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ANOREXIA NERVOSA - BULIMIA

"... Like a creature obsessed, neither tasting nor thinking, I burrowed through the cupboards, refrigerator, cookie jar, and freezer. Grabbing fistfuls of Mallomars and brownies, gulping ice cream, Jell-O, and cheese, I was indiscriminate in my gorging. Frenzied, as though possessed by some malevolent phantom, I raced through the larder and could quit only after collapsing in glutted agony. Then, when the spell finally broke, I loathed myself for such weakness and raged at my failure of willpower.

"Staggering to the bathroom, I presented myself for penitence. The mirror mercilessly cast its wrath upon me. My reflection resembled that of a bloat-bellied malnutrition victim. I weighed under 90 pounds. My scrawny arms, bony chest, and spindly legs cried out in protest against the distended abdomen of my cruel binge. The sight nauseated me. No doubt about it, eating was evil. My faith in the virtue of abstinence grew the longer I stood examining myself. It sent me into spasms of remorse over my greed and propelled me into a program of redemption.

"I began by vomiting ..."

Aimee Liu[1]

Anorexia nervosa and bulimia are common syndromes characterized by bizarre eating patterns which become the central focus of the patient's life. Occurring primarily in young women, they represent life-disrupting illnesses for the afflicted and their families and lead to death in a small but significant number of cases. In this review anorexia and bulimia will be considered variant expressions of the same underlying disorder. Although the clinical manifestations and outcome of the two syndromes are distinctive, overlap features suggest that the root disorder is the same: an obsessive fear of being fat. In anorexic patients the primary reactive mechanism is the rigid restriction of food intake; with bulimia, loss of control in the drive to eat is compensated for by induced vomiting and laxative use.

I. History and prevalence

It is generally considered that anorexia nervosa was first described by Richard Morton who in 1689 reported the case of a 17 year old girl who was "like a Skeleton only clad with skin" (1). He concluded that she had "a nervous consumption." The name "anorexia nervosa" appears to have been coined by Sir William Gull. Gull and Charles Lasègue, a French contemporary, both published accurate descriptions of the clinical manifestations. Anorexia nervosa became

[1] Liu, A: Solitaire. Harper Colophon Books, Harper and Row, New York, 1979.

confused with pituitary apoplexy for a number of decades because of Simmonds' report of death by "emaciation" in a woman with pituitary destruction, but the issue was reclarified in 1930 when Berkman published his experience with 117 patients emphasizing that the physiologic abnormalities were due to a psychic disturbance (1,2).

While the true prevalence of anorexia and bulimia are not known, there is a widespread feeling that both are increasing dramatically, especially in developed countries. Crisp and coworkers (3) estimated a prevalence of 1% in upper class adolescent girls in the United Kingdom while Ballot et al (4) estimated a prevalence of 2.9% for school girls in South Africa using a body weight of 20% below standard as the criterion for diagnosis. The problem of prevalence is confounded by the existence of subclinical or covert disease not sufficient to arouse the suspicion of family, friends or physician. Button and Whitehouse (5), using the Eating Attitudes Test, which is thought to discriminate well between subjects with anorexia nervosa and normal female undergraduates, estimated that 5% of postpubertal females have a subclinical form of the disease.

1. Lucas AR: Toward the understanding of anorexia nervosa as a disease entity. Mayo Clin. Proc. 56:254-264, 1981.
2. Berkman JM: Anorexia nervosa, anorexia, inanition, and low basal metabolic rate. Am. J. Med. Sci. 180:411-424, 1930.
3. Crisp AH, Palmer RL, Kalucy RS: How common is anorexia nervosa? A prevalence study. Brit. J. Psychiat. 128:549-554, 1976.
4. Ballot NS, Delaney NE, Erskine PJ, Langridge PJ, Smit K, Van Niekerk MS, Winters ZE, Wright NC: Anorexia nervosa - a prevalence study. S. Afr. Med. J. 59:992-993, 1981.
5. Button EJ, Whitehouse A: Subclinical anorexia nervosa. Psychol. Med. 11:509-516, 1981.

II. Diagnosis

The diagnosis of anorexia nervosa - bulimia is usually not difficult from a clinical standpoint when the full syndromes are present. Since 1972, the criteria of Feighner et al (6) have been most widely used in research studies on anorexia. These are shown in Table 1.

Table 1 (ref 14)

Table 1. Diagnostic Criteria for Anorexia Nervosa*

Age of onset before 25 years of age
Anorexia with accompanying weight loss of at least 25% of original body weight
A distorted implacable attitude toward eating food, or weight
Denial of illness
Enjoyment in weight loss
A desired body image of thinness
Unusual hoarding or handling of food
No other medical illness that could, per se, account for the weight loss
No other known psychiatric disorder
At least two of the following:
Amenorrhea
Lanugo
Bradycardia
Periods of overactivity
Episodes of bulimia
Emesis (may be self-induced)

The criteria listed by the Diagnostic and Statistical Manual of Mental Disorders III of the American Psychiatric Association (7) are less specific:

A. Intense fear of becoming obese, which does not diminish as weight loss progresses.
B. Disturbance of body image, e.g., claiming to "feel fat" even when emaciated.
C. Weight loss of at least 25% of original body weight or, if under 18 years of age, weight loss from original body weight plus projected weight gain expected from growth charts may be combined to make the 25%.
D. Refusal to maintain body weight over a minimal normal weight for age and height.
E. No known physical illness than could account for the weight loss.

Both sets of criteria have been attacked as being too rigid, especially the 25% requirement for weight loss (8,9). Children in particular may be severely ill without having lost a fourth of the original body weight. From a clinical standpoint the major issues are an intense fear of becoming fat, a history of major weight loss either presently (classic anorexia nervosa) or in the past (bulimia) , the absence of organic illness sufficient to cause weight loss, the absence of primary psychiatric illness leading to loss of interest in eating and the presence of unusual eating habits, either extreme dieting or gorging/regurgitation. The absence of one or more of the other features mentioned by Feighner et al should not exclude the diagnosis, including amenorrhea which is perhaps the most invariant of the biologic expressions of classic anorexia nervosa (9). Although a disturbance of body image is common, recent studies indicate that this is not a very specific finding as discussed below.

An entirely deviant set of diagnostic criteria were developed by Norris (10) based on his evaluation of 54 consecutive patients admitted to a treatment center for anorexia in Johannesburg, South Africa (Table 2).

Table 2 (ref 10)

TABLE DIAGNOSTIC CRITERIA FOR PRIMARY ANOREXIA NERVOSA (CRITERIA PRESENT IN 75% OR MORE OF PATIENTS)

	Number	Number present	%
'Positive' perception of family	54	53	98
Psychosexual unawareness or guilt (aged 16 +)	39	35	90
Onset of illness between 13 - 15 years	54	47	87
Shy, obsessional and compulsive, compliant or dependent (any 2)	54	47	87
'Enmeshment' with a parent	28	24	86
Close, intact family denying conflict	28	24	86
Above-average intelligence	44	35	82
More female children than males	54	44	81
Change of personality at or before onset	50	38	76
'Dominant' mothers	28	21	75
Mothers anxious and overprotective or indulgent and self-martyring	28	21	75

The idea was to establish definitive criteria that could be used by the average practitioner to select anorexic or potentially anorexic subjects from teenagers simply dieting for cosmetic reasons. The focus was on the family structure which was intact but unhealthy. All patients fully evaluated had at least 8 of the 11 criteria. In my opinion the study is of more interest for its evaluation of family relationships than from its utility in diagnosis.

Strict criteria for the diagnosis of bulimia have not been defined, although the clinical presentation is quite characteristic as will be discussed. In general the picture is that of a heavier anorexic patient whose weight loss is not sufficient to produce the usual physiologic and physical accompaniments of malnutrition or cachexia.

