Eating disorders

Janet Treasure

Abstract

Eating disorders are common problems affecting 5–10% of young people. The bulimic forms of eating disorders became dominant in the last half of the twentieth century and are caused in part by the tensions between the easy availability of high-palatability food and a culture that idealizes thinness. Genetic factors, high anxiety and environment stress also contribute. Anorexia nervosa is associated with high levels of physical disability but problems also occur in social, vocational and parenting functioning. Psychological forms of treatment are effective. In the case of anorexia nervosa it is particularly helpful if the family are involved as they can be drawn into unhelpful patterns of interaction.

Keywords anorexia nervosa; binge-eating disorder; bulimia nervosa; eating disorders

What's new?

- Starvation, stress, and intermittent access to high-calorie food are the conditions that lead to binge-eating in animals. The underlying process is similar to that involved in the addictive use of substances such as cocaine, amfetamine and alcohol. Binge-eating may be a gateway into addiction
- Epidemiological surveys show that the lifetime prevalence of the bulimic forms of eating disorders continues to increase
- Sharing information and skills with family members improves patient's well-being and may improve the outcome of the eating disorder
- Drugs used for obesity can be beneficial for people with binge-eating disorder

Definition

Anorexia nervosa (AN) is defined according to Diagnostic and statistical manual of the American Psychiatric Association (DSM-IV) as a refusal to maintain weight at or above a minimally normal weight (< 85% of expected weight for age and height, or body mass index < 17.5 kg/m^2), or a failure to show the expected weight gain during growth. In association with this there is often an intense fear of gaining weight, preoccupation with weight, denial of the current low weight and its adverse impact on health, and amenorrhoea. Bulimia nervosa (BN) shares some features with AN, such as an intense preoccupation with weight and shape; however, it is characterized by regular episodes of uncontrolled eating (> 1000 kcal in one sitting) associated with various methods of counteracting weight gain (vomiting and laxative abuse being most common). Binge-eating disorder (BED) is characterized by significant binge eating in the absence of extreme compensatory behaviours, such as purging, and so is associated with obesity.

Epidemiology

Eating disorders (ED) and related behaviours are a common problem in pre-adolescents and adolescents. A recent study on a large sample of American young people (aged 9–14 years) found that 34% of boys and 43.5% of girls had some eating disorder traits.[1]

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The lifetime female prevalence rates from the USA National Comorbidity study are: 0.9 AN, 1.5 BN, 3.5 BED, 0.6 subclinical BED percentage of the population[2]; similar rates are found in Europe.[3,4] Comorbidity between disorders is the rule rather than the exception.

Abundant historical records of AN date from the 19th century. The clinical and behavioural features are present in diverse cultural settings but the content of the psychopathology varies over time and place. BN was recognized as a new syndrome in 1979.[5] It is more common in urban areas and the peak onset is at times of transition to college at the age of 18 years. (A degree of autonomy and disconnection from the social aspects of eating are needed to allow the illness to develop.) BED has not yet been accredited as a new syndrome in the American Psychiatric Association's diagnostic and statistical manual and is part of the group of 'eating disorder not otherwise specified' (EDNOS) disorders. These form a residual category who can have severe impairment and present for treatment but do not meet the full criteria for the prototypical disorders. BED is particularly common in clinical populations with obesity and diabetes. The gender bias varies across diagnosis and setting. For example, the female to male ratio is 10 to 1 for AN and BN, but in the community and in child and adolescent settings the ratio is 3 to 1 and in BED there is less of a disparity between the sexes. The finding that the proportion of male cases presenting to clinical services is less than that found in the community suggests that there are barriers in terms of awareness, recognition and stigma about having a 'girl's illness'.

Pathology and pathogenesis

Family, biological, social, and cultural factors may play a role in either the development or the maintenance of eating disorders. The evidence for such risk factors is collated in two systematic reviews.[6,7] Reviews focusing on the biological risk factors have been recently compiled.[8–10] The risk of eating disorders in first-degree relatives is increased ten-fold and twin studies suggest that this is due to inherited factors. Other psychiatric disorders such as depression, generalized anxiety disorder, and

obsessive-compulsive disorder are slightly over represented in the families of people with eating disorders. Family members also have a three-fold risk of obsessive–compulsive personality disorder (see also pages 436-441). Furthermore, obsessive–compulsive traits, such as perfectionism, and rigidity in childhood increase the risk of developing an eating disorder.[11] Prematurity, particularly if the baby was small for gestational age, increases the risk of developing AN six-fold. Later, adversity during childhood, such as sexual and physical abuse, increases the risk of bulimic disorders.[12] Also, the risk of BN is increased amongst those who have had a robust appetite during childhood, were heavy and who were teased or criticized about their weight or eating. A model showing the evolution of risk factors into the development of eating disorders is shown in Figure 1.[8,9,13,14]

