Office of Health Economics

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INTRODUCTION

‘On a planet where millions die of starvation or the complications of malnutrition each year, it is a tragic paradox that in some of the world’s great agricultural heartlands... individuals are damaging and even killing themselves by eating too much, or too little, or alternating between these unhealthy behaviours in a pathological way’ (West, 1987).

This report is concerned with people who persistently starve themselves (anorexia nervosa), as well as those who follow chaotic eating patterns (bulimia nervosa). Both conditions are associated with considerable morbidity and mortality.

The medical consensus is that fundamentally both have a psychological causation, being often the outward expression of deep psychological and emotional turmoil. Social pressures in society towards thinness may encourage those with emotional problems to turn to anorexia nervosa (although not consciously) as a coping mechanism or outlet for these difficulties; without this social emphasis, it is possible that anorexia nervosa sufferers might have found another, less physically harmful outlet. Bulimia nervosa is a related eating disorder, in which sufferers share the morbid fear of fatness, but binge-eat, and often purge themselves by vomiting or using laxatives to prevent weight gain. The term bulimia nervosa is also applied to cases of extreme dieting, consisting of abstinence from food followed by binge-eating, but without involving purging as well.

Anorexia nervosa and bulimia nervosa have received considerable publicity in recent years. These disorders usually affect young women in their teens to mid-twenties, though males can also suffer. While on the surface the issue appears to be about food, for most sufferers eating disorders express a fundamental unhappiness, which may originate from a number of different sources. It is believed that by concentrating all their energies around food and eating, sufferers can avoid facing other, more painful, issues in their lives.

Anorexia nervosa literally means ‘loss of appetite for nervous reasons’. However, the term is rather misleading, since sufferers have not lost their appetite; they simply no longer have the ability to allow themselves to satisfy it. The disorder is characterised by the obsessive pursuit of a thin body, achieved through self-starvation. People with anorexia nervosa often pass through a phase akin to mental elation, but as food consumption falls and weight drops, they start to suffer all the medical effects of starvation. They become obsessed with calories, thinking about food all the time. At the same
time, they often lack confidence, have very low self-esteem, and are troubled by feelings of worthlessness. They also find it very difficult, even frightening, to acknowledge that they are ill, so that many will deny that they have a problem, even when it is obvious to all around them. Some of the symptoms of anorexia nervosa generate other symptoms and distortions in the patient's life. A sufferer may withdraw socially, in part due to starvation and in part due to lowered feelings of self-worth; this in turn results in other problems, such as isolation and loneliness.

Serious medical complications can result from anorexia nervosa. Gastrointestinal symptoms such as constipation, fullness after eating, bloatedness and vague abdominal pains are often present, as is a heightened sensitivity to cold. Blood pressure and pulse will be low and there may be dependent oedema. Hypercholesterolaemia is often present and life threatening hypoglycaemia very occasionally occurs. In longstanding cases osteopaenia and osteoporotic fractures are sometimes evident, secondary to oestrogen deficiency and low weight. The effects of starvation in chronic cases may, of course, lead to death.

Bulimia nervosa is characterised by compulsive binge-eating, accompanied by self-induced vomiting, periods of starvation, and purging with laxatives (to alleviate guilt and also to reduce the chances of gaining weight). Whilst the normal food intake for an adult is 2,000-3,000 calories per day, a sufferer may consume 15,000 calories in a matter of one or two hours during a binge (Farley, 1992). A 'binge' is what happens when eating becomes wholly out of control: immense quantities of food are consumed in a frenzy, food even being eaten uncooked or straight from the freezer! Bingeing episodes tend to be carried out in secret, are sometimes pre-planned, and are often followed by guilt, depression, and even suicidal behaviour. Bulimia nervosa is a condition which increasingly controls the sufferer's life: whilst people with bulimia nervosa may be sociable by nature, they are forced into leading increasingly private lives, to avoid the possibility of being found out. Feelings of shame and isolation add to those of inadequacy and unattractiveness.