6. Feighner JP, Robins E, Guze SB, Woodruff RA Jr, Winokur G, Munoz R: Diagnostic criteria for use in psychiatric research. Arch. Gen. Psychiatry 26:57-63, 1972.
7. Diagnostic and Statistical Manual of Mental Disorders III, American Psychiatric Association, Washington, D. C., 1980.
8. Irwin M: Diagnosis of anorexia nervosa in children and the validity of DSM-III. Am. J. Psychiatry 138:1382-1383, 1981.
9. Kirstein L: Diagnostic issues in primary anorexia nervosa. Int. J. Psych. Med. 11:235-244, 1981-82.
10. Norris DL: Clinical diagnostic criteria for primary anorexia nervosa. An analysis of 54 consecutive admissions. S. Afr. Med. J. 56:987-993, 1979.

III. Clinical picture

1. Anorexia nervosa. The literature describing the clinical syndrome of classic anorexia nervosa is massive. The usual features are well delineated in four recent reviews which provide access to the literature for specific points the reader may wish to check (11-14). An overview of the clinical picture is shown in Table 3.

Table 3 (ref 14)

Table . Clinical Features of Anorexia Nervosa

Demographic and historical features
Predominance of women
Onset in late childhood and adolescence
Predominantly white
Middle to upper class families
History of overweight period
Preoccupation with food, nutrition
Preoccupation with physical exercise
Symptoms
Amenorrhea, primary or secondary, in almost 100% of patients
Constipation often with complaints of abdominal pain
Cold intolerance
Agitation or lethargy
Emesis
Physical findings
Cachexia
Skin abnormalities; increased lanugo-like body hair
Bradycardia
Hypotension
Hypercarotenemic skin
Peripheral edema
Hypothermia
Laboratory findings
Decreased thyroid function test values
Abnormalities in cortisol and growth hormone secretion
Decreased gonadotropins
Hypercarotenemia
Other evidence of hypothalamic dysfunction
Elevation of blood urea nitrogen

a. Demographic features. Anorexia nervosa is primarily a disease of women, only 4-6% of affected subjects being males. The age of onset ranges from prepuberty to the early 30's. The most common time of appearance is 4-5 years after menarche (13). The disease appears to occur primarily in Caucasians, usually in families from the middle or upper class. A disproportionate number are of Jewish heritage. There is an increased risk for anorexia in parents and siblings of index cases (15). In the families studied by Crisp et al (13), 29 of 102 had a parent or sibling who was at least 20% below the mean weight of a matched population. Only 10 family members were as much as 20% overweight. Since the prevalence of obesity in the general population is greater than 10%, it can be concluded that fatness in families is not excessive. This is important since in the past it was considered that anorexia nervosa might have something to do with a reaction to obesity in a parent. In a high percentage of cases overt onset of disease appears to be related to a stressful event in the subject's life.

b. Behavioral characteristics. As has been repeatedly pointed out the term "anorexia" is really inappropriate since there is not true loss of appetite until very late in the course. Patients are not free of hunger; rather, they are obsessed with the fear of being fat such that hunger sensations are ignored or denied. An intense preoccupation with food is usually discernible. Although anorexic patients drastically restrict their own food intake, it is not unusual for them to enjoy preparing elaborate meals for others, collect recipes and hoard food in the home. Most subjects appear knowledgeable on nutritional matters, particularly the caloric content of food, although up to 25% may show lesser insight than matched controls (16). It is usually stated that carbohydrates are avoided preferentially, but carbohydrate intake was found to be normal in the study by Beumont et al (16). Fat intake was decreased and protein content was high. In the series of Crisp et al (13) sporadic dieting usually began about a year prior to start of the disease proper, often at the point where maximal weight was reached.

In order to assist weight loss it is common for patients to exercise excessively, often in ritualistic fashion. A significant percentage induce vomiting and use laxatives or diuretics. Periodic gorging of the type seen in the bulimic variant/phase of the disease may also occur in classic anorexia nervosa.

c. Perceptual abnormalities. It is characteristic of patients with anorexia nervosa to deny illness, at least until the disease is far advanced. Resistance to treatment is profound. Patients deny hunger, fatigue and change in physical appearance. There is no question that affected subjects have a disturbance of body image which makes them see themselves as continually fat. Several types of objective evaluation show the propensity of anorexic patients to overestimate true body size although the capacity to accurately assess other objects is maintained. This image disturbance has been built into a number of formal definitions of anorexia as described above (7). A representative illustration of self-imaging is shown in Fig 1.

Figure 1 (ref 17)

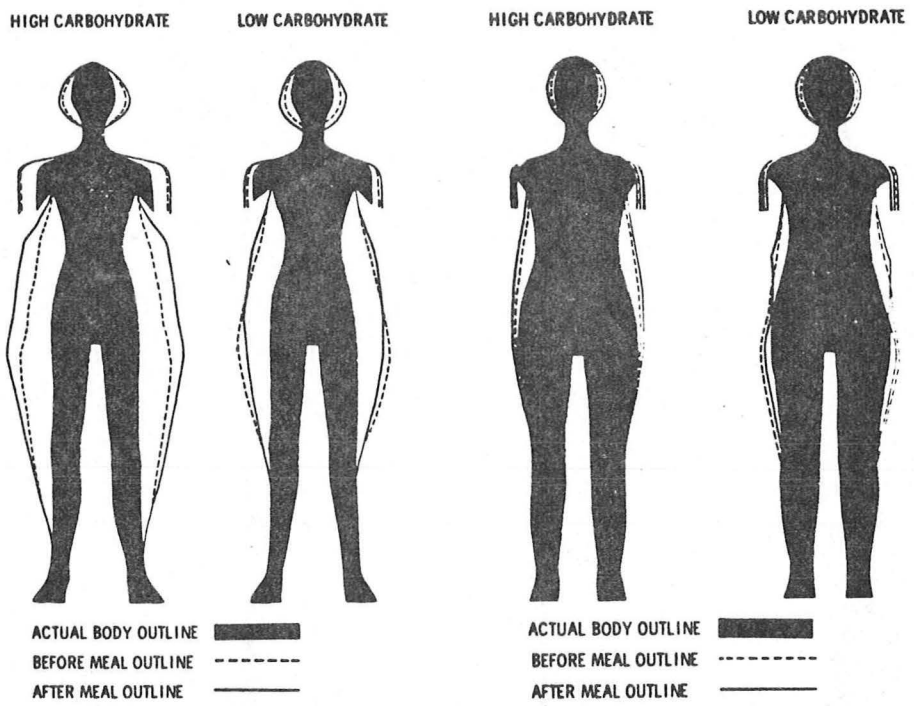

FIG. Estimated versus actual body widths. **Left:** Two sets of outlines of an anorexia nervosa patient, aged 15 years, height 1.72 m, premorbid weight 56 kg, present weight 42.8 kg, target weight 58 kg. **Right:** Two outlines of the same patient recovered from anorexia nervosa. Black areas represent actual measured body dimensions; dotted outlines indicate pre-meal self-estimations of body widths; continuous outlines indicate self-estimations of body widths shortly after high- and low-carbohydrate isocaloric meals. The figures show (a) the characteristic gross overestimation of body width by an anorectic, (b) the specific and symbolic impact of carbohydrate on this process, (c) the restoration toward accuracy of these perceptions following recovery. (From ref. 14 with permission.)

On the left are actual and perceived outlines of the body on low and high carbohydrate diets during active disease while on the right are shown similar representations after weight gain. Recent studies have cast considerable doubt on the specificity of distorted body image in anorexia nervosa, however (18-21). It has been unequivocally shown that similar distortions can be seen in control populations. The tendency to overestimate body size is usual in adolescence and tends to ameliorate or disappear with age or maturation. For this reason Hsu (21) has suggested that disturbance of body image be deleted from the diagnostic criteria for anorexia nervosa, stating: "The evidences, however, are overwhelmingly against overestimation of body width as pathognomonic of anorexia nervosa."

d. Symptoms. As noted above, the syndrome of denials that characterizes anorexia nervosa tends to minimize spontaneous revelation of symptoms, although almost all patients will discuss amenorrhea when asked. Sleep disturbances are fairly common (13,15,22). Constipation is not unusual although diarrhea may occur with laxative use (14). Complaints of early satiety and abdominal pain are frequent. The cause of these gastrointestinal symptoms is not known although abnormally slow gastric emptying has been reported (23). Cold intolerance is often acknowledged and true hypothermia has been reported (11). Traditionally

this has been attributed to "functional hypothyroidism" but abnormality of the hypothalamic temperature regulating centers may be a more likely explanation (24). Patients with anorexia nervosa do not defend well against either heat or cold challenge (Fig 2).