Diagnosis

A positive diagnosis of an eating disorder is usually made from the history, especially if informants contribute to the consultation. It is useful to screen for eating disorders in populations with high risk (e.g. diabetes, college students) and in pregnancy where the risk for the unborn child can be increased (Figure 2). The screening questions should include asking for a pattern of extreme weight fluctuations (especially if they have caused concern to others), an increased salience of food and shape/weight issues, and extreme weight control behaviours (vomiting, fasting, exercise, etc.). In a typical case it is necessary to exclude other medical conditions (Table 1). Assessment procedures for use by student counsellors or in general practice are available online.[15]

Course

Over 50% of cases of AN have a protracted course over 6 years and a third of cases never make a full recovery. Approximately a third of cases develop bulimic features. People with BN have a relapsing and remitting course. Fewer than 10% have a persistent eating disorder at 10 years. BED also persists over many years.[16]

Investigations

An erythrocyte sedimentation test and thyroid function tests rule out the most common differential diagnoses. It is rare for albumin to be reduced, but haematological deficiencies and electrolyte disturbances are common (see also pages 393-398). The most useful investigations needed to define the acute medical risk are shown in Table 2. The majority of body systems are affected by the negative energy balance. The markers of nutritional decompensation which signal the need for urgent care are shown in Table 2.

Management

Stigma

Eating disorders are often dismissed by the lay public and even professionals as merely slimming taken to extreme for reasons of vanity. The media's interest in size zero fuels this misperception.[17] However, the cost of these illnesses for the individual, their family and society is high and it is important that prevention, early intervention and treatment are available.

Engagement

The most difficult aspect of management is engaging the person with eating disorders into treatment. This is particularly pertinent for AN in which it is a characteristic feature that the individual concerned does not accept that anything is wrong, which is in contrast to the concerns of close others. This can cause conflict (usually covert) within the medical encounter echoing that seen within the family. Issues of confidentiality frequently cause problems. Models of health behaviour change can help structure the therapeutic interaction. Most people with AN are not ready for active

