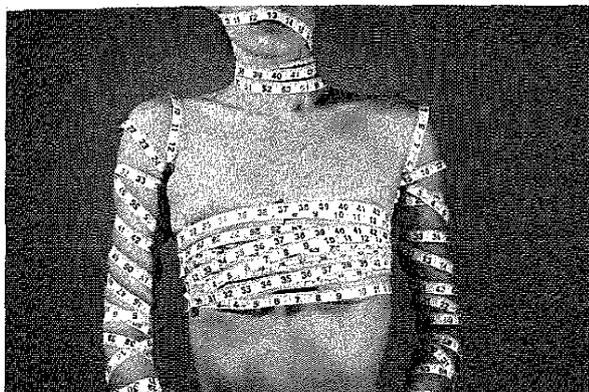


Anorexia nervosa and bulimia nervosa

By Michelle Bellingan



Eating disorders typically occur in adolescent girls or young women, although recent findings indicate that these disorders are increasingly being diagnosed in older women and in younger children.

Anorexia nervosa (AN) and bulimia nervosa (BN) are the two best characterised eating disorders. According to the DSM-IV classification system, patients who do not meet the criteria for either AN or BN may be diagnosed as 'eating disorder not otherwise specified'. Binge-eating disorder has been the most intensively researched disorder within the category of 'eating disorder not classified' (EDNOS).¹

Table 1 outlines the DSM-IV criteria used in the diagnosis of AN and BN.

AN may be defined by the successful pursuit of thinness through dietary restriction and other measures, resulting in a body weight below the normal range (<17.5 kg/m²). BN, by contrast, is characterised by recurrent binge-eating and regular compensatory behaviour to influence the individual's body weight and shape. The body weight of individuals with BN is typically normal or low normal, although it should be considered that the condition may occur in overweight individuals too.¹

In addition to their effects on psychological wellbeing, eating disorders have potentially devastating effects on health through the physiological sequelae of altered nutritional status or purging.³ The mortality rate associated with AN alone, at 0.56% per year, is more than 12 times as high as the mortality rate among young women in the general population.⁴

Epidemiology and outcomes

AN and BN affect an estimated 0.3-0.7% and 1.5-2.5%, respectively, of females in the general population.⁵ Eating disorders typically occur in adolescent girls or young women, although recent findings indicate that these disorders are increasingly being diagnosed in older women and in younger children.³ Girls as young as 10 and 11 are being diagnosed with AN.⁶ Despite the female predisposition to eating disorders, 5-15% of cases of AN and BN occur in males.³

Table 1: DSM-IV Diagnostic criteria for anorexia nervosa and bulimia nervosa²

DSM-IV Diagnostic criteria for anorexia nervosa	
A	Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g. weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
B	Intense fear of gaining weight or becoming fat, even though underweight.
C	Disturbance in the way in which one's body weight 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.
D	In postmenarcheal females, amenorrhoea, i.e. the absence of at least three consecutive menstrual cycles.
DSM-IV Diagnostic criteria for bulimia nervosa	
A	Recurrent episodes of binge-eating. An episode of binge-eating is characterised by the following (1) eating, in a discrete time period (e.g. within any two hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances (2) a sense of lack of control over eating during the episode (e.g. a feeling that one cannot stop eating or control what or how much one is eating).
B	Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting or excessive exercise.
C	The binge-eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for three months.
D	Self-evaluation is unduly influenced by body shape and weight.
E	This disorder does not occur exclusively during episodes of anorexia nervosa.

Long-term follow-up studies indicate that nearly 50% of patients with AN make a full recovery. It is estimated that 20-30% of these individuals show residual symptoms, while 20% have no substantial improvement in symptoms. Tragically, 5-10% of AN sufferers die of a related cause.^{1,3} Death from suicide is 32 times that expected in this age group.⁷

BN has a chronic course and is self-perpetuating. Remission over time has been estimated to be from 31-74%. Relapse is, however, very common.⁸ The majority of patients with BN have