Figure 2 (ref 24)

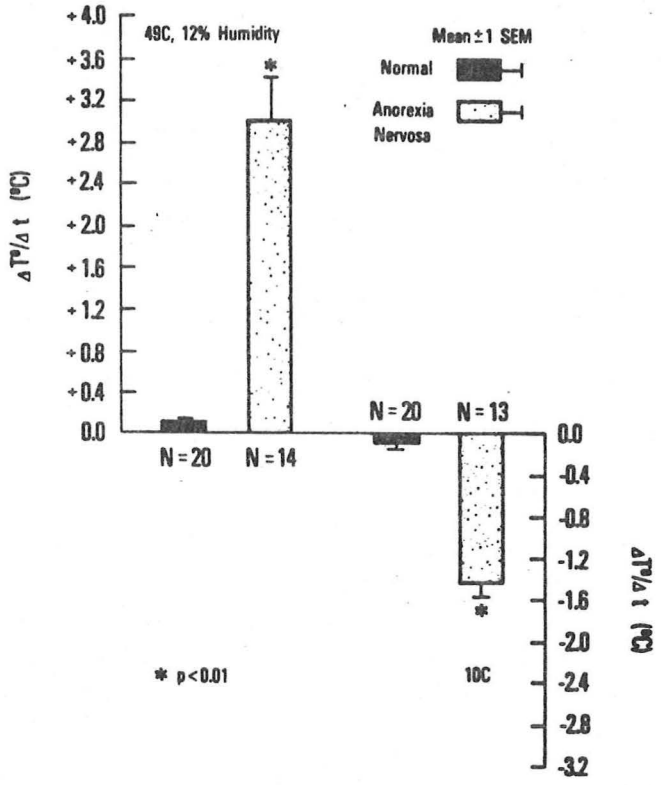

FIG. Response (mean ± SEM) of normal subjects and patients with anorexia nervosa to hyperthermia and hypothermia. The $\Delta T°/\Delta t$ is the rate of change of core temperature (°C) per hour, which is a measure of thermoregulatory ability.

The bars in this figure represent change in core temperature per hour. It can be seen that core temperature rises inappropriately at 49° and falls disproportionately with cold. Patients with anorexia also develop excessive vasoconstriction, cyanosis and numbness of the extremities on exposure to cold which may reflect an abnormal sensitivity of the vessels to low temperatures (25). Raynaud's phenomenon has been noted.

e. Physical findings. The physical examination in classic anorexia is characterized pre-eminently by cachexia so severe as to remind one of the victims of German war camps. In the fully dressed state the degree of weight loss may not be appreciated because the victims tend to wear masking clothes (long sleeves, long skirts, slacks). Parotid enlargement due to malnutrition may soften the angularity of the face expected with this degree of weight loss. As in all forms of semi-starvation, the pulse rate is slow and blood pressure is on the low side. The basal metabolic rate is decreased reflecting a diminished body mass. Peripheral edema is not uncommon. It is usually not due to hypoalbuminemia but to a failure to mobilize the normal ECF volume with starvation. An increase in body hair, usually quite fine, is not unusual. A yellow cast to the skin due to carotenemia is helpful since carotene levels are low in other forms of malnutrition. A summary of the physical findings in 65 patients (26) is shown in Table 4.

Table 4 (ref 26)

TABLE *Abnormalities noted on physical examination of 65 patients with anorexia nervosa*

Abnormality	No. affected	% Affected
A. Skin (hairiness, scaliness, dirtiness, desquamation)	57	88
B. Hypothermia (rectal temperatures <96.6°F)	55	85
C. Bradycardia (<60 beats per min)	52	80
D. Cachexia	47	72
E. Bradypnea (<14 breaths per min)	43	66
F. Hypotension (systolic pressures below 70 mm Hg)	34	52
G. Heart murmurs	25	38
H. Peripheral edema	15	23

11. Halmi KA: Anorexia nervosa: recent investigations. Ann. Rev. Med. 29:137-148, 1978.
12. Drossman DA, Ontjes DA, Heizer WD: Anorexia nervosa. Gastroenterology 77:1115-1131, 1979.
13. Crisp AH, Hsu LKG, Harding B, Hartshorn J: Clinical features of anorexia nervosa. A study of a consecutive series of 102 female patients. J. Psychosom. Res. 24:179-191, 1980.
14. Schwabe AD, Lippe BM, Chang RJ, Pops MA, Yager J: Anorexia nervosa. Ann. Intern. Med. 94:371-381, 1981.
15. Halmi KA: Anorexia nervosa: demographic and clinical features in 94 cases. Psychosom. Med. 36:18-26, 1974.
16. Beumont PJV, Chambers TL, Rouse L, Abraham SF: The diet composition and nutritional knowledge of patients with anorexia nervosa. J. Hum. Nutr. 35:265-273, 1981.
17. Crisp AH, Kalucy RS, Lacey JH, Harding B: The long-term prognosis in anorexia nervosa: some factors predictive of outcome, in Anorexia Nervosa, RA Vigersky (ed), Raven Press, New York, 1977, pp 55-65.
18. Strober M, Goldenberg I, Green J, Saxon J: Body image disturbance in anorexia nervosa during the acute and recuperative phase. Psychol. Med. 9:695-701, 1979.
19. Ben-Tovim DI, Whitehead J, Crisp AH: A controlled study of the perception of body width in anorexia nervosa. J. Psychosom. Res. 23:267-272, 1979.
20. Garner DM: Body image in anorexia nervosa. Can. J. Psychiatry 26:224-227, 1981.
21. Hsu LKG: Is there a disturbance in body image in anorexia nervosa? J. Nerv. Ment. Dis. 170:305-307, 1982.
22. Halmi KA, Goldberg SC, Eckert E, Casper R, Davis JM: Pretreatment evaluation in anorexia nervosa, in Anorexia Nervosa, RA Vigersky (ed), Raven Press, New York, 1977, pp 43-54.
23. Holt S, Ford MJ, Grant S, Heading RC: Abnormal gastric emptying in primary anorexia nervosa. Brit. J. Psychiat. 139:550-552, 1981.
24. Vigersky RA, Loriaux DL: Anorexia nervosa as a model of hypothalamic dysfunction, in Anorexia Nervosa, RA Vigersky (ed), Raven Press, New York, 1977, pp 109-121.
25. Luck P, Wakeling A: Increased cutaneous vasoreactivity to cold in anorexia nervosa. Clin. Sci. 61:559-567, 1981.

26. Silverman JA: Anorexia nervosa: clinical and metabolic observations in a successful treatment plan, in Anorexia Nervosa, RA Vigersky (ed), Raven Press, New York, 1977, pp 331-339.

2. Bulimia. The term bulimia literally means "ox-hunger" or a voracious appetite. It has come to stand for a syndrome of astonishing food intake over short periods of time in young women who usually have a previous or present picture of anorexia nervosa . The gorging is then followed by induced vomiting and often by the use of laxatives in large amounts. If one selects patients for anorexia nervosa by the Feighner criteria, about 40-50% of subjects admit bulimia-vomiting (27,28) but some patients may exhibit binge eating without ever going through an anorexic phase (29). Two fundamental features characterize the syndrome: (1) an irresistible urge to overeat and (2) a marked fear of becoming fat. The former predominates in this form of the illness but there are other features than distinguish it from classic anorexia nervosa. In simple terms patients with non-bulimic anorexia nervosa deal with the fear of being fat by restricting food intake ("restrictors"). Their phobia of being fat appears to be so powerful that control over eating is not lost. Bulimic patients, on the other hand, lose control and thus become "gorgers," controlling weight gain only by vomiting and use of laxatives. The careful study of Russell (29) illustrates the ontogeny of the bulimic syndrome from his experience with 30 patients.

