Classic diseases revisited

Anorexia nervosa – diagnosis, aetiology, and treatment

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Summary

The aetiology, assessment and treatment of anorexia nervosa are reviewed in the light of the classical accounts of Morton, Lasègue and Gull. The core symptoms are deliberate weight loss, disturbed body image and amenorrhoea, and complications may include cardiac failure, electrolyte disturbances, hypothermia and osteoporosis. Common clinical findings are described. Disturbed brain serotonin activity is implicated in the aetiology of anorexia nervosa, but there is little support for the use of pharmacological treatments. Psychological theories of aetiology are discussed with reference to Bruch, Crisp, Palazzoli and Minuchin: the common theme is the reaction of the patient and her family to the physical and social changes of puberty. Individual and/or family psychotherapy is seen as central to the treatment of anorexia nervosa, and the relevant clinical research is reviewed. The roles of general practitioners, general psychiatrists and eating disorder specialists are discussed in the light of recent consensus treatment guidelines.

Keywords: anorexia nervosa, diagnosis, aetiology, treatment

Diagnosis

“Mr Duke’s daughter in S. Mary Axe, in the year 1684 and the eighteenth year of her age, in the month of July fell into a total suppression of her monthly courses from a multitude of cares and passions of her mind but without any symptom of the green-sickness following upon it . . . I do not remember that I did ever in all my practice see one, that was conversant with the living so much wasted with the greatest degree of a consumption, (like a skeleton clad only in skin) yet there was no fever . . . only her appetite was diminished . . . she was after three months taken with a fainting-fitt, and died.”

Morton’s vivid description of ‘nervous consumption’, published in 1694, is immediately suggestive of what we now refer to as anorexia nervosa.¹ During the 18th and 19th centuries there were frequent references in the medical literature to the condition of *chlorosis*,² which was confined to young women. This disease, also known as ‘greene-sickness’ or the ‘*virgin’s disease*’ was characterised not only by a curious greenish discolouration of the skin, but also by amenorrhoea, pallor, weakness, loathing of food, accompanied at times by emaciation and ‘bulimia emetica’.

The first cases which clearly conformed to the modern concept of anorexia nervosa were described more or less simultaneously in the 1870s by Lasègue and Gull.¹ These were girls who had deliberately decided not to eat, and stubbornly persisted in this pursuit in the face of increasing ill-health and dramatic emaciation. In most respects they conformed to the current diagnostic criteria in DSM-IV (box 1).³

Anorexia nervosa is not easily confused with other disorders if one keeps in mind the core symptoms: deliberate weight loss, disturbed body image and amenorrhoea (box 2). However, one should be aware that many patients conceal their weight reduction strategies such as self-induced vomiting, laxative and diet pill abuse and compulsive exercise, and often give false accounts of their eating habits. Anorexia nervosa should therefore be considered in the differential diagnosis of any unexplained weight loss, and may, for example, be confused with insulin-dependent diabetes mellitus, thyrotoxicosis, malignancy, malabsorption syndromes or infective conditions such as HIV. Anorexia nervosa may also be confused with a depressive disorder, but the latter is characterised by a disinterest in (rather than an avoidance of) food, and there is unlikely to be a preoccupation with weight and shape. Common physical findings include emaciation, bradycardia, hypotension, pallor, acrocyanosis, ankle oedema and breast atrophy with scanty pubic hair. Stunting of growth due to premature epiphyseal fusion may be evident in individuals with an early pubertal onset.⁴