Table 2: Signs and symptoms: Anorexia nervosa⁶ and bulimia nervosa¹³

Anorexia nervosa	
Excessive weight loss	The individual avoids eating whenever possible
Hair, nail and skin problems	Lack of nutrients leads to scaly skin, brittle nails and hair loss
Cessation of menstruation	Rigid dieting reduces hormone levels, disrupting the menstrual cycle
Extreme sensitivity to cold temperatures	Loss of muscle and fat makes it difficult for the body to retain heat
Body hair growth	Fine hair (lanugo) may begin to grow on the body in an effort to keep warm
Unusual eating habits	Extremely rigid guidelines about eating, such as eating small bites or restricting themselves to a certain number of bites of food
Weighing often	Sometimes 10 or more times a day
Extreme physical activity	To accelerate weight loss
Social isolation	Withdrawal from their family, friends and peers
Frequent sensations of dizziness and fatigue	As a result of hypotension, hypoglycaemia and/or dehydration
Poor self-esteem	Low self-esteem accompanies (or possibly precedes) anorexia nervosa. This may lead to depression and suicidal tendencies.
Wearing large clothing	The individual resorts to wearing oversized clothing in an attempt to hide their body.
Denial of a problem	Since the person's image of themselves is distorted, they will usually deny that they are obsessed with their weight and insist that they need to lose more weight.
Bulimia nervosa	
Exercises excessively	To accelerate weight loss and to appease the guilt experienced after a bingeing episode
Laxative use, diuretic or diet-pill use	
Russell sign	Cuts and callouses on the back of the hands and knuckles from repeated attempts to induce vomiting
Visits to the bathroom after eating	Self-induced vomiting
Damaged, stained teeth	Dental enamel is eroded by repeated exposure of the teeth to gastric acid (some bulimics vomit up to 7-8 times a day). Dentists are usually the first health care providers to identify the signs of bulimia.
Blood shot eyes	Due to the strain of repeated vomiting

positive outcomes, with up to 50% symptom-free at five years or more.⁹ Childhood obesity, substance abuse and personality disorders are predictors of poor outcomes.¹⁰

Suggested aetiology

The aetiology of eating disorders remains largely unknown, although they appear to be caused by a combination of genetic, neurochemical, psychodevelopmental and sociocultural factors.³ There is a growing acknowledgement that neurobiological vulnerabilities make a substantial contribution to the pathogenesis of AN and BN. Evidence suggests that altered brain serotonin function contributes to the dysregulation of appetite, mood, and impulse control in AN and BN.⁵

There is also significant evidence for a genetic component in the pathogenesis of anorexia nervosa. A region on chromosome 1 has been identified as a susceptibility locus. Research is ongoing to identify which gene or genes in the localised area of chromosome 1 may play a role.¹¹ It is anticipated that there won't be a single gene responsible for anorexia nervosa but rather a number of genes that contribute to a person's predisposition to the illness. Thus far, researchers have identified six core traits that appear to be linked to genes associated with AN and BN. These core traits are: obsessiveness (a form of perfectionism); age at menarche; anxiety; lifetime minimum body mass index (BMI); concern over mistakes and food-related obsessions.

It was found that minimum BMI, concern over mistakes, age at menarche and food-related obsessions were more closely linked to BN; whereas obsessiveness and anxiety were more closely linked to AN.¹²

Symptoms

Table 2 summarises signs and symptoms of AN and BN. There is considerable overlap among the features of eating disorders, particularly in anorexics with the subclassification of 'binge-eating/purging'. In addition to the signs listed in this table, anorexics have a constant preoccupation with food, weight and calories. It is a fallacy that anorexics are never hungry; they are always hungry. Feeling hunger gives them a feeling of control over their lives and their bodies.