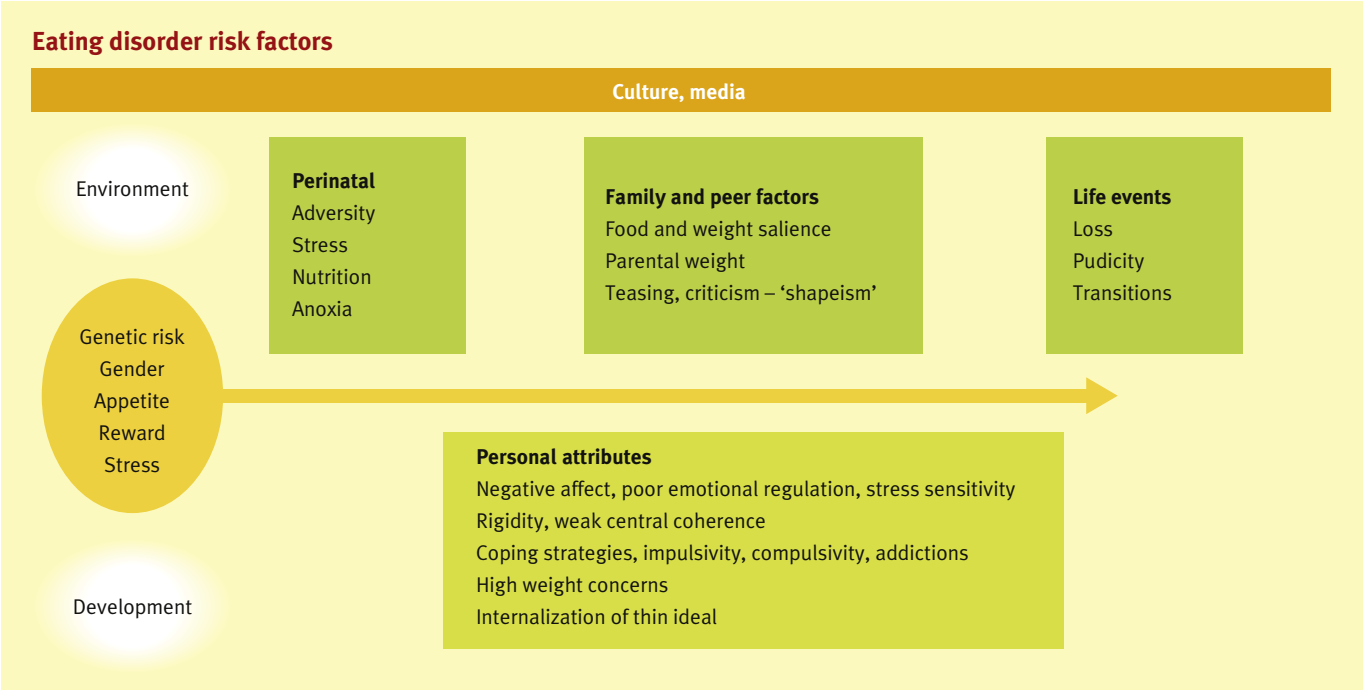

Figure 1

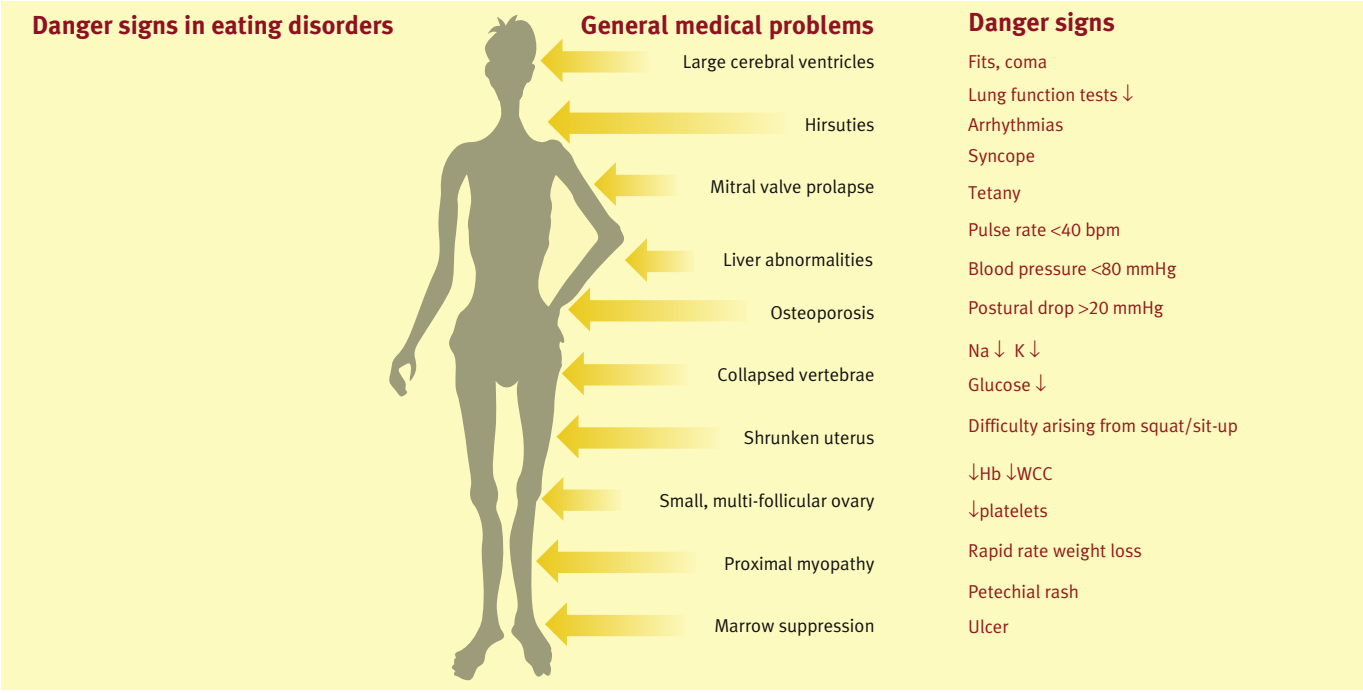

Figure 2

change and hold positive beliefs about the symptoms and interpersonal consequences. Techniques such as motivational interviewing can be used to explore mixed feelings about changes to move people towards action. People with BN want to stop their binging but may be reluctant to stop their weight control strategies.