Aetiology: constitutional contributions

“The immediate cause of this distemper I apprehend to be in the system of the nerves proceeding from a preternatural state of the animal spirits, and the destruction of the tone of the nerves . . .” (Morton).¹ The biology of anorexia nervosa has generated diverse theories, such as that of pituitary dysfunction prevalent in the first half of this century, without a great deal of supporting evidence.⁵ More recently, the question of biological aetiology has been approached via the study of the biochemistry of appetite and eating behaviour, in animals as well as humans. These investigations have pointed towards the importance of, for example, dopamine, noradrenaline, endogenous opiates, and other peptides such as CCK, neuropeptide Y and substance P. This is a confused and confusing body of research, but there is one neurotransmitter which has emerged with more credentials than most: serotonin. In the first place, serotonin (5-hydroxytryptamine or 5-HT) has been shown in animal experiments to be responsible for suppression of food intake and may be selectively involved in satiation. Serotonin may regulate body weight, via thermogenesis and activity as

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DSM-IV diagnostic criteria for 307.1 Anorexia nervosa

- refusal to maintain body weight at or above a minimally normal weight for age and height (eg, 85% of that expected)
- intense fear of gaining weight or becoming fat, even though underweight
- disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight
- in postmenarcheal females, amenorrhoea, ie, the absence of at least three consecutive menstrual cycles

Specify type:

- restricting type
- binge-eating/purging type

Box 1

Anorexia nervosa: typical case history

Diana, a 28-year-old woman, was referred to a specialist eating disorder unit with a history of restricted eating and self-induced vomiting from the age of 21. With regard to her personal background, she was born prematurely with hydrocephalus, which was corrected surgically. Her parents, both teachers, were very protective of her as a child. Diana was shy with few friends, and avoided any contact with boys. She directed all her energy into her schoolwork and left school with good results. In her final year at university she made a potentially lethal suicide attempt just prior to her final examinations, and her anorexia nervosa developed soon afterwards. At the age of 23 she was admitted to a general hospital, and over the following four years she was re-admitted on numerous occasions and treated with nasogastric tube feeding, chlorpromazine and insulin. During this time her weight fluctuated between 27 kg and 45 kg. At the age of 27 she was referred to a psychiatric hospital, and referred to the specialist unit a few months later, when she weighed 31 kg (60% of her ideal weight). At the specialist unit she was required to gain weight up to a target of 55 kg, and was treated with individual psychotherapy, group therapy and family therapy. She succeeded in gaining this weight, despite increasing anxiety, depression and suicidal feelings, and continued with treatment as an outpatient. Two years later Diana is still in treatment, despite having lost weight again to 46 kg.

Box 2

well as feeding, and may control nutrient selection, favouring carbohydrate intake. Appetite, the stress response, mood, sexual and reproductive function are all influenced by serotonin, making it a plausible element in the aetiology of anorexia nervosa. Furthermore, in humans, serotonin function is dramatically different in men and women, which fits with the epidemiology of the eating disorder. Studies of anorectic patients have suggested attenuated serotonin function at low weight, and possibly elevated serotonin function following weight restoration. Although some authors suggest that abnormal brain serotonin function represents a biological diathesis for anorexia nervosa, the causative relationship remains unclear. Disturbed serotonin activity may be secondary to the state of starvation, or may be related to non-specific elements of the disorder such as disorders of mood or impulse control.⁶⁻⁹ There is clearly a genetic element in the aetiology of anorexia nervosa, as monozygotic twins have a concordance of 55% for this disorder whereas dizygotic twins have a concordance of only 7%.¹⁰ This vulnerability may be mediated, however, by diverse factors such as growth rate in childhood, a predisposition to obesity, personality type, or predisposition to psychiatric illness in general.