Figure 3 (ref 29)

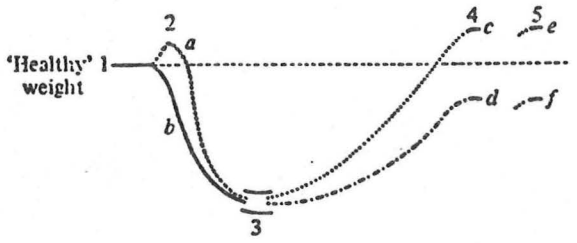

FIG. 3. Schematic representation of variations in the weights of patients with bulimia nervosa in the course of the disorder (see text).

In this scheme 1 represents the healthy weight of the individual; 2 is the change of weight at the start of the disorder; 3 is the point of lowest weight; 4 represents a stable period after weight gain; and 5 marks the point where treatment is sought. Eleven of 30 patients began their illness after a period of weight gain (a) while 19 started with weight loss (b). At point 3 all patients began to gain weight. Ten followed course c reaching normal or above normal weight, but only 4 remained at this level by the time treatment was begun. Twenty patients followed course d. Thus at the time of treatment 24 subjects were underweight (f), 2 were normal weight and 4 were above healthy weight (e). Between points 2 and 3 patients were in the restrictive mode; at point 3 there was failure of control and bulimic behavior began. Characteristics of the bulimic syndrome follow.

a. Demographic features. As with classic anorexia nervosa the majority of patients are women. Major demographic features are

similar in the two groups (Table 5) although the premorbid weight and

Table 5 (ref 28)

Table —Clinical Demographic Features in Two Groups of Patients With Anorexia Nervosa

	Bulimic	Restricting	Level of Statistical Significance, *P*
Sex, F/M	66/2	69/4	NS
Age on consultation, yr*	21.5 ± 0.6	21.2 ± 0.8	NS
Marital status, %			
Single	82.4	86.3	NS
Married	17.6	12.3	NS
Separated	0	1.4	NS
Religion, %			
Jewish	20.0	23.6	NS
Catholic	23.3	18.1	NS
Protestant	36.7	36.1	NS
Other	20.0	22.2	NS
Duration of illness, mo	49.6 ± 4.0	38.8 ± 0.2	NS
Social class†	2.5 ± 0.2	2.6 ± 0.2	NS

*Mean ± SEM.
†Using Hollingshead scale.[35]

weight at time of assessment appear to be higher in bulimic subjects (Table 6).

Table 6 (ref 28)

Table —Weight-Related Clinical Features in Two Groups of Patients With Anorexia Nervosa

	Bulimic	Restricting	Level of Statistical Significance, *P*
Birth weight, kg	3.2 ± 0.2	3.1 ± 0.2	NS
Maximum premorbid weight, kg	62.0 ± 1.3	56.4 ± 1.3	< .01
% maximum of average*	106.0 ± 2.2	96.7 ± 2.0	< .01
Frequency of premorbid Obesity (%)†	20/63 (32)	7/66 (10)	< .05
Minimum weight, kg	38.9 ± 0.8	36.6 ± 0.9	= .05
% minimum of average*	62.8 ± 1.3	60.1 ± 1.4	NS
Weight at initial assessment, kg	44.8 ± 1.3	39.6 ± 0.9	< .001
% of average (at consultation)	76.5 ± 2.1	67.9 ± 1.4	< .001
Height, cm	163.3 ± 0.8	162.8 ± 0.9	NS
Menarche, yr	12.9 ± 0.2	12.8 ± 0.2	NS

*Average weights for age and height.[36]
†Defined as greater than 115% of average for age and height.
‡Recorded on 4-point scale from 1, absent to 4, marked.

Maximum weight loss is also less. Mothers of bulimic patients have a higher prevalence of obesity than mothers of restrictors but the percentage is still not excessive relative to the general population.

b. Behavioral characteristics. The drive to eat in bulimic patients is overwhelming. Thoughts are constantly on food and even dreams may focus on eating. The drive is not from hunger. One of Russell's patients describes it as follows: "It is not hunger. Hunger is a feeling of a gap inside you. You eat something small to stop that feeling. I go on eating after I've satisfied that hunger. I want to keep on eating until I feel full - it's the final limit - you can then eat no more" (29). The amount of food ingested can be enormous, up to 50,000 calories a day. In the series of 40 patients reported by Mitchell, Pyle and Eckert (30) the mean duration of binge-eating episodes was 1.2 hours but could last as long as 8 hours. On average gorging occurred 12 times a week, but the range was from as little as 1 to as many as 46. The mean number of calories taken in per episode was 3415 but could reach 11,500 at one sitting. In these 40 patients the major foods ingested, in order of frequency, were ice cream → bread-toast → candy → doughnuts → soft drinks → other. Usually more than one food is used in an episode. Overeating is ordinarily carried out secretly and alone, generally in the afternoon and evening (29,30). Often the episodes appear to be precipitated by ingestion of a "forbidden" high carbohydrate food, setting up an unstoppable chain-reaction. If the urge to eat the first morsel can be controlled, binges do not occur. (Some have likened it to the "first drink" phenomenon in alcoholics.) This may account for an "all or none" pattern to the eating. Palmer has coined the term "dietary chaos" to describe the eating behavior in bulimic subjects (31). It is an accurate description.

Following the gorge, essentially all patients with bulimia induce vomiting (27 of 30 and 37 of 40 in references 29 and 30, respectively). This most often is done by inducing the gag reflex with the fingers or a toothbrush, although some subjects learn to regurgitate spontaneously. Russell noted calluses on the fingers of two of his patients (29). The use of emetics is very rare. The vomiting may become ritualistic with patients requiring a fixed number of retchings to be satisfied that all food has been removed.

A high percentage of patients also use laxatives, although cathartic abuse is not as common as vomiting. It is probable that other forms of weight control, such as excessive exercise and use of diuretics, occur to a similar extent in the anorexic and bulimic syndromes.

A striking feature of bulimia, noted in all series, is the propensity to carry out anti-social behavior (27-29,32). Twelve to fourteen percent of patients with bulimia admit stealing (most often food) and the actual percentage is doubtless higher. Stealing is not reported in classic anorexia nervosa with major weight loss (32). As shown in Table 7, patients in the bulimic phase use both street drugs

Table 7 (ref 28)

Table —Impulse-Related Clinical Features in Two Groups of Patients With Anorexia Nervosa

	Bulimic	Restricting	Level of Statistical Significance, *P*
Use of alcohol, % weekly of more	20.4	4.8	< .05
Street drug use, % at any time	28.6	11.6	< .05
Stealing, %	12.1	0	< .05
Self-mutilation, %	9.2	1.5	< .05
Suicide attempts, %	23.1	7.1	< .05
Lability of mood*	1.7 ± 0.1	1.2 ± 0.1	< .001

*Recorded on 4-point scale ranging from 1, absent to 4, marked.

and alcohol to a greater extent than anorexic subjects. Self-mutilation and suicide attempts are 3-4 times more common in bulimia than anorexia. Although most patients with eating disorders are disinterested in sex, 4 of Russell's 30 cases were sexually promiscuous (29).

c. Perceptual abnormalities. I was not able to find a formal series testing body image perception in the bulimic subset, although Casper et al (27) mention than overestimation of body size was greater than in a control group. It is likely that there is no major difference from classic anorexia.

d. Symptoms. In contrast to classic anorexia nervosa amenorrhea was present in only 11 of 28 subjects when cessation of menses was not used as part of the selection criteria (29). This is likely due to the fact that weight loss was less severe in the bulimic group. The other major complaint, often spontaneously voiced, is of depression. In view of the recurrent vomiting with hypokalemia one would expect complaints of weakness to be frequent, but this was not obvious in the cited series. Convulsions and tetany have been reported but are rare (29). The cause of the former is not known while hypokalemia presumably accounted for the latter. I could not find specific comments on constipation, abdominal pain or cold intolerance.

e. Physical examination. Bulimic patients are usually not emaciated and as a consequence usually do not exhibit bradycardia, relative hypotension, parotid enlargement or hypothermia. They may have scars from self-mutilation or suicide attempts.