Health risks

Both disorders can lead to severe health issues. The starvation of AN causes the body to slow down to preserve energy. Many bodily functions cease to function properly. The kidneys and the liver, in particular, are influenced and show signs of size reduction. In extreme cases, kidney failure results from dehydration. Other problems include: irregular heart rate, extreme muscle loss and very low blood pressure. Blood abnormalities include leucopenia, thrombocytopenia, hypoalbuminaemia. Mild disturbances of thyroid functioning are also common. Related problems that may develop include osteopenia and osteoporosis.⁶ It is important that anorexics be

assessed using bone densitometry since bone loss is a serious problem that can accompany amenorrhoea and under-nutrition. In 50% of women with AN, bone-density measurements are more than 2 SD below normal.¹⁴ Bone loss can occur in young women in as little as six months^{14,15} and can also occur in men. AN usually occurs during adolescence, when accrual of bone mass is at its peak. Bone loss and inadequate bone formation at this stage of development can lead to severe osteopenia.³

BN is perceived as being the less 'harmful' of the two eating disorders. It is important that sufferers are made aware of the damaging effects this disorder has on the digestive system. These digestive changes can result in electrolyte imbalances, which, in turn, damages organs.

Table 3. Common adverse physical effects of bulimia nervosa¹³

Dehydration
Hypokalaemia and cardiac dysrhythmia
Hypochloremic alkalosis
Hypomagnesaemia and hypophosphataemia
Parotid and salivary gland enlargement
Abdominal pain
Bowel irregularities and bloating
Cathartic colon
Chronic constipation
Fertility problems
Gastric or oesophageal rupture (rare but high mortality)

Psychiatric comorbidities

Evaluation for concomitant psychiatric illness should be routine in individuals with eating disorders since these conditions are frequently accompanied by mood, anxiety and personality disorders. In addition, AN is frequently accompanied by obsessive-compulsive disorder. BN and binge-eating disorder are often associated with substance abuse and impulsive behaviours.³

Treatment approaches

Treatment options are available for these eating disorders, however, up to 50% of the cases go unrecognised. A person with an eating disorder may not disclose symptoms or may even conceal them, due to a lack of understanding of the impact on their health, ignorance of available treatment, shame at the prospect of discussing the symptoms, or unwillingness to consider relinquishing them. When eating disorders are detected, even dangerously ill patients can be averse to accepting appropriate treatment.³

The ideal end points of treatment for all eating disorders include the following: stabilisation of medical and nutritional status, identification and resolution of psychosocial

precipitants of the disorder and reestablishment of healthy eating patterns.

Anorexia nervosa

Finding effective treatments for AN remains a challenge. Despite being the 'oldest' eating disorder, AN is highly resistant to numerous forms of intervention.¹

Family therapy is the most researched form of treatment for AN. The Maudsley model,^{16,17} a specific form of family therapy, has had encouraging results. This intervention for adolescent patients involves 10-20 family sessions held over 6-12 months. In the first phase of treatment, the parents are directed to take complete control over their anorexic child's eating and are coached on finding effective methods to do so. Once the child begins to comply with parental authority, external control is reduced. The endpoint of this therapy is the adolescent's right to age-appropriate autonomy being explicitly linked to the resolution of the eating disorder.¹

The Maudsley model has been particularly effective in younger patients with a recent onset of AN. It has been more effective than individual therapy. For patients with an older age at onset, or a longer history of illness, neither treatment appears beneficial.¹

Cognitive behavioural therapy (CBT) is the most frequently tested individual treatment for AN. A CBT framework for conceptualising and treating AN has been described by Garner, Vitousek and Pike (1997).¹⁸ The recommended approach specifies one to two years of individual therapy for patients who begin treatment at low weight, and approximately one year for those who are weight restored.

Antidepressants – There is no empirical basis for the use of antidepressants in eating disorders. Fluoxetine is ineffective with low-weight patients,¹⁹ and initial indications that fluoxetine use might support maintenance of weight gain after inpatient treatment have not been confirmed.²⁰

Inpatient versus Outpatient Care – The National Institute for Clinical Excellence (NICE) eating disorders guidelines specify that most anorexic patients should be managed on an outpatient basis using psychological treatment.¹⁰ Indications for inpatient care include extremely low weight (75% or less of expected body weight), severe electrolyte imbalances, cardiac disturbances, severe or intractable purging, psychosis or a high risk of suicide.³ There are numerous arguments for minimising the use of inpatient treatment for AN. The benefits of more rapid weight gain must be balanced against the disadvantages of separation from familiar environment and increased identification with the disease.²¹ Qualitative studies reveal that AN patients often view inpatient treatment as demeaning and resent the loss of control over their condition – although most acknowledge simultaneously that enforced intervention can be life-saving in some cases. Another remarkable finding is that many of these patients report that exposure to 'thinner and more experienced patients' can have

clinical update

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deleterious effects, prompting competition to be the 'best anorexic' in the ward.¹