Treatment: medical

“The medical treatment probably need not be considered as contributing much to the recovery. The want of appetite is, I believe, due to a morbid mental state” (Gull).¹ Early authorities regarded loss of appetite as essential for the diagnosis of anorexia nervosa, but there has been a progressive realisation that while most anorectics deny hunger, they are in fact in a starvation-driven state of extreme preoccupation with food.¹¹ Anorectics defend themselves against this craving with stubborn determination, and it is not surprising that appetite-stimulating drugs have been of so little therapeutic use. Lasègue would have predicted this: “Woe to the physician who, misunderstanding the peril, treats as a fancy without object or duration an obstinacy which he hopes to vanquish by medicines, friendly advice or by the still more defective resource, intimidation.”¹ Drugs used to promote food intake and weight gain such as cyproheptadine, amitriptyline, clonidine and opiate antagonists, have all provided disappointing results.¹² Paradoxically, one might expect drugs with appetite-suppressant effects to be more useful, as these might ameliorate the intense anxiety with which anorectics regard mealtimes, and facilitate behavioural control of eating. In the light of the recently elucidated role of serotonin in appetite regulation, there has been some tentative exploration of the use of fluoxetine, a selective serotonin re-uptake inhibitor (SSRI) with appetite-suppressant effects.^{13,14} A number of open trials using fluoxetine in weight-restored anorectics have had promising results, but we should await the results of blind and controlled treatment studies before using SSRIs in clinical practice. In 1995, as in 1874, there is little if any role for drugs in the treatment of anorexia nervosa.

Arrest of further physical deterioration and reversal of weight loss has to be the first step in any treatment programme. Anorexia nervosa is a potentially life-threatening condition with a mortality of up to 20% from a variety of causes at 20-year follow-up,¹⁵ and the clinician should be aware of the range of physical complications of this psychiatric disorder (box 3).^{4,16} The most serious complication is the risk of sudden death from cardiac arrhythmias, in the context of myocardial atrophy and the hypokalemia consequent on self-induced vomiting. Furthermore, the state of starvation accounts for many of the psychological characteristics of the ill anorectic, such as obsessionality, preoccupation with food, irritability and apathy, so psychotherapy cannot proceed until the state of starvation is corrected. Nasogastric or parenteral feeding is to be used only in extreme situations as a life-saving measure, not only because rapid re-feeding has been known to cause life-threatening complications in some cases (hypophosphatemia and pneumothorax in the case of parenteral nutrition),⁴ but also because of the effect on other, interpersonal aspects of treatment.

Aetiology: family and psychodynamic contributions

Anorexia nervosa has always been associated with psychological determinants, and among the classical authors, these antecedents were most clearly articulated by Lasègue: “A young girl, between fifteen and twenty years of age, suffers from some emotion which she avows or conceals. Generally it relates to some real or imaginary marriage project, to a violence done to some sympathy, or to some more or less conscient desire.”¹ The conflict was generally perceived to be of a sexual nature, and during the zenith of classical psychoanalysis this was formulated as the so-called ‘oral impregnation fantasy’. Careful investigators

Figure 1 An anorectic with life-threatening weight loss

Anorexia nervosa: medical complications

- cardiovascular: bradycardia, hypotension, ventricular arrhythmias, congestive cardiac failure (terminal event)
- gastrointestinal: erosion of dental enamel, benign parotid enlargement, oesophagitis and oesophageal ulcers, acute gastric dilatation, acute pancreatitis
- renal: electrolyte abnormalities, hypokalemia, hyponatremia, hypochloremia, hypophosphatemia, hypokalemic nephropathy
- haematological: pancytopenia, bone marrow hypoplasia
- skeletal: osteoporosis, pathological fractures
- endocrine: hypothalamic hypogonadism, high cortisol with DST non-suppression, neurogenic diabetes insipidus
- metabolic: impaired temperature regulation, impaired glucose tolerance, hypercholesterolemia
- dermatological: lanugo, carotenoderma, thin, dry skin, purpura

Box 3

failed to find any evidence of such fantasies except in the minds of psychoanalysts, however.⁵