27. Casper RC, Eckert ED, Halmi KA, Goldberg SC, Davis JM: Bulimia. Its incidence and clinical importance in patients with anorexia nervosa. Arch. Gen. Psychiatry 37:1030-1035, 1980.
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IV. Laboratory abnormalities

Although many systems of the body are affected in severe anorexia nervosa, most of the laboratory changes are of little consequence and not unique since they occur in other forms of semi-starvation. Hematologic findings include anemia, leukopenia (relative neutropenia, lymphocytosis), thrombocytopenia, low erythrocyte sedimentation rate and decreased fibrinogen levels in plasma (11,12,14,33). The anemia and occasional pancytopenia appear to be due to hypoplasia of the bone marrow which is filled with a gelatinous mucopolysaccharide. Peripheral blood smears may show acanthosis.

Plasma proteins tend to be normal although occasionally hypoalbuminemia may be seen (12,14,34). Essential amino acids are not low, in contrast to kwashiorkor, probably because of the relatively high protein intake of anorexic subjects (35).

As noted earlier, β-carotene levels in plasma are high, together with vitamin A and its derivatives (14). The mechanism of this elevation is not clear. However, the fact that anorexic subjects who vomit have serum carotene levels only one-half those of non-vomiters suggests that dietary intake plays a major role (36).

An interesting finding, known since 1965, is the frequent occurrence of hypercholesterolemia in anorexia nervosa. The cholesterol elevation is in the LDL fraction and both HDL and VLDL levels are normal (37). Values in 18 patients and 15 controls are shown in Table 8.

Table 8* (ref 37)

Table Clinical and Laboratory Data of Patients With Anorexia Nervosa and Controls

Subjects	Age (yr)	Height (cm)	Weight (kg)	Fasting Blood Sugar (mg/100 ml)	Total Protein (g/100 ml)	T_4 (μg/ml)	T_3 (μg/ml)	Serum Amylase (IU)	Plasma Cholesterol (mg/100 ml)	Plasma Triglycerides (mg/100 ml)	Plasma Phospholipids (mg/100 ml)	LDL (mg/100 ml)		HDL (mg/100 ml)		VLDL (mg/100 ml)	
												Chol	TG	Chol	TG	Chol	TG
Patients (n = 18)	22	162.8	34.6	80 ± 6	6.6 ± 0.7	81 ± 18	1.15 ± 0.30	348 ± 29	265 ± 34	119 ± 41	274 ± 36	185 ± 36	24 ± 8	40 ± 8	14 ± 7	39 ± 9	76 ± 18
Controls (n = 15)	23.6	164.5	53.2	82 ± 8	7.3 ± 0.6	88 ± 20	1.44 ± 0.24	176 ± 34	194 ± 19	100 ± 24	194 ± 28	115 ± 14	19 ± 7	42 ± 8	16 ± 5	25 ± 6	65 ± 15

*Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein; VLDL, very low density lipoprotein; Chol, cholesterol; TG, triglycerides.

* Note that there is a unit error on the thyroid hormone values in this table which should read ng rather than μg.

Plasma triglyceride concentrations are normal despite low values for hepatic and lipoprotein lipase activities. The cause of the hypercholesterolemia is not known, although neutral sterol and bile acid secretion appear to be low (38). One possibility is that hepatic LDL receptors decrease with severe weight loss, but this has not been studied.

In view of the known relationship between malnutrition and depressed immune function (variable effect on humoral immune function, profound effect on cellular immunity) there has been considerable interest in the immune response in anorexia nervosa. In a series of 5

patients recently published (39), mean levels of IgG, IgM and transferrin were low prior to hyperalimentation (Table 9), although

Table 9 (ref 39)

Individual and mean serum concentrations for immunoglobulins, transferrin and albumin before and after alimentation in five patients with anorexia nervosa

Patient	IgG		IgM		IgA		Transferrin		Albumin	
	Before	After	Before	After	Before	After	Before	After	Before	After
	mg/dl		mg/dl		mg/dl		mg/dl		g/dl	
1	646	696	58	218	103	52	161	224	3.3	4.2
2	572	596	29	35	26	67	217	231	4.1	3.8
3	795	1120	87	169	160	173	182	341	4.6	6.1
4	547	546	87	129	160	232	189	225	4.0	4.3
5	820	1065	116	166	72	108	217	252	4.0	4.6
Patients (Mean ± SEM)	676 ± 25	805 ± 11	75 ± 6.6	143 ± 13.6	104 ± 12	126 ± 15	193 ± 5	255 ± 10*	4.0 ± 0.1	4.6 ± 0.2
Control (Mean ± SEM)	1035 ± 29**		113 ± 6*		151 ± 14		285 ± 7**		4.3 ± 0.1	
% increase with treatment	19		91		21		32		15	

Serum concentration below the normal range is underscored.

Significance of difference between means of pretreatment and postreatment groups and pretreatment and control groups analyzed by Student's *t* test: $*p < 0.05$, $**p < 0.001$.

not all patients showed the change. A number of alternate complement pathway control proteins were also low: C1q, C2, C3, factor B, β1H, C3B inactivator, properdin and C4 binding protein. The mechanism was thought to be decreased synthesis. When 22 consecutively admitted patients were studied by an anergy panel to test delayed hypersensitivity, only 6 showed defective responses (40). This is in accord with the view that most patients with anorexia nervosa are surprisingly free of infection (40-42). Occasionally infection does occur as indicated by a death from herpes simplex encephalitis (41).

A number of other abnormalities have been reported, none of which is of major significance clinically. Glomerular filtration rate is generally slightly low and prerenal azotemia with BUN levels as high as 60-70 $mg.dl^{-1}$ may be seen (26). Renal concentrating ability is impaired. The latter does not respond to ADH (12). Non-specific ST-T changes may be seen on ECG.

Serum amylase levels may be elevated in the absence of clinical signs of pancreatitis (11,37). The reason for this is not obvious.

In 30 hospitalized patients with anorexia nervosa plasma zinc and copper were low, although hair content of these metals was normal (43). Iron binding capacity was decreased but plasma iron and ceruloplasmin were normal. Hypogeusia (taste impairment) was noted, most marked for bitter and sour stimuli.

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V. Endocrine findings

Considerable interest has focused on the endocrine system in anorexia nervosa. There are probably two reasons for this. First, there was the earlier period of confusion between pituitary insufficiency and anorexia nervosa that needed to be clarified. Second, there is the fact of essentially constant amenorrhea in the classic form of the disease. It now seems clear that the endocrine changes are all secondary; i.e., there is no evidence for primary pituitary, gonadal, thyroid or adrenal dysfunction.

1. Amenorrhea. Amenorrhea in anorexia has been extensively investigated and reviewed (e.g., 11,12,14). About half of patients develop secondary amenorrhea concomitant with the onset of dieting while 16-20% cease menses prior to onset of overt disease. The remaining patients undergo secondary failure of menses only after weight loss is significant (14,44). Presumably early amenorrhea is due to psychologic stress anteceding clinical illness (45). It is now generally accepted that the primary defect is localized in the hypothalamus and operates via impaired release of gonadotropin-releasing hormone (LHRH). Baseline luteinizing hormone (LH) and follicle stimulating hormone (FSH) values are low and the 24 hour LH profile regresses either to a prepubertal pattern (all values low) or a pubertal pattern (sleep-dependent LH release only) as originally described by Boyar and colleages (46). The prepubertal pattern is most common, being found in 14 of 16 patients by Pirke et al (47). With weight gain reversal of the LH abnormality occurs, the pubertal pattern appearing at about 70% ideal body weight (IBW) and the adult pattern near 80% IBW. The pituitary response to gonadotropin-releasing hormone is abnormal with severe weight loss but reverses to normal with weight gain (48). A representative experiment illustrating this point is shown in Fig 4.