Although the medical complications arising from bulimia nervosa are usually less severe than those associated with anorexia nervosa, they can be serious. Ulcers, stomach and bowel disorders, mouth and throat irritation, and gum disease are all associated with bulimia nervosa, caused in the main by vomiting. Constant purging and vomiting creates severe mineral imbalances in the body, as the delicate balance of salts becomes deranged. The correct balance of mineral salts is vital for the proper functioning of organs such as the
heart and kidneys. Fatigue or complete exhaustion are likely to follow, with, in the most severe cases, stupor, coma, and possibly death.

Whilst anorexia nervosa has been recognised for decades, bulimia nervosa was identified as a separate medical condition much more recently (Russell, 1979). The typical symptoms and characteristics of sufferers of these disorders are listed in Table 1.

A further classification has recently emerged, known as 'binge-eating disorder', to describe people who binge but do not take any steps to compensate for this overeating (e.g. by vomiting or using laxatives) to avoid weight gain. Its importance should not be trivialised, although relatively little is known about the characteristics and causes of this disorder at the time of writing and

Table 1 Some common symptoms and characteristics of eating disorder patients

<table>
<thead>
<tr>
<th>ANOREXIA NERVOSA</th>
<th>BULIMIA NERVOSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe weight loss</td>
<td>Binge eating and vomiting or purging or extreme dieting</td>
</tr>
<tr>
<td>Distortions and misconceptions about weight and body size</td>
<td>Distortions and misconceptions about weight and body size</td>
</tr>
<tr>
<td>Perfectionism, obsessionality and frigidity</td>
<td>Sore throat and erosion of teeth enamel due to vomiting</td>
</tr>
<tr>
<td>Feeling cold, poor circulation</td>
<td>Menstrual disturbances</td>
</tr>
<tr>
<td>Loss of menstrual periods</td>
<td>Emotional instability and mood swings</td>
</tr>
<tr>
<td>Irritability, emotional instability, poor concentration</td>
<td>Lethargy</td>
</tr>
<tr>
<td>Difficulty sleeping</td>
<td>Devious behaviour; increasingly reclusive/secretive</td>
</tr>
<tr>
<td>Vomiting and/or purging</td>
<td>Feeling out of control, helpless, and lonely</td>
</tr>
<tr>
<td>Devious behaviour</td>
<td>Disappearing after meals to get rid of food</td>
</tr>
<tr>
<td>Low self-esteem, social withdrawal (isolation and loneliness)</td>
<td>Dehydration and poor skin condition</td>
</tr>
<tr>
<td>Excessive exercising</td>
<td>Tension at home due to food shortages caused by bingeing</td>
</tr>
<tr>
<td>Growth of downy hair mainly on arms, trunk and face</td>
<td></td>
</tr>
</tbody>
</table>

Source: Adapted from the Eating Disorders Association, Information Pack.
this paper does not consider this phenomenon further. It is believed, however, to be associated with impairment in work and social functioning, general psychopathology, a history of drug/alcohol abuse, severe obesity, and treatment for emotional problems (Spitzer et al, 1993).

**BOX 1 Definitions of Anorexia and Bulimia Nervosa**

Russell produced sets of criteria for anorexia nervosa in 1970 and for bulimia nervosa in 1979. These led to a considerable degree of consensus concerning diagnosis among clinicians. However, Russell's criteria have largely been superseded by the DSM-III-R criteria of the American Psychiatric Association.

**Diagnostic criteria for anorexia nervosa**

1. Refusal to maintain body weight over a minimal normal weight for age and height, e.g. weight loss leading to maintenance of body weight 15 per cent below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15 per cent below that expected.
2. Intense fear of gaining weight or becoming fat, even though underweight.
3. Disturbance in body image, e.g. the person claims to 'feel fat' even when emaciated or believes one area of the body to be 'too fat' even when obviously underweight.
4. In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur.

**Diagnostic criteria for bulimia nervosa**

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time).
2. A feeling of lack of control over eating behaviour during the eating binges.
3. The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.
4. A minimum average of two binge eating episodes a week for at least three months.
5. Persistent overconcern with body shape and weight.