We owe much of our current psychodynamic understanding of anorexia nervosa to the overlapping ideas of Bruch and Crisp.^{5,11,17} Both authors reject any simplistic symbolic interpretations of the patients' refusal to eat, and see the illness in the context of the girls' current family and social relationships, rather than the distant experiences of infancy. Bruch saw them as children of overinvolved mothers, with a poorly developed identity and a pervasive sense of ineffectiveness. In adolescence, faced with the prospect of physical and emotional separation from their mothers and families, they resort to dieting as a way of preserving their self-respect and sense of autonomy. On the other hand, Crisp saw anorexia nervosa as a phobic avoidance of normal body weight as a means of a 'psycho-biological' regression (a delay or reversal of the normal maturational processes) from the developmental challenges of puberty, specifically with regard to sexual development, heterosexual relationships and separating from the family of origin. Crisp saw that the parents of these girls also had avoidant coping styles, and were often themselves troubled by sexual conflicts and poor self-esteem. As their children entered puberty, their developing sexuality and autonomy threatened to destabilise the family.

The family perspective on anorexia nervosa was also apparent to Lasègue, who stated that "It must not cause surprise to find me thus always placing in parallel the morbid condition of the hysterical subject and the preoccupations of those who surround her. These two circumstances are intimately connected, and we should acquire an erroneous idea of the disease by confining ourselves to an examination of the patient. . . the hysterical subject has been constituted really a sick person, no longer taking part in the free movements of common life."¹ Lasègue was suggesting that the family derives some benefit from it's preoccupation with the young girl's condition, and this anticipated the highly influential formulations of Minuchin and Palazzoli, a century later.^{18,19} Minuchin suggested that the families of anorectics are characterised by an inability to tolerate and resolve conflict within the family: consequently they become rigid and unable to adapt to change, for example, when a daughter enters adolescence and threatens to separate from the family. Rather than facing their own differences, the parents might involve the daughter in a triangular relationship in which she (and her anorexia nervosa) becomes the focus of all anxiety and concern. Palazzoli focused on the cross-generational alliances and the strategies employed by the families to avoid the direct expression and resolution of discord. Families of anorectics are often of social class I, II and III, and place great value on educational and social achievement. There has subsequently been some experimental validation of Minuchin's concepts of enmeshment, overprotectiveness, rigidity and lack of conflict resolution.²⁰

Figure 2 Stepped care for anorexia nervosa. (From Royal College of Psychiatrists Council Report CR14. *Eating disorders*, 1992, with permission)

The role of the general practitioner

- pick up young people at risk of anorexia nervosa or showing early signs of the illness (exaggerated concern about weight or shape, disturbed eating patterns, declining weight)
- in such cases provide advice about diet and about anorexia nervosa, and monitor weight
- provide the young person and her family with self-help information
- carry out physical examination, and special investigations (haematology, electrolytes, serum protein, possibly radiology) if indicated
- make the diagnosis of overt anorexia nervosa based on standard diagnostic criteria, and refer to general psychiatric or specialist services
- engage in a working relationship with the patient and her family and help them engage with specialist services
- this is a chronic disorder. The general practitioner can be a line of continuity between various medical and psychiatric services, perhaps over a period of decades
- with established chronic or recurring cases, monitor weight and be aware of possible medical complications

Box 4

Treatment: psychological

“The treatment required is obviously that which is fitted for persons of unsound mind. The patients should be fed at regular intervals, and surrounded by persons who would have moral control over them; relations and friends being generally the worst attendants.” (Gull).¹

A well-trying approach is a multidisciplinary in-patient(s) treatment programme which attempts in the first place to facilitate weight gain through close nursing supervision, using behavioural and cognitive principles.²¹ It is recognised that weight restoration is associated with intense distress and anxiety, requiring a great deal of psychological support from nursing staff. Behaviour therapy, in the simple sense of graded privileges and rewards for weight gain, does not seem to be effective in isolation; patients often ‘eat their way out’ of hospital and relapse soon afterwards.²² The multidisciplinary packages often include a component of cognitive therapy, which educates the anorectic about the nature of the disorder and about nutritional principles, encourages self-monitoring and helps the patient to modify her dysfunctional thoughts about weight and shape.²³ Once weight has been restored, the family and intrapsychic aspects of the illness are addressed, using individual, group and family therapies.²¹