Figure 4 (ref 48)

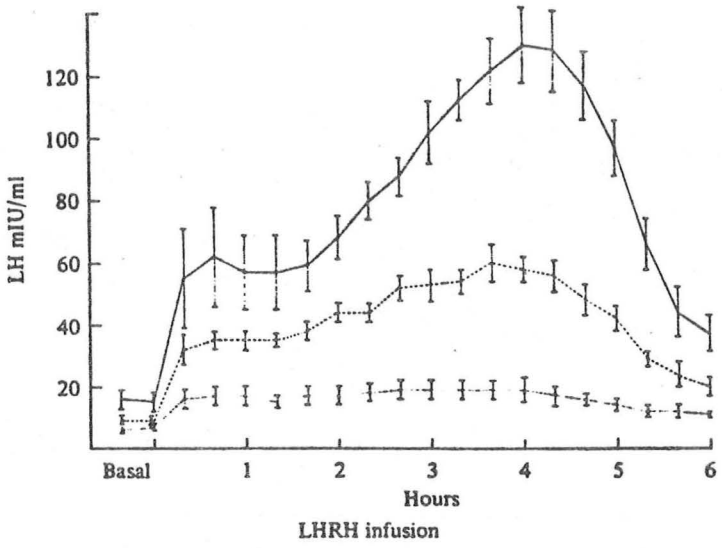

FIG. The LH response to continuous infusion of LHRH: female patients during various stages of refeeding. ——, SBW > 90 % (N = 4); – – –, SBW = 80 % (N = 9); · · · ·, SBW < 65 % (N = 7).

The bottom line of the figure, representing a mean weight loss to less than 65% of standard (SBW) shows only minimal response to a 4 hour infusion of LHRH. The middle line, 80% of SBW, indicates an intermediate response while the top line, equivalent to 90% SBW, shows the expected biphasic release of LH characteristic of mature adults. Pituitary responsiveness to gonadotropin-releasing hormone can also be restored if low doses of LHRH are given every 2 hours for 5 days as shown in Fig 5 (49).

Figure 5 (ref 49)

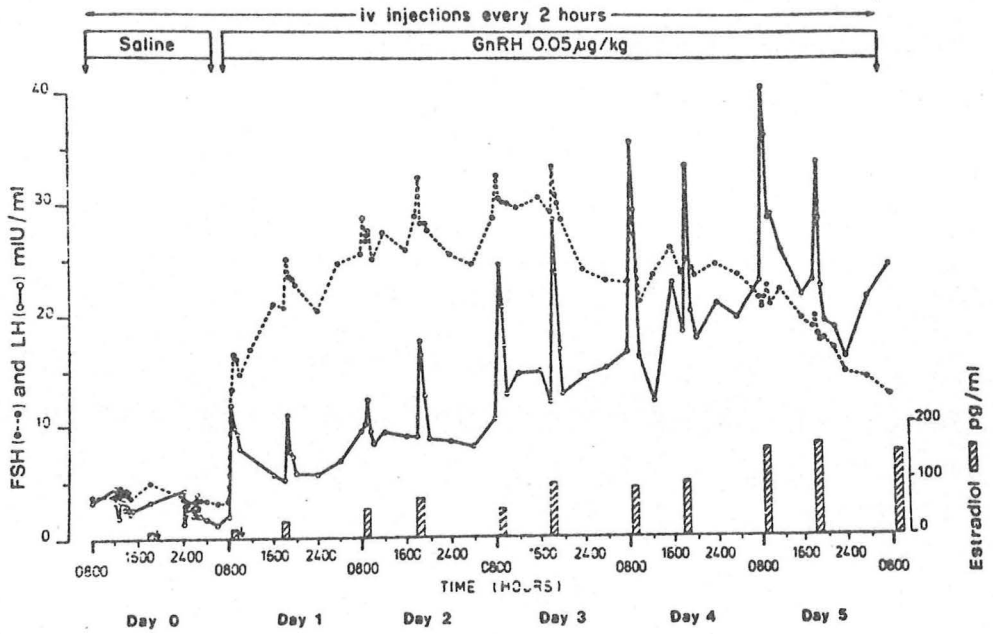

FIG. Plasma FSH (●), LH (○), and estradiol responses to iv GnRH (0.05 μg/kg) every 2 h in patient 1. The *arrow* indicates values below the sensitivity of the estradiol assay.

The greater early response of FSH and subsequent higher response of LH mirrors events taking place during normal puberty. Presumably the lack of pituitary responsiveness to acute stimulation of LHRH represents removal of a trophic effect of that hormone as its production or release in the hypothalamus is impaired for a given period of time (exact length undetermined). Why the hypothalamus is unable to release LHRH in anorexia nervosa is not known, although abnormalities in norepinephrine and dopamine metabolism in the central nervous system have been postulated (14). Bromocriptine, a dopaminergic agonist, has no effect on the abnormalities, however (48). The hypothalamic-pituitary axis is likewise unresponsive to clomiphene (50).

The low estrogen levels and failure to ovulate in anorexia appear to be solely due to gonadotropin deficiency since ovulation can be induced either by exogenous gonadotropins or administration of LHRH for prolonged periods (51,52). Although menses usually return with weight gain, this is not invariably so as psychologic factors can continue to override the reversal of cachexia. Frisch (53) has claimed that following secondary amenorrhea a body weight about 10% greater than that needed for menarche will be required. A chart for predicting the weight necessary to achieve menses in primary and secondary amenorrhea is shown in Table 10.

Table 10 (ref 53)

TABLE *Minimal weight for particular height necessary for the onset or restoration of menstrual cycles*

		Menarche or primary amenorrhea			Secondary amenorrhea		
		Minimal[a] weight (10th percentile)		Average weight (50th percentile)	Minimal[b] weight (10th percentile)		Average weight (50th percentile)
Height (inches)	Height (cm)	(lb)	(kg)	(kg)	(lb)	(kg)	(kg)
53.1	135	66.7	30.3	34.9	74.6	33.9	38.9
53.9	137	68.6	31.2	36.0	76.8	34.9	40.1
54.7	139	70.6	32.1	37.0	79.0	35.9	41.2
55.5	141	72.6	33.0	38.0	81.2	36.9	42.4
56.3	143	74.4	33.8	39.0	83.4	37.9	43.5
57.1	145	76.3	34.7	40.1	85.6	38.9	44.7
57.9	147	78.3	35.6	41.1	87.8	39.9	45.8
58.7	149	80.3	36.5	42.1	90.0	40.9	47.0
59.4	151	82.3	37.4	43.1	92.2	41.9	48.1
60.2	153	84.3	38.3	44.2	94.4	42.9	49.3
61.0	155	86.2	39.2	45.2	96.6	43.9	50.4
61.8	157	88.2	40.1	46.2	98.8	44.9	51.5
62.6	159	90.2	41.0	47.2	101.0	45.9	52.7
63.4	161	92.2	41.9	48.3	103.2	46.9	53.8
64.2	163	93.9	42.7	49.3	105.4	47.9	55.0
65.0	165	95.9	43.6	50.3	107.6	48.9	56.1
65.7	167	97.9	44.5	51.4	109.8	49.9	57.3
66.5	169	99.9	45.4	52.4	112.0	50.9	58.4
67.3	171	101.9	46.3	53.4	114.0	51.8	59.6
68.1	173	103.8	47.2	54.4	116.2	52.8	60.7
68.9	175	105.8	48.1	55.5	118.4	53.8	61.8
69.7	177	107.8	49.0	56.5	120.6	54.8	63.0
70.5	179	109.6	49.8	57.5	122.8	55.8	64.1
71.3	181	111.8	50.8	58.5	125.2	56.9	65.3

From ref. 15, Figs. 1 and 2.
[a] Equivalent to 17% fat/body weight.
[b] Equivalent to 22% fat/body weight.