Some studies have made reference to a partial syndrome of anorexia and bulimia nervosa where some, but not all, of the aforementioned criteria are present in the sufferer. Whilst some form of illness may be clearly apparent in such people, there are diagnostic and definitional problems when one attempts to move away from the 'full' syndrome.
Anorexia and bulimia nervosa are important areas of study not only because of the morbidity and mortality associated with them, but also due to the growing reported incidence of these conditions in the population. Agras (1984) reported that over the previous 30 years, reported cases of anorexia nervosa had been doubling every decade. Pope et al (1983) claimed that whereas prior to 1980 few patients would have met the DSM-III criteria (see Box 1) for bulimia nervosa, more recently, 8-20 per cent of high school and college students in America were found to meet these criteria.

This paper will attempt to highlight several important aspects of these eating disorders. Chapter two outlines how understanding of the two conditions has developed over time; chapter three discusses their incidence and prevalence; chapter four looks at theories of causation; chapter five examines treatment options and evidence of their effectiveness; chapter six seeks to identify costs to the NHS of these conditions, and chapter seven sets out our conclusions.
HISTORY

Testimony exists to suggest that anorexia nervosa, far from being a twentieth-century phenomena, has been evident for several centuries.

John Reynolds (1669), physician and minister, wrote the following account of one Martha Taylor: 'she began to abstain from all solid food... as also from all other sorts both of meat and drinks, except now and then a few drops of syrup and stew'd prunes, water and sugar, or the juice of a roasted raisin, but these repasts are used so seldom, and in such small quantities as are prodigiously insufficient for sustenation... and though her upper parts be less emaciated (though too much) yet her lower parts are very languid and inept for motion...’ A similar tale was told by the eminent English philosopher Thomas Hobbes in 1688, when he wrote to a friend describing a young lady as follows: 'part of her belly touches her backbone. She began to lose her appetite in December last, and tis thought that she cannot last much longer'.

An early account of what may have been anorexia nervosa is credited to Richard Morton, who related two relevant case histories in ‘Phthsiologica – or a Treatise on Consumption’ (1694). The first, dating from 1684, concerned an 18 year-old girl who was 'like a skeleton clad only with skin'. She had no appetite and eventually died after a fainting fit. The second case related to a minister’s son aged 16 who ‘fell gradually into total want of Appetite’. However, he appeared to recover his health following Morton’s orders to 'abandon his studies, to go into the country air, and to use riding, and a milk diet... for a long time'. Morton concluded that 'nervous consumption' was ‘attended by a want of appetite... At first it flatters and deceives the patient, for which reason it happens for the most part that the physician is consulted too late’.

The emergence of anorexia nervosa as a recognised clinical condition dates from reports by Lasegue in Paris and Gull in England (Guy’s Hospital) which appeared almost simultaneously. In 1873, Gull used the term ‘anorexia nervosa’ to describe a condition which he said usually affected females aged 16-23 years, although it was occasionally encountered in males. He noted symptoms such as amenorrhoea, constipation, loss of appetite, slow pulse, slow respiration, emaciation, oedema of the lower extremities, and hypothermia but also stated that no physical cause could be identified. Lasegue’s article, also published in 1873, went under the title ‘De l’anorexie hysterique’. The accounts of Gull and Lasegue differed in their nature, with Gull precise and direct and
Lasegue conveying a sense of the feelings of these people, the nuances of their disturbed relationships, and the subtleties of their intrapsychic turmoil.

Lasegue identified anorexia nervosa as affecting girls aged 15-20 years, who suffered some emotion which was avowed or concealed; this was said to be generally related to some real or imagined marital project. He believed that the strangest element of this illness was the patient’s contentment with their condition, so that no desire was felt for recovery. He described how gradually, the sufferer would reduce her food intake further and further, while furnishing pretexts for doing so. After a few weeks, she would no longer feel the need for an excuse for not eating, arguing that she had sufficient nourishment. Lasegue saw the excuse ‘I cannot eat, I am unwell’ being replaced by ‘I do not suffer and therefore must be well’, and he regarded the whole illness as summed up in this ‘intellectual perversion’. He described the illness as reaching a stage where extreme emaciation occurs, general debility increases, and exercise becomes laborious, but thought that the prognosis was good if the patient could be separated from her family.

Lasegue’s paper led to much debate on the subject in France, with Deniau (1883) proposing the term ‘anorexie mentale’ as a more appropriate name for the condition and this term has persisted in French literature to this day. Gull was already a prominent figure in British medicine before his work on anorexia nervosa appeared, and it is likely that had a less eminent figure written the piece, it would not have gained as much attention. There was also an awareness of anorexia nervosa in Italy (Brugnoli, 1875) and America (Hammond, 1879) during this period.

