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Leading article

Bulimia nervosa and a stepped care approach to management

Bulimia nervosa is a common psychiatric disorder which may present to a variety of specialists including the gastro-enterologist. Often the disorder goes unrecognised for some considerable time, in part because these patients have difficulty in admitting to the problem. This paper describes the disorder, discusses the issue of detection from the perspective of the gastroenterologist, and outlines a 'stepped care' approach to management.

It is only 11 years since the first formal description of bulimia nervosa was published. In this seminal paper Russell drew attention to 'an ominous variant of anorexia nervosa,' a disorder which closely resembled anorexia nervosa except that body weight was in most cases within the normal range. Since then there has been intense clinical and research interest in bulimia nervosa, stimulated by the large number of patients presenting for treatment. From epidemiological studies it is clear that bulimia nervosa is more common than anorexia nervosa, with a prevalence among young women in the region of 1%. The disorder is uncommon in men. It is as yet uncertain how best to provide treatment for the many women who have this condition, a stepped care approach being one solution.

Three features are required to make the diagnosis of bulimia nervosa. The first is loss of control over eating associated with recurrent episodes of overeating (bulimia). These episodes (often referred to as binges) mostly occur in secret and are a source of shame and self disgust. The food eaten typically consists of energy-rich items which the patients are trying to exclude from their diet, the total calorie intake being of the order of 3500 to 5000 kcals.³⁻⁵ Some patients eat larger amounts and there have been several reports of acute gastric dilatation and rupture.⁶⁻⁹ The frequency of bulimic episodes varies greatly: they may occur many times each day or they may be only occasional. Between the episodes of bulimia most, but not all, patients do their best to restrict their food intake.

The second diagnostic feature is extreme behaviour intended to control shape and weight. The behaviour patterns include self induced vomiting, severe dieting, the misuse of purgatives or diuretics, and, in a minority of patients, vigorous exercise. Often these patterns of behaviour occur in combination and, like the overeating, their frequency varies.

The third diagnostic feature is a characteristic set of attitudes to shape and weight, which resemble those in anorexia nervosa. These attitudes have been described as a morbid fear of becoming fat¹ or, more recently, as a persistent overconcern with shape and weight. The essence of this core psychopathology (as it has been termed) is that the patients judge their self worth largely, or even exclusively, in terms of their appearance or weight. As a result they do their utmost to avoid weight gain or fatness, and some strive to be thin. Most features of bulimia nervosa are secondary to these attitudes.

In addition to these three features which define the disorder many others may be present, the most common being depressive and anxiety symptoms.¹³ The patients' social function is usually impaired, and there is a subgroup of patients who have other associated problems – for example, the abuse of alcohol or drugs.¹⁴

The physical features of bulimia nervosa are variable. Few abnormalities are present on examination, and body weight is usually within the normal range. In a small proportion of cases there is pronounced hypertrophy of the salivary glands, particularly the parotids. ¹⁵⁻¹⁷ This swelling is usually bilateral and painless. Its pathophysiology is unclear. Patients who have vomited frequently for some years have erosions of the dental enamel, especially of the lingual, palatal, and posterior occlusal surfaces of the teeth. ¹⁸⁻²¹ This is especially prominent on the inner surface of the upper teeth. In these areas fillings tend to be proud of the surface of the enamel. If patients have repeatedly used their fingers to stimulate the gag reflex they may have a characteristic distribution of calluses on the dorsum of the hand (Russell's sign). ¹