Males with anorexia nervosa appear to have the same abnormalities in gonadotropins seen in females and in consequence testosterone levels are low (54).

2. Other pituitary hormones. Basal growth hormone values have been found elevated in some studies on anorexia nervosa (Table 11).

Table 11 (ref 24)

TABLE *Pituitary–end organ function in anorexia nervosa*

	Normal	Anorexia nervosa
Growth hormone (ng/ml)	10.4 ± 12.2[a]	48.1 ± 80.7[b]
Prolactin (ng/ml)	16.2 ± 5.5	19.4 ± 14.4
TSH (μU/ml)	N.D. in 77% <7.6 in 23%	N.D. in 30% <6.0 in 70%
LH (mIU/ml)	7.9 ± 4.4	3.9 ± 2.5[b]
FSH (mIU/ml)	15.3 ± 2.7	5.6 ± 5.2[c]
Cortisol (μg/ml)	14.4 ± 3.9	14.8 ± 5.4
Free T_4 (ng/100 ml)	1.5 ± 0.2	1.5 ± 0.2
T_3 (ng/100 ml)	156 ± 30	76 ± 30[b]
Estradiol (pg/ml)	–	<10

[a] Values are mean ± SD.
[b] $p < 0.001$.
[c] $p < 0.01$.

Thus Vigersky and Loriaux (24) found basal values 4 times higher in 29 patients with anorexia nervosa than in a control population. (The values for growth hormone reported in this paper for controls are high, but the results are probably valid in relative terms.) The careful review of Halmi (11) emphasizes that overall only about one-third of patients have elevated growth hormone levels although response to provocative stimuli may be impaired. Plasma somatomedin activity (bioassay) was low in 8 of 12 patients with anorexia nervosa and unresponsive to growth hormone administration (55). This probably is a consequence of both decreased synthesis and inhibitors of somatomedins in plasma, features characteristic of malnutrition and weight loss of any cause. Plasma prolactin levels are usually normal in anorexia (24,56,57) although they may rise paradoxically after LHRH administration (58). I was not able to find measurements of vasopressin in anorexic patients.

3. Thyroid function. Despite the slow pulse and low basal metabolic rate that characterize anorexia nervosa and other forms of weight loss, there is no evidence of hypothyroidism (59,60). The usual picture is low normal T4, low T3 and increased reverse T3 (Table 12).

Table 12 (59)

TABLE *Serum thyroid hormone parameters, prolactin, and growth hormone in patients with anorexia nervosa*

Patient	% Below ideal weight	T4 (μg/dl)	Free T4 (ng/dl)	T3 (ng/dl)	Reverse T3 (ng/dl)	3,3'T2 (ng/dl)	Prolactin (ng/ml)	Growth hormone (ng/ml)
1	45	4.1	1.3	54	68	–	15	180
2	38	4.0	1.3	27	49	–	<10	70
3	52	5.9	–	51	54	–	<10	360
4	49	6.6	1.7	60	76	44	<10	87
5	52	7	–	68	37	–	–	104
6	40	6.6	1.4	72	69	16	–	–
7	40	4.5	1.4	47	77	25	8.9	12
8	21	6.3	1.4	85	67	–	13	3
9	31	5.8	1.3	68	27	–	13.8	7
10	37	4.7	–	84	–	51	–	11
11	30	6.8	1.2	107	–	42	22.8	8.8
12	40	4.4	0.7	106	–	20	17.4	12.5
13	22	6.1	1.4	87	–	–	16.9	13.4
14	32	6.0	1.4	84	–	–	15.9	18.4
15	35	5.8	1.4	59	53	–	16.5	7
16	16	4.2	0.9	87	–	–	31.4	19
Mean	36	5.6	1.3	72	58	33	15.5	60.9
SE	3	0.3	0.07	5	5	6	1.7	25
N	16	16	13	16	10	6	13	15
Normal values								
Mean	–	8.5	1.5	156	49	17	16.2	10.4
SE	–	0.1	0.03	4	3	1	1.2	3.9
N	–	1000	50	47	29	18	21	10

There are two forms of the "euthyroid sick" syndrome, one in which the T3 is low and both T4 and rT3 are elevated (61), the other in which both T4 and T3 are low (62). The former mimics hyperthyroidism, the latter hypothyroidism. Anorexic patients usually fall in the low T4, low T3 category. The low T4 is thought to be due to an inhibitor that blocks binding of T4 to thyroid binding globulin (62). Moore and Mills (63) showed reversal of the thyroidal abnormalities with weight gain in 33 patients suffering from anorexia nervosa. Interestingly, 4 had an overshoot of T3 levels accompanied by symptoms of mild hyperthyroidism in the recovery phase.

4. Adrenal function. Mean plasma cortisol levels measured over 24 hours are in the upper normal range or frankly elevated. Boyar et al (64) suggested that this was primarily due to decreased metabolism of cortisol since its half-life was prolonged and excretion rates were low. More recently it has been claimed that cortisol production rates may also be slightly increased, based on a fall in production seen with recovery (65). Dexamethasone suppression tests are abnormal in anorexia (66,67). It is uncertain whether this is due to concomitant depression or intrinsic to the primary condition, although Gerner and Gwirtsman (67) feel it is independent of depression. A number of enzymic abnormalities have been found in the adrenal (e.g., 5α-reductase deficiency), but these are of more interest from a research than a clinical standpoint and will not be outlined here (11,14,66).

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VI. Psychological accompaniments

Although the diagnosis of anorexia nervosa is designed to exclude primary psychiatric illnesses such as schizophrenia or severe depression, it is clear that a significant percentage of patients have psychoneurotic symptomatology (68), depression (69) and transient psychoses (70). Considerable emphasis has been placed on impaired psychosexual maturation in girls with anorexia, but this was not confirmed by Beumont et al (71) who found this aspect of emotional development to be normal prior to illness in Australian patients at least.

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VII. Etiology

The cause of anorexia nervosa is not known. While it has been argued by Vande Wiele (72) that hypothalamic dysfunction is primary, the evidence appears to me persuasive that the disorder is a psychiatric one. The psychodynamics are not clear and, in fact, may not be fixed. The construct presented by Yager (in ref 14) represents a widely held view:

> "If one were to subscribe to a current view of the psychologic development of 'typical' anorexia nervosa, conditions might be as follows: the patient comes from an upper middle class, highly achievement-oriented family that values slimness and physical exercise. Mother and perhaps others in the family are constantly vigilant about weight. The family presents a facade of psychologic health but certain conflicts lurk below the surface between the parents. Because of lack of fulfillment as a couple, the parents find themselves striving for personal fulfillment in other areas: the mother in her children and the father in his occupation. The family communicates along few, narrow lines (among which is food). There is a channeling of concern toward the preanorectic child so that the mother might become excessively involved with her. The mother might be overdirective, yet fail to acknowledge the child's individuality, and at the same time fearful of the child's impending or beginning adolescent psychosexual development and separation from home and family. The overdirected preanorectic child pursues the high achievement orientation so valued by the family and becomes more concerned with external approval than with internal satisfaction. The preanorectic child feels that there are very few areas for real self-determination and self-control and develops a fragile self-image. Parental approval might be used to fill the child's inner void in place of feelings of personal effectiveness and autonomy, resulting in a shaky sense of self. At a point of family disequilibrium during the preanorectic child's adolescence - perhaps due to increasing parental friction, the illness or death of a relative, moving away to school, an initial heterosexual encounter, or the mother's increased fearful awareness that her daughter's move toward independence and separation might provoke an 'empty nest' - the anorexia nervosa syndrome develops. It often begins during the course of an ordinary diet in which the patient 'just wants to lose a few pounds,' often with the agreement and blessing of her family and friends."