It is unfortunate that 5-10% of anorexic patients remain resistant to all forms of treatment, despite the best efforts made.¹ Small increases in weight as a result of inpatient treatment are not maintained once the person is discharged and the disease becomes a lifelong struggle for the anorexic and for those who care about them. Under these circumstances, it is important that the family or caregivers maintain realistic goals. The goal is more to stabilise the anorexic individual's health as best as possible rather than to 'cure' the anorexia.¹

Bulimia nervosa

The vast majority of BN patients can be treated as outpatients. In-patient or day patient treatment is usually only indicated in the event of risk of suicide or self-harm.¹⁰

Cognitive behavioural therapy – The NICE eating disorders guidelines recommend a manual-based CBT for the management of bulimia nervosa. This structured psychotherapy was developed by Fairburn, Marcus and Wilson,²² all opinion leaders in the area of eating disorders. The therapy is based on a cognitive model of the mechanisms that are thought to maintain bulimia nervosa and aims to enhance motivation for change, replace dysfunctional dieting with a regular and flexible pattern of eating, decrease concern regarding body shape and weight, and prevent relapse. Treatment typically consists of 16 to 20 sessions of individual therapy over four to five months. Group therapy has also been shown to be effective with bulimic patients.¹

CBT has been translated into guided self-help and self-help forms^{23,24} that can be applied in primary care and for which there is good evidence of efficacy in the Australian primary care setting.²⁵ It reduces the level of general psychiatric symptoms and improves self-esteem. Therapeutic improvement is reasonably well maintained at one-year follow-up.

Antidepressants – CBT is reportedly more effective than antidepressant medication, particularly in terms of halting binge-eating and purging. Evidence of long-term efficacy of antidepressant therapy in bulimic patients is generally lacking.¹ The only drug currently approved by the Food and Drug Administration (FDA) for the treatment of BN is fluoxetine (80mg per day).³ Desipramine (up to 300mg/day), imipramine (up to 300mg/day) and fluvoxamine also appear to have some efficacy in reducing the frequency of bingeing.²⁶

An electrocardiogram must be performed to determine whether hypokalaemia or palpitations are present in both AN and BN patients. This is essential to assess the safety of any planned psychopharmacologic management.³

Interpersonal psychotherapy assists patients to identify and modify current interpersonal problems that are hypothesised to be maintaining the eating disorder. The treatment is

nondirective and does not focus on the eating disorder itself.¹ Interpersonal psychotherapy is regarded as second-line therapy and appears to take longer to generate change than CBT.²⁷

Summary

Eating disorders are associated with serious medical complications, yet they often go undetected and untreated. Early identification and treatment heighten the chance of faster resolution of the condition, particularly in adolescents.

Open communication between health care professionals, families and the person with the eating disorder should always be encouraged. If families can share information and support with their anorexic relative and the professionals who are treating them, the likelihood of recovery is generally thought to be better.⁷