In about half the patients there is electrolyte disturbance, the most common abnormalities being hypochloraemia, hypokalaemia, hyponatraemia, and a raised bicarbonate concentration.22 Very occasionally these abnormalities are life threatening. The electrolyte disturbance is a consequence of self induced vomiting and the misuse of purgatives or diuretics, and its nature depends on which behaviour predominates. Raised serum amylase levels are found occasionally, 23-26 which are caused by an increase in the salivary isoenzyme. There have been two studies of gastric emptying. In one, more than half the patients had delayed emptying, 27 but in the other gastric emptying was normal.28 Studies of taste, 29 30 satiety, 31 32 and cholecystokinin secretion 33 have detected abnormalities, but these have yet to be replicated and their importance is uncertain. They are probably secondary effects of the disorder, although they may perpetuate it. The endocrine state of these patients has not been extensively studied. Some changes are secondary to 'starvation.'34 35 Menstrual irregularities are common, even among those patients whose weight is in the normal range, 13 36-38 but in most cases regular menstruation returns once healthy eating habits have been restored.39 With the exception of severe electrolyte disturbance, the physical abnormalities associated with bulimia nervosa rarely merit treatment in their own right.

The aetiology of bulimia nervosa is poorly understood. It often starts as normal adolescent dieting, which becomes progressively more extreme. As a result body weight falls, and in one third to one half of cases diagnostic criteria for anorexia nervosa are eventually met. Then, control over eating breaks down and the extreme dieting becomes punctuated by episodes of overeating. Vomiting, purgatives, and diuretics may be used in an attempt to minimise the effect on weight of the overeating, but eating habits worsen and the lost weight is regained. Gradually the eating disorder becomes entrenched and, after a variable length of time, treatment may be sought. Typically patients are in their mid-20s by the time they present for help.

While it is becoming commoner for patients with bulimia nervosa to seek treatment for their eating disorder, this is often still only after many years' delay. In the intervening period they may present to general practitioners with a variety of symptoms and be referred on for specialist help. Psychiatrists may be asked to advise on depressive and anxiety symptoms, gynaecologists may be consulted about menstrual irregularities, and gastroenterologists may be consulted on account of gastrointestinal symptoms. In each instance the eating disorder may go undisclosed and unrecognised.

In view of the prevalence of bulimia nervosa it is appropriate for doctors to maintain a high index of suspicion of bulimia nervosa when apparently healthy young women of normal weight present with unexplained symptoms. Patients presenting to gastroenterology clinics may describe abdominal fullness or pain or feelings of being bloated, or they may complain of facial swelling. Most do not mention either the overeating or self induced vomiting, and it is also uncommon for them to complain of laxative induced diarrhoea. In our experience, however, some patients complain of recurrent episodes of spontaneous vomiting.

Detection is possible by asking patients in a sensitive manner about their eating habits, and in particular about their control over eating. It is important to emphasise that these women are deeply ashamed of their behaviour and may find it difficult to divulge the problem in a busy outpatient clinic. Physical signs or laboratory abnormalities rarely help the diagnosis.

Two forms of treatment have received particular attention. A series of controlled trials has shown that antidepressant drugs are more effective than placebo in reducing the frequency of overeating and the intensity of some of the other features of the disorder. 40-42 There is no evidence, however, that they affect the patients' disturbed attitudes to shape and weight or their extreme attempts to diet. 43 44 This may explain why there is a tendency for the disorder to relapse even if these drugs are continued.45

The main alternative approach is a specific short term psychological treatment, a form of cognitive behaviour therapy. This treatment is designed not just to change the eating habits of these patients but also to modify their disturbed attitudes to shape and weight.46 It has been evaluated in a series of controlled trials, and the immediate effects of treatment compare favourably with those obtained with antidepressant drugs. 47 48 Significantly, the changes are maintained for at least the first year after treatment. This treatment, however, his two limitations: firstly, it is time consuming, requiring about 20 treatment sessions over five months; and secondly, it requires specialist training. Simpler forms of treatment are needed. Less intensive treatment has been advocated including various forms of group therapy and dietary advice,49 and the use of self help manuals.50 None of these approaches has been subjected to rigorous evaluation, but it seems likely that at least some patients will respond to simpler interventions of this type.