Whatever other factors operate in the genesis of the disease it must be emphasized that the "enmeshed" family is critical: there are blurred generational boundaries so that parents and children are constantly involved in each other's problems.

Bruch has for years suggested that both major eating disorders, anorexia nervosa and obesity, have as a fundamental characteristic a "paralyzing sense of ineffectiveness" induced by early events in family life (73). They experience themselves as "acting only in response to demands coming from others and as not doing anything because they want to." She goes on to say: "The development of anorexia may be conceived as a shouting and unrelenting 'No' which extends to every area of living, though most conspicuous in the food refusal. Uncontrolled obesity, on the other hand, is the manifest expression of despair, of having given up all efforts to establish a sense of inner control and

independent identity" (73). (Note: It is highly unlikely that obesity is a purely psychiatric disorder, in contrast to anorexia nervosa, since underlying metabolic defects, probably genetic in origin, are necessary for its expression.)

Although family structure appears to play a primary role in the genesis of anorexia nervosa, it is becoming increasingly clear that culture is also important. This point has been emphasized by Garfinkel (74). He notes that in contemporary western society the ideal female figure is that of a slender prepubertal girl bearing the secondary sexual characteristics of a mature woman. He cites the intriguing observation that at Madame Toussaud's Wax Museum there is an annual poll for the most attractive woman in the world. In 1970 the winner was Elizabeth Taylor while in 1976 the number one spot was occupied by Twiggy. Preoccupation with diets and weight loss is extremely common in normal teenage girls of the western world: up to 70% in the 12th grade (75). The prevalence of anorexia nervosa in dancers is ten times that of the general population, suggesting that even occupation may play a role. More recently, anorexia-like syndromes have been seen with increasing frequency in athletes who want to reduce their fat to 5-7% of body weight (76). It thus seems entirely possible that the increasing frequency of anorexia nervosa is due to relentless cultural pressures to diet, stay slim and exercise, this pressure selecting out those predisposed to develop the illness.

Patients with anorexia nervosa tend to have abnormal aversion tests to sucrose (77) and bulimic subjects respond abnormally to high carbohydrate preloading in eating tests (78), but it seems likely that both of these changes are secondary to the primary psychiatric disorder. In short, there is no evidence that anorexia nervosa is an organic disease; rather, the psychiatric disorder leads to the physiologic abnormalities.

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VIII. Treatment and outcome

There is no specific treatment for anorexia although multiple approaches have been tried, many of them controversial (79). A partial list includes: insulin, thyroid hormone, gonadotropins, antidepressants, antipsychotics, tranquilizers, electroconvulsive

therapy, appetite stimulants and leucotomy. From a psychologic and psychiatric standpoint, behavior modification, individual psychotherapy and relationship therapy have been tried, singly and in combination.

Most experts in the field agree that there is no one way to approach what is an incredibly difficult problem. However, certain general principles can be developed (79,80).

1. It is generally preferable to hospitalize the patient for initial treatment; the usual period is 6-8 weeks.
2. The immediate aim is to induce weight gain; psychotherapy is of little benefit until after nutritional status has improved.
3. The patient should never eat alone; either the patient must eat in the company of a nurse or combined patients and staff should eat together.
4. It is preferable to have a single staff person ("special") carry out primary interactions with the patient.
5. While the support staff should be friendly and reassuring about the "safety" of eating, lengthy intellectualizing about food and weight should be avoided.
6. Parenteral or enteral nutrition should be prescribed only as a life-saving measure; hyperalimentation is never indicated as a primary treatment (81).
7. Caloric intake should be increased gradually over 7 to 10 days from 1000 calories to a level twice that of a normal adult (3000 to 5000 calories depending on size).
8. Psychoactive drugs should be used if needed for depression or anxiety.
9. As nutritional status improves social therapy should be initiated (arts, crafts, games, etc.).
10. Parental involvement should be started early either as family therapy or with a parental group (79,80,82).
11. Following discharge the patient should be seen at least biweekly for support and guidance.

It is obvious that specialized treatment centers for anorexia nervosa are not available for the majority of patients and that psychiatrists specializing in eating disorders are not many. If the above principles are followed, however, non-psychiatric physicians may do reasonably well in treatment (83).

The long-term outlook in anorexia is difficult to ascertain, and impossible to relate to treatment programs. Crisp (84) reported his experience with 100 patients followed for 4-7 years after initial assessment. In this group 64 attained normal weight (54 ± 6 kg), 18 were improved but below normal (48 ± 8 kg), fourteen were still below pubertal weight (38 ± 12 kg) and 2 were obese (70 and 64 kg). Two patients died during the period of observation.

Two papers have recently appeared attempting to evaluate the results of the major follow-up studies in the English literature (85,86). The more complete is that of Hsu (85). However, since all studies did not report all findings of interest, only general impressions can be gained. The data on weight loss and menses are shown in Table 13.

Table 13 (ref 85)

Table —Nutritional and Menstrual Outcome

Source, yr	Nutritional Outcome	Menstrual Outcome
Hsu et al, 1979[7]	65 (62%) normal weight (± 15% MPMW) 13 (12%) normal weight but fluctuated 6 (6%) intermediate (75%-85% MPMW) 16 (15%) below 75% MPMW (including 2 deaths) 2 (2%) overweight (> 115% MPMW)	54 (51%) regular menses 17 (16%) sporadic 29 (28%) amenorrhea
Garfinkel et al, 1977[21]	58% average (± 20% average) 25% below 75% of average 7% overweight	17 (43%) regular menses 2 (5%) irregular menses
Pertschuk, 1977[15]	12 (41%) within ± 15% standard 9 (31%) 75%-85% standard 1 (3%) overweight 5 (17%) below 75% standard 2 (7%) untraced	9 (33%) menstruating
Morgan and Russell, 1975[13]	22 (55%) normal weight (within ± 15% average) 6 (15%) normal weight but fluctuated 3 (7%) intermediate (75%-85% average) 8 (20%) < 75% average 2 (5%) overweight	18 (47%) regular periods 15 (39%) amenorrhea 3 (8%) sporadic
Theander, 1970[22]	48 (51%) normal weight (± 15% of average) 27 (29%) outside ± 15% average 2 (2%) > 10% overweight	62 (66%) regular menses 12 (13%) amenorrhea
Dally, 1969[23]	Normal weight not defined	80 (59%) regular menses
Warren, 1968[24]	10 (50%) normal weight for age (3rd-90th percentile) 1 (5%) weight still variable 5 (25%) underweight	10 (50%) regular menses 1 (5%) irregular menses 3 (15%) amenorrhea (+ 2 premenarchal)
Thoma, 1967[16]	Normal weight not defined	Information available in 22: 12 (55%) menstruating 6 (27%) amenorrheic
Farquharson et al, 1966[25]	Normal weight not defined	8 (66%) had regular menses or had children
Crisp, 1965[26]	17 (81%) normal weight (± 12.5% of average) 2 (10%) low weight	12 (63%) (including 1 pregnant) normal menses 2 (10%) irregular 1 (5%) sporadic
Kay et al, 1954,[27] 1965[28]	Normal weight not defined	40% normal 60% absent or irregular menses
Beck and Brochner-Mortenson, 1954[1]	Normal weight not defined	17 (61%) regular menses 4 (14%) irregular 4 (14%) amenorrhea

Overall it appears that when patients are followed for 2 years or longer about 50% achieve normal weight, 20-25% are improved but underweight, 20% are unchanged from the anorexic state, 5% are obese and 6% are dead. Perhaps 50-75% of women start menses again, although irregularity is common. Despite the fact that the majority of patients gained weight, eating disorders (restriction, bulimia, vomiting, laxative use) continued to be common (up to 70%). As many as 50% of patients had recognizable psychiatric difficulties not directly related to the eating disorders. Thus, although many patients with anorexia nervosa get better and are able to function in society, the prognosis for normal physical and mental health is poor. There is considerable evidence that non-bulimic patients fair better than bulimic subjects (17,29).

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