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References

1. Wilson GT, Grilo CM, Vitousek KM. Psychological Treatment of Eating Disorders. *American Psychologist* 2007;62:199-216.
2. Diagnostic and Statistical Manual of Mental Disorders, text revision (DSM-IV-TR®). 2000. ed. First. MR. 4th edition. Washington DC: American Psychiatric Publishing Inc.
3. Becker AE, Grinspoon S.K, Klibanski A, Herzog D.B. Eating Disorders. *NEJM* 1999 Apr 8;340:82-8.
4. Sullivan PF. Mortality in anorexia nervosa. *American Journal of Psychiatry* 1996;152:1073-4.
5. Kaye W. Neurobiology of anorexia and bulimia nervosa. *Physiology and Behaviour* 2009;94:121-35.
6. Shaw EM. The Dangers of Eating Disorders. *American Fitness* 2002. Jan/Feb;54-60.
7. RANZCP. 2005. Anorexia nervosa – Australian Treatment Guide for Consumers and Carers. Jun 2005. 1-48. At: www.ranzcp.org
8. Ben-Tovim DJ, Walker K, Gilchrist F, Freeman R, Kalucy R, Esterman A. Outcome in patients with eating disorders: A 5-year study. *Lancet* 2001;357:1254-7.
9. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and BSD in young women. *Arch Gen Psychiatry* 2003;57:659-65.
10. National Institute for Clinical Excellence (NICE). 2004. Eating Disorders: core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related disorders. Clinical Guideline number 9. London: NICE.
11. Grice DE, Halmi KA, Fichter MM, Strober M, Wood DB, Kaplan AS, et al. Evidence for a susceptibility gene for anorexia nervosa on chromosome 1. *Am J Hum Genet* 2002;70(3):787-92.
12. Anorexia, bulimia prove gene related. In *USA Today* 2006;17:7.
13. Birmingham CL, Beumont P. Medical management of eating disorders. Cambridge: Cambridge University Press, 2004.
14. Diller BM, Saxo V, Herzog DB, Rosenthal DI, Holzman S, Klibanski A. Mechanisms of osteoporosis in adult and adolescent women with anorexia nervosa. *J Clin Endocrinol Metab* 1999;89:548-54.
15. Bachrach LK, Guio D, Katzmann D, Litt IF, Marcus R. Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics* 1990;85:440-47.
16. Ders C, Eisler I. Family therapy for anorexia nervosa. In Garner D & Garfinkel PE (Eds.), *Handbook of treatment for eating disorders* (2nd ed., 2007:333-48). Chichester, England: Wiley.
17. Lock J, Le Grange D. Family-based treatment of eating disorders. *International Journal of Eating Disorders* 2005;37(Suppl):S64-S67.
18. Garner DM, Vitousek K, Pike KM. Cognitive behavioural therapy for anorexia nervosa. In Garner D & Garfinkel PE (Eds.), *Handbook of treatment for eating disorders* (2nd ed., 1997:91-144). Chichester, England: Wiley.
19. Attia E, Haiman C, Walsh BT, Flier SR. Does fluoxetine augment the inpatient treatment of anorexia nervosa? *American Journal of Psychiatry* 1996;155:548-51.
20. Kaye WH, Nagata T, Weltzin TE, Hsu LK, Sakol MS, McConaha C, et al. Double-blind placebo-controlled administration of fluoxetine in restricting- and restricting-purging-type anorexia nervosa. *Biological Psychiatry* 2001;49:544-52.
21. Vitousek KM, Gray JA. Outpatient management of anorexia nervosa. In P.J. Cooper & A. Stein (Eds.), *Childhood feeding problems and adolescent eating disorders*. (2006:247-90). Chur, Switzerland: Harward Academic.
22. Fairburn CG, Marcus MD, Wilson GT. Cognitive behaviour therapy for binge eating and bulimia nervosa: A comprehensive treatment manual. In Fairburn CG & Wilson GT (Eds.), *Binge eating. Nature, assessment and treatment* (1993:361-404). New York: Guilford Press.
23. Stafeno SC, Becaltichuk J, Bay SL, Hay P. Self help treatments for disorders of recurrent binge-eating: a systematic review. *Acta Psychiatrica Scand* 2008;113:462-9.
24. Perkins SJ, Murphy R, Schmidt L, Williams C. Self help and guided self help for eating disorders. *Cochrane Database Syst Rev* 2006;3:CD004191.
25. Baresiak S, Paxton SJ, Hay PJ. Guided self help for bulimia nervosa in primary care: a randomised controlled trial. *Psychol Med* 2005;35:1283-94.
26. Becker AE, Hamburg P, Herzog DB. 1998. The role of psychopharmacological management in the treatment of eating disorders. In: Dunner DL, Rosenbaum JR (Eds.) *Journal of drug therapy*. Philadelphia: WB Saunders.
27. Hay PJ. Understanding bulimia. *Aust Fam Phys* 2007;36:708-12.

CPD questions on pages 664