Since it is not yet possible to predict which patients will respond to which form of treatment, it seems sensible to adopt a 'stepped care' approach. At least five 'levels' of intervention can be envisaged. The first level is some form of self help using written material. The second is dietary education and advice, perhaps provided in a group setting. The third is antidepressant drug treatment in combination with advice and support, the drug of choice being either desipramine or fluoxetine, since both have been extensively used in these patients and are well tolerated. The fourth is outpatient psychological treatment on an individual basis, with cognitive behaviour therapy being the approach of choice. The final level is a period of day patient or inpatient care, and subsequent outpatient treatment.

Until a stepped care approach of this type is of proven value, how should these patients be managed? Undoubtedly the initial goal should be to help patients admit to the problem. Disclosing what has been a guilt ridden secret for many years is a major advance, and one which will give the clinician the opportunity to educate and advise the patient. Thereafter, for cases in which the disorder does not seem unduly entrenched or complex, some of the simpler behavioural strategies contained in the established cognitive behavioural treatment might be used. 42 51 In theory this should be possible in an outpatient clinic. Alternatively, referral to a dietitian might be considered, although many dietitians are often not well trained to deal with disorders of this type. In longstanding cases and those in which other problems are present the patient should be referred to a psychiatrist or clinical psychologist, preferably with a special interest in the treatment of eating disorders.

> **CG FAIRBURN** R C PEVELER

Oxford University, Department of Psychiatry, Warneford Hospital, Oxford OX3 77X

Correspondence to: Dr Christopher Fairburn.

- Russell GFM. Bulimia nervosa: an ominous variant of anorexia nervosa. Psychol Med 1979; 9: 429-48.
 Fairburn CG, Beglin SJ. Studies of the epidemiology of bulimia nervosa. Am J Psychiatry 1990; 147: 401-8.
 Mitchell JE, Laine DC. Monitored binge-eating behavior in patients with bulimia. Int J Eating Disorders 1985; 4: 177-83.
 Kissileff HR, Walsh BT, Kral JG, Cassidy SM. Laboratory studies of eating behavior in women with bulimia. Physiol Behav 1986; 38: 563-70.
 Kaye WH, Gwirtsman HE, George DT, Weiss SR, Jimerson DC. Relationship of mood alterations to bingeing behaviour in bulimia. Br J Psychiatry 1986:

- of mood alterations to bingeing behaviour in bulimia. Br J Psychiatry 1986;
- 6 Saul SH, Dekker A, Watson CG. Acute gastric dilatation with infarction and

- Saul SH, Dekker A, Watson CG. Acute gastric dilatation with infarction and perforation. Gut 1981; 22: 978-83.
 Mitchell JE, Pyle RL, Miner RA. Gastric dilatation as a complication of bulimia. Psychosomatics 1982; 23: 96-7.
 Breslow M, Yates A, Shisslak C. Spontaneous rupture of the stomach: a complication of bulimia. Int J Eating Disorders 1986; 5: 137-42.
 Abdu RA, Garritano D, Culver O. Acute gastric necrosis in anorexia nervosa and bulimia. Arch Surg 1987; 122: 830-2.
 American Psychiatric Association. DSM-III-R. Washington DC: American Psychiatric Association, 1987.
 Fairburn CG, Garner DM. Diagnostic criteria for anorexia nervosa and bulimia nervosa: the importance of attitudes to shape and weight. In: Garner DM, Garfinkel PE. eds. Diagnostic issues in anorexia nervosa and bulimia nervosa. Garfinkel PE, eds. Diagnostic issues in anorexia nervosa and bulimia nervosa. New York: Brunner/Mazel, 1988: 36-55.
- New York: Brunner/Mazel, 1988: 36-95.
 12 Fairburn CG. The uncertain status of the cognitive approach to bulimia nervosa. In: Pirke KM, Vandereycken W, Ploog D, eds. The psychobiology of bulimia nervosa. Berlin: Springer-Verlag, 1988: 129-36.
 13 Fairburn CG, Cooper PJ. The clinical features of bulimia nervosa. Br J Psychiatry 1984; 144: 238-46.
 14 Lacey JH, Evans CDH. The impulsivist: a multi-impulsive personality disorder. Br J Addict 1986; 81: 641-9.
 15 Levin PA, Falko JM, Dixon K, Gallup EM, Saunders W. Benign parotid enlargement in bulimia. Ann Intern Med 1980; 93: 827-9.
 16 Brady JP. Parotid enlargement in bulimia. J Fam Pract 1985; 20: 496-502.
 17 Burke RC. Bulimia and parotid enlargement case report and treatment.

