficits. This was studied by Fox [68], who compared testing results of 15 anorexic inpatients to those of 15 other psychiatric inpatients without eating disorders. Anorexics had significantly higher digit substitution, significantly lower general information, and significantly lower arithmetic than other psychiatric patients. The author attributes the improved digit substitution to increased mobilization of psychomotor activity and suggests that impaired arithmetic, like impaired body awareness and awareness of illness, could be related to right hemisphere dysfunction. Small et al [69] administered pre- and post-treatment WAIS and Rorschach tests to 27 hospitalized patients. Abnormal arithmetic and Digit Span results indicated poor potential for weight gain, while personality features and thought disturbances were not of prognostic significance. It was suggested that the inability to organize and sustain a cognitive focus made newly-admitted patients inaccessible to therapy. Witt et al [70] found associative learning as measured by the Symbol-Digit Learning Test to be significantly impaired in anorexics compared with matched depressed inpatients, diabetic outpatients, and healthy controls. The impairment correlated with duration of illness. Digit substitution, measured by the WAIS Digit Symbol subtest, was comparable to controls, in contrast to the study by Fox [68].

The intrapsychic experience of patients with anorexia nervosa was delineated by Bruch [71], who posited that patients are unable to acknowledge and identify their own affects and bodily sensations such as hunger. Both types of stimuli are perceived as externally-derived, and restrictive behavior is an autonomy-seeking response to perceived external threat (overwhelming hunger). Crisp [72] agrees that the anorexic's behavior may be experienced by her as adaptive to her needs, and adds that the sexual and maturational challenges of puberty may provide additional externally-perceived threats to the patient. Reliable body image identification could then be related to the extent of self-acceptance a patient has in areas such as autonomy and sexuality. As a measure of autonomy, Basseches and Karp [73] compared field dependence in 16 anorexics, 16 obese subjects and 16 matched controls. Both obese and anorexic patients showed significantly less capacity of autonomous functioning than controls. Leon et al [74] found significantly more negative self-assessment in sexuality, body state, and other parameters among 47 anorexic inpatients compared with controls. Responses indicated patients wanted to lose weight while knowing that they would be more sexually attractive at higher weights. Garner and Garfinkel [75], in reviewing the body image literature, note certain methodological issues, including reliability, construct validity, selection of patients and controls, and the effects of the experimental setting. (See also chapter by Meerman et al, this volume.) "Body image distortion" may refer to body misperception (visualization, unusual body experiences), or to body disparagement (evalaution) with normal perception. Measures of misperception, such as the Movable Caliper Technique, Image Marking Method, and Distorting Photograph Technique have shown considerable variability but have as a whole suggested body image overestimation by anorexics. Hsu [76] notes that body overestimation as an index of body image distortion is neither sensitive nor specific for anorexia nervosa and cannot be considered pathognomonic. Studies that assessed body disparagement [77,78,79], while showing a preference for thinness among both patients and controls, have been supportive of more body disparagement in anorexics.

DIFFERENTIAL DIAGNOSIS

Because of the weight-loss criterion required for the diagnosis of anorexia nervosa [5], bulimia cannot be diagnosed as a disorder in the presence of anorexia. However, the symptom of bulimia (binge-eating) is reported in 45% to 50% of patients with anorexia nervosa [43,80], and self-induced vomiting is present as frequently [43]. Also, 50% of patients with diagnosed bulimia have a history of anorexia nervosa, and it may

Table 22.4 Differential Diagnosis of Anorexia Nervosa

Bulimia	Hyperthyroidism
Affective disorders	Hypothyroidism
Schizophrenia	Adrenal insufficiency
Obsessive-compulsive disorder	Hypopituitarism
Conversion disorder	Inflammatory bowel disease
Personality disorders	Brain tumors

be ten times as prevalent as anorexia in the female population between ages 13 and 40 [82]. Evidence supporting diagnostic distinctions in the form of personality differences [83], family features [84], and parental personalities [85], as well as new similarities [86] continues to be defined. Current nosology would appear to favor at least two separate disorders, (restrictive) anorexia nervosa and (normal-weight) bulimia, with anorexics who binge-eat resembling patients with normal weight bulimia more than they resemble exclusively restrictive anorexics [85]. (See also chapters by Hsu, Pyle and Leichner, this volume.) Thus, Garner et al [85], using self-reporting of demographic, clinical, and psychometric variables in 59 patients with normal-weight bulimia, 59 with the bulimic subtype of anorexia nervosa, and 59 with restrictive anorexia, found the two bulimic groups to be more alike than either was to the restrictive group in the areas of impulse control, perceived family conflict, predisposition to obesity, and possibly proclivity toward affective disorder.