Although Bruch was sceptical of the efficacy of classical psychoanalysis¹⁷ an eclectic form of psychodynamic psychotherapy does seem to be effective, even when used with out-patient(s) in a fairly brief programme.²⁴ Dietary advice alone has been found to be surprisingly helpful, but this intervention was carried out by highly specialised dieticians and probably contained several elements of the cognitive behavioural therapy described by Fairburn.²³ A randomised, controlled trial has compared the in-patient(s) treatment described above with out-patient(s) individual and family therapy, out-patient(s) group therapy for patients and parents separately, and assessment only.²⁵ In this study all three treatment groups had significantly better outcomes at one year than the assessment only group, many of whom had extensive treatment elsewhere. It was noteworthy, however, that patients in the two out-patient(s) groups appeared to sustain a higher weight at one year than the in-patient(s) group: it is likely that treating patients while they are in their usual environment may bolster their resistance to relapses. A separate series of studies have examined the effectiveness of individual *versus* family therapy in patients who have restored their weight in hospital.^{26–28} It appears that family therapy is the treatment of choice in adolescent patients and in those with an illness of relatively brief duration, whereas adult patients and those with a chronic illness tend to do somewhat better with individual treatment.²⁶ Two further studies clarified that treatment of adolescents and adults, respectively.^{27,28} In adult patients, psychoanalytic psychotherapy was not much more effective than simple supportive psychotherapy, and in adolescent patients, there was a similar response to conventional whole-family therapy and to ‘family counselling’ (in which the parents are seen separately while the child has individual supportive sessions). The latter option seemed to work best in families with high levels of hostile and critical interaction.

An approach to the anorectic patient

These clinical trials should be interpreted with circumspection because of the enormous practical difficulties in psychotherapy research,^{25,28} but we are in a position to set out guidelines for the treatment of anorectic patients (figure 2).^{29,30} In the first instance, cases need to be identified early by general practitioners, paediatricians or physicians who come into contact with girls who are underweight or unduly preoccupied with their weight and shape (box 4). I have referred to anorectics throughout as female, but it should be remembered that young men account for about 10% of cases of anorexia nervosa.¹¹ Pre-clinical cases can be managed by dietary advice and counselling in a primary care setting, and information should be provided about self-help resources (Appendix). If weight loss progresses and the individual meets the diagnostic criteria for the condition (box 1),³ she should be referred to an adolescent or adult psychiatrist. At this point she should undergo a thorough multidisciplinary assessment, which would include assessment of her physical condition, her personal psychopathology and the family system. The general psychiatrist should be able to intervene by providing individual supportive psychotherapy, with counselling for the parents if the patient is still living at home, and many adolescent units can provide specialised family therapy and in-patient(s) treatment. Patients with chronic, refractory illnesses or recurrent episodes should be referred to specialist eating disorder units, which may admit for in-patient(s) treatment or attempt

out-patient(s) or day-patient treatment with suitable cases. Emergency admission to a medical or general psychiatric ward is indicated in the event of life-threatening physical deterioration, or depression when there is a concern about suicide. In extreme cases, use of compulsory admission under the mental health act may be necessary, but this is to be avoided if at all possible as such coercion may do irreparable harm to the long-term therapeutic relationship and to the patient's already vulnerable sense of self-esteem and autonomy. We have refined our understanding of anorexia nervosa since 1694, but in the end it is difficult to improve on Morton's recommendations of '... the conversation of his friends ... for this disease does always proceed from sadness, and anxious cares ... an open, clear and very good air (and) a delicious diet'.¹

Appendix: Self-help resources for eating disorders

The Eating Disorders Association

Sackville Place, 44 Magdalen Street, Norwich, Norfolk NR3 1JE, UK Tel 01603 621414

Further reading:

- *Anorexia nervosa and the wish to change*. AH Crisp, N Joughlin, C Halek, C Bowyer. Department of Mental Health Sciences, St George's Hospital Medical School, London, 1989.
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