- Brady JP. Parotid enlargement in bulimia. J Fam Pract 1985; 20: 496-502.
 Burke RC. Bulimia and parotid enlargement case report and treatment. J Otolarymgol 1986; 15: 49-51.
 Wolcott RB, Yager J, Gordon G. Dental sequelae to the binge-purge syndrome (bulimia): report of cases. J Am Dental Assoc 1984; 109: 723-5.
 Simmons MS, Grayden SK, Mitchell JE. The need for psychiatric-dental liaison in the treatment of bulimia. Am J Psychiatry 1986; 143: 783-4.
 Roberts MW, Li S-H. Oral findings in anorexia nervosa and bulimia nervosa: a study of 47 cases. J Am Dental Assoc 1987; 115: 407-10.
 Altshuler BD, Dechow PC, Waller DA, Hardy BW. An investigation of the oral pathologies occurring in bulimia nervosa. Int J Eating Disorders 1990; 9: 191-9.
 Mitchell JE, Pyle RL, Eckert ED, Hatsukami D, Lentz R, Electrolyte and
- 22 Mitchell JE, Pyle RL, Eckert ED, Hatsukami D, Lentz R. Electrolyte and other physiological abnormalities in patients with bulimia. *Psychol Med* 1983; 13: 273-8.
- 13: 273-6.
 23 Gwirtsman HE, Yager J, Gillard BK, Lerner L. Serum amylase and its isoenzymes in normal weight bulimia. Int J Eating Disorders 1986; 5: 355-61.
 24 Humphries LL, Adams LJ, Eckfeldt JH, Levitt MD, McClain CJ. Hyperamylasemia in patients with eating disorders. Ann Intern Med 1987; 106:
- 50-2
- 25 Kaplan AS. Hyperamylasemia and bulimia: a clinical review. Int J Eating Disorders 1987; 6: 537-43.
- 23 Aspian AS. Hyperamylasemia and buildina: a Chilical review. Int J Bating Disorders 1987; 6: 537-43.
 26 Gwirtsman HE, Kaye WH, George DT, Carosella NW, Greene RC, Jimerson DC. Hyperamylasemia and its relationship to binge-purge episodes: development of a clinically relevant laboratory test. J Clin Psychiatry 1989; 50: 196-202.
- 204.
 27 Humphries L, Shih W-J. Gastric emptying time in anorexia nervosa and bulimia. Arch Surg 1988; 123: 783.
 28 Robinson PH, Clarke M, Barrett J. Determinants of delayed gastric emptying in anorexia nervosa and bulimia nervosa. Gut 1988; 29: 458-64.
 29 Drewnowski A, Halmi K, Pierce B. Taste and eating disorders. Am J Clin Nutr 1987; 46: 442-50.
- 30 Rodin J, Bartoshuk L, Peterson C, Schank D. Bulimia and taste: possible interactions. J Abnorm Psychol 1990; 99: 32-9.
 31 Owen W, Halmi K, Gibbs J, Smith G. Satiety responses in eating disorders. J Psychiatr Res 1985; 19: 279-84.
- J Psychiatr Res 1985; 19: 279-84.
 Chiodo J, Latimer P. Hunger perceptions and satiety responses among normal-weight bulimics and normals to a high calorie, carbohydrate rich food. Psychol Med 1986; 16: 343-9.
 Geracioti TD, Liddle RA. Impaired cholecystokinin secretion in bulimia nervosa. N Engl J Med 1988; 319: 683-8.
 Levy AB. Neuroendocrine profile in bulimia nervosa. Biological Psychiatry 1989; 25: 98-109.
 Ficher MM. Fields VM. Endocrine deficiency in the control of the cont

- 35 Fichter MM, Pirke KM. Endocrine dysfunctions in bulimia nervosa. In: Fichter MM, ed. Bulimia nervosa: Basic research, diagnosis and therapy. Chichester: Wiley, 1990: 235-57.