The relationship of affective disorders to eating disorders continues to be extensively studied, with new reports supporting correlations [87-90] as well as new criticism [91] of hypotheses linking affective and eating disorders. With more recognition of distinctions between anorexic and bulimic syndromes, bulimia appears more exclusively related to affective disorders, particularly major depression, than does anorexia. (See also chapters in this volume by Hsu, Chaitin, and Mitchell and Pyle for further discussion of classification, affective disorders, and bulimia, respectively.)

While other Axis I diagnoses can coexist with anorexia nervosa, they can be distinguished from anorexia when the characteristic pursuit of thinness and distorted body image are absent (as in cyclic conversion vomiting [92] and obsessive-compulsive disorder) or when thought disorder, delusions, or hallucinations predominate (as in schizophrenia). Personality disorder diagnoses such as schizoid, borderline, histrionic, and antisocial personality disorder are made in anorexic patients [45] and may be the primary diagnosis for patients in whom anorexia nervosa is only partially or occasionally manifest. (See also chapter by Sacher and Sporty relating eating disturbances to other psychiatric disorders.)

Medical diagnoses may account for some signs seen in anorexia nervosa and are excluded clinically and by laboratory examination. Hyperthyroid patients may show weight loss and hyperactivity, but they also have increased food intake, hyperthermia, heat intolerance, and increased serum thyroid hormones. Hypothyroidism mimics anorexia with symptoms of weakness, constipation, bradycardia, hypothermia, and cold intolerance, but hypothyroid patients often show weight gain, hypoactivity, and increased serum TSH. Adrenal insufficiency may cause bradycardia, hypotension, lethargy, and decreased oral intake, but it also causes hyperpigmentation, decreased intertriginous hair, hyperkalemia, and low serum cortisol. Rarely, pituitary dysfunction will cause amenorrhea, but there will often also be secondary hypothyroidism, adrenal insufficiency, or changes in prolactin if caused by a mass lesion. Inflammatory bowel disease and other causes of gastrointestinal dysfunction may be clinically similar to some manifestations of eating disorders but may be diagnosed by abnormal diarrhea and by laboratory and clinical indications of inflammation. Finally, chronic illnesses such as tuberculosis and malignancies may caused cachexia, but these are not accompanied by the desire for thinness and distorted body image and usually present other clinical signs [46].

PROGNOSIS

A critical comparison of anorexia nervosa outcome studies is difficult. Steinhausen and Glanville [93] reviewed 45 studies, and noted the methodological limitations: diagnostic criteria vary, follow-up duration is sometimes too short (less than four years), and treatment has usually been multimodal with few centers having enough subjects to compare one distinct consistently applied method with another. Data are usually by subjective report and often obtained by telephone or letter instead of by direct interview. Typically, only one or a few indices of improvement such as weight restoration or resumption of menses are used, while different outcome data are obtained if one includes such parameters as resolution of eating distubance and psychosocial improvement. There is variable inclusion of information regarding coexisting or subsequent other psychiatric diagnoses in the subjects. Often, there is a high dropout rate, and conclusions may be made based on atypical samples.

This is exemplified by Vandereycken and Pierloot's [94] observation of a differential outcome among patients who initially did not respond to follow-up inquiries, a group that accounted for 31% of a 128-patient study. They also found significant differences in demographics and outcome between patients who were followed more or less than five years, suggesting that the natural course of the illness can confound comparison of such subgroups. Halmi [45] confirms the above concerns and adds that more standardized forms of data collection are needed.