Inpatient treatment should be considered if the person falls into the high risk-category. If, despite maximal support (including meeting with the family) in outpatients, the individual remains at risk yet declines admission then use of the Mental Health Act may be appropriate.

Treatment strategies

Treatment for AN involves a focus on restoration of the nutritional balance in combination with psychotherapeutic techniques to overcome factors that maintain the illness. The core maintaining factors which vary across the diagnoses are described in a recent model and include the following.[14,18]

Differential diagnosis

- Differential diagnosis of weight loss – malabsorption (e.g. coeliac disease or inflammatory bowel disease), neoplasm, illicit drug use, infection (e.g. tuberculosis), autoimmune disease, endocrine disorders (e.g. hyperthyroidism, Addison's disease)
- Differential diagnosis of amenorrhoea – pregnancy, primary ovarian failure, polycystic ovary syndrome, pituitary prolactinoma, uterine problems and other hypothalamic causes
- Psychiatric differential diagnosis – depression, obsessive compulsive disorder, somatization and, rarely, psychosis

Table 1

- A predisposition to obsessive–compulsive traits which is underpinned by inflexibility and a bias to detail over the bigger picture.
- High levels of anxiety with avoidance, dependence and compulsivity used as a coping strategy particularly for AN. Impulsivity and addictive behaviours more common in those with BN and BED.
- Interpersonal factors can play a key role in AN where the core symptoms are highly visible and can lead family members to accommodate to the behaviours with over-protection or criticism (high expressed emotion) which can escalate the spiral of anxiety.[19]

A variety of pro-eating disorder beliefs develop in association with the above mechanisms.

It is usually helpful to include carers in treatment particularly for AN in which the interpersonal element is a core maintaining feature. Books which provide information and describe management strategies are available.[20]

Cognitive–behavioural treatment produces full remission in 40% of people with BN[21] and is recommended in the NICE guidelines. Again books and web-based programmes are available.

New treatments which have more of a focus on the emotional and information processing aspects of these disorders are in process of development.

Stabilization of 'at risk' cases

Nutritional risk – the deficits in AN develop slowly and are general rather than specific. Therefore, it is preferable to rectify them slowly, orally and with ordinary food supplemented with multi-vitamin/multi-mineral preparations (e.g. Forceval 1–2 or Seravit capsules). In the first phase (3–7 days), a soft diet of approximately 30–40 kcal/kg/day spaced in small portions throughout the day is recommended.

Measuring risk parameters in people with eating disorders

	Parameter	Moderate risk	High risk
Nutrition	BMI	<15	<13
	BMI centiles	<3	<2
	Weight loss per week (kg)	>0.5	>1.0
	Purpuric rash		+
Circulation	Systolic blood pressure (BP) (mmHg)	<90	<80
	Diastolic BP (mmHg)	<60	<50
	Postural drop (mmHg)	>10	>20
	Pulse rate (beats per minute)	<50	<40
	Oxygen saturation (%)	<90	<85
	Extremities		Dark blue/cold
Musculoskeletal (squat test*)	Unable to get up without using arms for balance	+	
	Unable to get up without using arms as leverage		+
Core temperature (°C)		<35	<34.5 °C
Blood tests	Full blood count, urea, electrolytes, (incl. PO_4), liver function tests, albumin, creatinine kinase, glucose	Concern if outside normal limits	K <2.5 Na <130 PO_4 < 0.5
Electrocardiogram (ECG)		Rate <50	Rate <40 Prolonged QT interval

*The squat test: see if the patient can stand up from a squat position without, if possible, using her hands.

Table 2

Fluid and electrolyte balance – some weight-change strategies such as vomiting, diuretic and laxative abuse can result in severe dehydration (or over-hydration), acute renal failure and electrolyte imbalance. Oral replacement is preferable (e.g. using Dioralyte). Serum potassium levels may remain low even with potassium supplements if vomiting persists. It has been suggested that proton pump inhibitors (such as lanzoprazole) to inhibit gastric acid secretion may reduce metabolic alkalosis and help to conserve potassium.