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- 36 Copeland PM, Herzog DB. Menstrual abnormalities. In: Hudson JI, Pope HG, eds. The psychobiology of bulimia. Washington DC: American Psychiatric Press, 1987: 31-54.
- Press, 1987: 31-54.

 37 Cantopher T, Evans C, Lacey JH, Pearce JM. Menstrual and ovulatory disturbance in bulimia. Br Med J 1988; 297: 836-7.

 38 Pirke KM. Menstrual cycle and neuroendocrine disturbances of the gonadal axis in bulimia nervosa. In: Fichter MM, ed. Bulimia nervosa: Basic research, diagnosis and therapy. Chichester: Wiley, 1990: 258-69.

 39 Fairburn CG, Kirk J, O'Connor M, Cooper PJ. A comparison of two psychological treatments for bulimia nervosa. Behav Res Ther 1986; 24: 629-43.
- 40 Walsh BT. Antidepressants and bulimia: where are we? Int J Eating Disorders
- 40 Walsh BT. Antidepressants and bulimia: where are we? Int J Eating Disorders 1988; 7: 421-3.
 41 Agras WS, McCann U. The efficacy and role of antidepressants in the treatment of bulimia nervosa. Annals of Behavioral Medicine 1987; 9: 18-22.
 42 Mitchell JE. Bulimia nervosa. Minneapolis: University of Minnesota Press,
- 43 Rossiter EM, Agras WS, Losch M. Changes in self-reported food intake in bulimics as a consequence of antidepressant treatment. *Int J Eating Disorders* 1988; 7: 779–83.

- 44 Mitchell JE, Fletcher L, Pyle RL, Eckert ED, Hatsukami DK, Pomeroy C.
- 44 Mitcheil JE, Fletcher L, Fyle RL, Eckert ED, Hatsukami DK, Fomeroy C.
 The impact of treatment on meal patterns in patients with bulimia nervosa. Int J Eating Disorders 1989; 8: 167-72.

 45 Pope HG, Hudson JI, Jonas JM, Yurgelun-Todd D. Antidepressant treatment of bulimia: a two-year follow-up study. J Clin Psychopharmacol 1985; 5: 220. 320-7.

- of Dullmila: a two-year follow-up study. J Chin I sychophamical 220-7.
 46 Fairburn CG. A cognitive behavioural approach to the management of bulimia. Psychol Med 1981; 11: 707-11.
 47 Wilson GT, Smith D. Cognitive-behavioral treatment of bulimia nervosa. Annals of Behavioral Medicine 1987; 9: 12-7.
 48 Fairburn CG. The current status of the psychological treatments for bulimia nervosa. J Psychosom Res 1988; 32: 635-45.
 49 Beaumont PJV, O'Connor M, Lennerts W, Touyz SW. Nutritional counselling in the treatment of bulimia. In: Fichter MM, ed. Bulimia nervosa: Basic research, diagnosis and therapy. Chichester: Wiley, 1990: 308-19.
 50 Huon GF. An initial validation of a self-help program for bulimia. Int J Eating Disorders 1985; 4: 573-88.
 51 Fairburn CG. Cognitive-behavioral treatment for bulimia. In: Garner DM, Garfinkel PE, eds. Handbook of psychotherapy for anorexia nervosa and bulimia. New York: Guildford Press, 1985: 160-92.