With these caveats, Steinhausen and Glanville [93] reviewed 45 studies from 1953 to 1981 and found general ranges of follow-up weight restoration and re-

Table 22.5 Outcome in Anorexia Nervosa

	Crisp (43)	Bassoe (95)	Morgan (96)	Hall (97)	Touyz (98)
Number of Patients	102	133	78	50	47
Treatment Population	52% OP 48% IP	34% OP 66% IP	"normally outpatient"	34% OP 66% IP	25% OP 75% IP
Treatment Modality	Mulitmodal	Educational-supportive	Multimodal & Medications	Refeeding & Individual	Refeeding & supportive
Follow-up duration (yr)	4-8	2-4 +	4-8.5	4-12	8.5
"Good" outcome (%)		58	58	36	37
Menstruating (%)	69				55
Normal body weight (%)	76				
"Intermediate" outcome (%)		28	19	36	18
No food preoccupation (%)	33				53
No weight preoccupation (%)	57				
"Poor" outcome (%)	20	14	19	26	10
Deaths (%)	2	0	1	2	2
Lost to follow-up (%)	0	0	0	0	33
Duration of illness among recovered (yr)	4.6				

OP: Outpatient
IP: Inpatient

sumption of menses to be 50% to 70%, with more return of menses after longer follow-up periods. Normalization of eating symptoms occurred in 30% to 70% of patients with the subjectivity of this parameter possibly accounting for the wide range. Recovery of social adjustment, another poorly defined measure, occurred in 50% to 80% of patients. Steinhausen and Glanville [93] considered the overall improvement rate of 30% to 50% to be conservative. Garfinkel and Garner [21], reviewing 724 patients included in relatively long follow-up studies, found 43% recovered, 28% improved, 20% unimproved, and 9% dead. Table 5 shows results in recent outcome studies.

Morgan and Russell [99] developed a 12-point average-outcome scale using self-rating of nutritional status, menstrual function, mental state, sexual adjustment, and socioeconomic state, and from this are derived the outcome categories of "good" (body weight 85% to 115% normal body weight), "intermediate" (body weight sometimes outside 85% to 115% and/or menstrual irregularity), and "poor" (body weight below 85%, amenorrhea) used in the last three studies in table 5 [96,97,98].

The mortality rate in anorexia nervosa has ranged from 0% to 21%, with the majority of series showing rates below 10% [93]. The rate may be still lower in pediatric series [21]. Patients usually die of complications of starvation or suicide, but they may also die of complications of bulimia and other ingestive behavior, such as hypolkalemia-induced cardiac arrhythmias, gastric perforation [100], and ipecac poisoning [101].

Reviews of prognostic indicators [21,22] and recent outcome studies [84,96,97,102-104] show considerable variability in identifiable predictors of outcome. The factors most consistently associated with a good prognosis are early age of onset, short pretreatment duration of illness, and fewer previous hospitalizations. Less reproducible factors include higher socioeconomic class, good relationship with parents, premorbid hyperactivity as a primary means of weight loss, hysterical personal-

ity, improved body image after treatment, and post-treatment phobic symptoms. No indicators of poor prognosis are consistently found, but bulimia, vomiting laxative abuse, severe weight loss, depression, obsessive-compulsive symptoms, psychosis, somatization, and male gender have been suggested in some studies. Recently, early age of onset as a prognostic indicator has been questioned by Hawlay [105], who found similar menstruation, psychosocial adjustment, sexual adjustment, weight, and survival in 21 patients with onset age under 13, followed eight years, as compared with follow-up results in patients with onset at a later age. This may represent recent convergence of improved outcome for both adult and childhood-onset anorexics.

APPROACH TO TREATMENT

Bassoe and Eskeland [95] note:

> Throughout the centuries many types of treatment have been advocated in the treatment of anorexia nervosa: psycotherapy, behavior modification, isolation, bedrest, drugs, acupuncture, endocrine therapy including pituitary transplantation, shock therapy, lobotomy, dialysis and exorcism.

The many therapies reflect the fact that patients with anorexia display disturbances in all of the areas of physical health, psychological and social functioning, family relationships, reproduction, and sexuality. Exclusive use of one treatment modality such as medications, refeeding, individual therapy, or family therapy will fail to address other aspects of the problem. Similarily, termination of treatment after short-term gains while hospitalized ("weight restored") does not comprise a good outcome. It is generally accepted [69,22] that nutritional rehabilitation is required at the outset of treatment to avoid medical complications and permit engagement in therapy. Establishment of a sound therapeutic alliance is important, although this should not be used as a rationale for permitting the patient to avoid refeeding. Regardless of one's assumptions as to the etiology of anorexia nervosa, the adolescent issues of separation and individuation will be present in this population and contribute to family interactions. In most cases, the family should be involved to effect adaptive separation. Although outpatient studies are lacking, recent-onset, mildly symptomatic anorexics may be treated without hospitalization, provided appropriate medical, behavioral, nutritional, and family interventions are possible [22].

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