Medication – the risk of harm, for example from the consequences of QT prolongation (on ECG), needs to weighed against the negligible evidence of benefit from antidepressants, antipsychotics and antihistamines in patients at low weight. Small doses of promethazine or atypical antipsychotic drugs can help the severe anxiety and overactivity associated with refeeding.

Refeeding – the primary goal is to facilitate nutritional self management. However, in some cultures, particularly in high-risk adolescents, tube feeding is used in the early phase of treatment. A recent randomized controlled trial (RCT) from France reported superior weight gain and maintenance with less binge-eating.[22] However, parenteral feeding may increase the risk of re-feeding syndrome and has other costs (exposure radiation, dependency, etc.). This is rarely needed if skilled nursing with assisted feeding is available in specialized units.

Refeeding syndrome

Severe medical complications, collectively known as the refeeding syndrome, can occur, especially in those with a BMI less than 12 kg/m^2 who binge and purge and those with concurrent physical conditions. Close monitoring is necessary as a range of electrolyte disturbances, including hypokalaemia, hypocalcaemia and hypomagnesaemia, and physical complications can occur during refeeding. Fatal consequences such as acute gastric dilatation and hypophosphataemia can occur quickly. Acute gastric dilatation can occur with rapid refeeding or if the patient binges. Hypophosphataemia can develop with a high carbohydrate load (including if given intravenous dextrose); if severe, it can cause cardiac and respiratory failure, delirium and fits.

Longer-term medical complications

Osteoporosis results from prolonged poor nutrition during the critical phase of development of peak bone mass. The most impressive treatment is weight restoration which can increase bone density by 10% a year. The place of hormonal replacement therapy is uncertain and can be harmful by causing premature closure of the epiphyses. The restoration of menstruation can be delayed especially if dietary abnormalities persist.

Prognosis

The factors that are relevant to the prognosis of AN are shown in Table 3. There is less certainty about the relevant prognostic factors in BN.

Prognostic factors in anorexia nervosa

- Severity illness: duration, degree of weight loss, hyperactivity
- Comorbidity: bulimia and purging, obsessive–compulsive symptoms, childhood difficulties
- Response to treatment: poor weight gain, weight remains below normal levels
- Age of onset (younger do better)

Table 3

Follow-up

The NICE guidelines recommend that people admitted to hospital with AN have continued treatment as an outpatient for a minimum of a year. Regular medical reviews are essential for severe enduring AN.

Prevention

General strategies such as fostering high self-esteem and good communication and emotional regulation skills in children set the scene for psychological well-being. More specific approaches such as moderating extreme personality traits such as compulsivity, threat sensitivity and impulsivity may also be of benefit. A Cochrane systematic review of prevention is available.[23] ◆

REFERENCES

1 Ackard DM, Fulkerson JA, Neumark-Sztainer D. Prevalence and utility of DSM-IV eating disorder diagnostic criteria among youth. *Int J Eat Disord* 2007; **40:** 409–17.

2 Hudson JI, Hiripi E, Pope Jr. HG, Kessler RC. The prevalence and correlates of eating disorders in the national comorbidity survey replication. *Biol Psychiatry* 2007; **61:** 348–58.

3 Wittchen HU, Jacobi F. Size and burden of mental disorders in Europe–a critical review and appraisal of 27 studies. *Eur Neuropsychopharmacol* 2005; **15:** 357–76.

4 Jacobi F, Wittchen HU, Holting C, et al. Prevalence, co-morbidity and correlates of mental disorders in the general population: results from the German Health Interview and Examination Survey (GHS). *Psychol Med* 2004; **34:** 597–611.

5 Russell G. Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychol Med* 1979; **9:** 429–48.

6 Stice E. Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychol Bull* 2002; **128:** 825–48.

7 Jacobi C, Hayward C, DeZwaan M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull* 2004; **130:** 19–65.

8 Treasure J. Getting beneath the phenotype of anorexia nervosa: the search for viable endophenotypes and genotypes. *Can J Psychiatry* 2007; **52:** 212–19.

9 Steiger H, Bruce K. Phenotypes, endophenotypes and genotypes in bulimia-spectrum eating disorders. *Can J Psychiatry* 2007; **52:** 220–27.

10 Steiger H. Eating disorder paradigms for the new Millennium do "Attachment " and "culture" appear on brain & genome scans. *Can J Psychiatry* 2007; **52:** 209–11.

11 Anderluh MB, Tchanturia K, Rabe-Hesketh S, Treasure J. Childhood obsessive-compulsive personality traits in adult women with eating disorders: defining a broader eating disorder phenotype. *Am J Psychiatry* 2003; **160:** 242–47.

12 Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *Am J Psychiatry* 2002; **159:** 394–400.

13 Southgate L, Tchanturia K, Treasure J. Building a model of the aetiology of eating disorders by translating experimental neuroscience into clinical practice. *J Mental Health* 2005; **14:** 553–66.

14 Treasure J, Lopez C, Roberts M. Moving towards the use of endophenotypes in eating disorders in order to make progress in terms of aetiologically based diagnosis, and treatment focused on the pathophysiology. *Paediatric Health* 2007; **1**.

15 www.eatingresearch.com

16 Berkman ND, Lohr KN, Bulik CM. Outcomes of eating disorders: a systematic review of the literature. *Int J Eat Disord* 2007; **40:** 293–309.

17 Treasure JL, Wack ER, Roberts ME. Models as a high-risk group: the health implications of a size zero culture. *Br J Psychiatry* 2008; **192:** 243–44.

18 Schmidt U, Treasure J. Anorexia nervosa: valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. *Br J Clin Psychol* 2006; **45:** 1–25.

19 Treasure J, Sepulveda A, Whitaker W, Todd G, Lopez C, Whitney J. Collaborative care between professionals and non professionals in the management of eating disorders: A description of workshops focused on interpersonal maintaining factors. *Eur Eat Disord Rev* 2007; **15:** 24–34.

20 Treasure J, Smith G, Crane A. Skills-based learning in caring for a loved one with an eating disorder: the new Maudsley method. Oxford: Routledge, 2007.

21 Hay PJ, Bacaltchuk J. Psychotherapy for bulimia nervosa and binging. *Cochrane Database Syst Rev* 2003;(1)**:** CD000562.

22 Rigaud D, Brondel L, Poupard AT, Talonneau I, Brun JM. A randomized trial on the efficacy of a 2-month tube feeding regimen in anorexia nervosa: A 1-year follow-up study. *Clin Nutr* 2007; **26:** 421–9.

23 Pratt BM, Woolfenden SR. Interventions for preventing eating disorders in children and adolescents. *Cochrane Database Syst Rev* 2002;(2)**:** CD002891.

FURTHER READING

Birmingham C, Beumont P. Anorexia nervosa: A practical guide to medical management. Cambridge: Cambridge University Press, 2002.

Crisp AH, Joughin N, Halek C, Bowyer C. Anorexia nervosa. The wish to change. Hove, East Sussex: Psychology Press, 1996.

Schmidt U, Treasure J. Getting better bit(e) by bit(e). Oxford: Brunner-Routledge, 1993.

Treasure J. Anorexia nervosa. A survival guide for sufferers and those caring for someone with an eating disorder. Hove: Psychology Press, 1997.

Treasure J, Schmidt U, Van Furth E. Handbook of eating disorders. Chichester: John Wiley & Sons, 2003.

Treasure J, Smith G, Crane A. Skills-based learning for caring for a loved one with an eating disorder. The New Maudsley Method. London & New York: Routledge, 2007.

Useful websites

Institute of Psychiatry/Maudsley Hospital for professionals, carers and users. www.eatingresearch.com
UK B-eat the users and carers organisation. www.b-eat.co.uk
US Eating Disorders Association. www.nationaleatingdisorders.org
NICE eating disorder guidelines. www.nice.org.uk

Practice points

- Approximately 10% of young women have some form of eating problems.
- A raised ESR should raise the suspicion of an alternative diagnosis.
- It is hard to engage people with anorexia nervosa into treatment as they hold positive attitudes about anorexia nervosa and they do not want to change.
- Bulimia nervosa is treated by cognitive–behavioural therapy (self-help adaptations are available).
- Admission should be considered if there are signs of any of the following: myopathy, reduced core temperature, circulatory decompensation, abnormal biochemistry and haematology.
- Wherever possible give nutrient and mineral replacements orally. Close physical monitoring is essential to prevent refeeding syndrome.
- Stabilization of high-risk anorexia nervosa requires the gradual introduction of food (starting with approximately 1000 kcal) supplemented with standard doses of vitamin and mineral supplements.