The next important contribution to the history of anorexia nervosa came from Simmonds, a pathologist, based in Hamburg. In 1914, he produced the first of several reports concerning hypophyseal insufficiency in man. ‘Although a syndrome of pituitary insufficiency would seem to be, and is, quite unrelated to anorexia nervosa, the two became inextricably associated in medical thinking and writing as a result of an erroneous emphasis upon weight loss’ (Bliss and Branch, 1960). Simmond’s work revived waning interest in anorexia nervosa, but also caused considerable confusion. Cases of Simmond’s Disease were reported which in retrospect, were almost certainly anorexia nervosa. Whilst he had

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1 The hypophysis, or pituitary stalk, connects the pituitary gland to the hypothalamus of the brain.
not intended his work to be regarded as a theory of anorexia nervosa, many people mistakenly considered it to be the first questioning of the views of Gull and Lasegue that the cause of anorexia nervosa was psychological. As a result it was suggested that anorexia nervosa may be an endocrinological disorder with incidental psychological features. In fact, the term 'anorexia nervosa' all but disappeared from the literature after this being replaced with references to 'Simmonds' Disease'.

It was not until 1930 that Berkman, describing 117 cases, re-established anorexia nervosa as a clinical entity; he viewed it as a physiological illness with secondary psychological disturbance. However, primary psychological causation was strongly espoused by Venables (1930) and this view was supported by Ryle, who published two papers in the late 1930s on anorexia nervosa which denied that his patients had any form of 'Simmond's Disease'. He stated that he hoped to show, 'some striking physical symptoms notwithstanding, that the origins of the disease are, as Gull maintained, to be sought in a disturbance of the mind and a prolonged insufficiency of food and nothing more'.

In the post-war period, awareness of anorexia nervosa steadily increased, and there must now be few members of the public who would not have some appreciation of it and of bulimia nervosa. However, bulimia nervosa was recognised much later. It was not until Russell (1979) published an article entitled 'Bulimia nervosa: an ominous variant of anorexia nervosa' that the condition really came to general medical notice. Nevertheless, Parry-Jones and Parry-Jones (1991) examining printed works dating from the fifteenth century concluded that, 'for centuries, the symptom of bulimia has been occurring in conjunction with a variety of other symptoms and signs'. They argue that the term 'bulimia' has a venerable history. Usage can be traced to ancient Greece, derived from the Greek bous, meaning ox, and limos, hunger, it denoted hunger severe enough for a person to eat a whole ox. The Oxford English Dictionary, in 1651, included the term 'bulimy', referring to a state of insatiability or dog-like appetite. During the 1980s, bulimia nervosa became widely publicised, not least because of articles and books claiming that the Princess of Wales had suffered from the disorder.

The next section of the report examines whether there has been an increasing prevalence of anorexia and bulimia nervosa in recent years.
Table 2 shows the results of studies of incidence rates. These vary widely, ranging from 0.08 per 100,000 per year in Sweden to 8.1 per 100,000 in Holland. Such variations are in part explained by differences in methods of ascertainment and in the diagnostic criteria employed.

The evidence suggests that there has been an increasing incidence in anorexia nervosa over time. Jones et al (1980) in the USA found that the number of diagnosed cases almost doubled from 0.35 (1960-69) to 0.64 per 100,000 (1970-76). This increase occurred solely in the

<table>
<thead>
<tr>
<th>COUNTRY/AUTHOR</th>
<th>ASCERTAINMENT METHOD</th>
<th>YEAR</th>
<th>INCIDENCE PER 100,000 PER YEAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweden Theander (1970)</td>
<td>Hospital records</td>
<td>1931-1940</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1951-1960</td>
<td>0.45</td>
</tr>
<tr>
<td>England Kendall et al (1973)</td>
<td>Case register</td>
<td>1965-1971</td>
<td>0.66</td>
</tr>
<tr>
<td>USA Kendall et al (1973)</td>
<td>Case register</td>
<td>1960-1969</td>
<td>0.37</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1970-1976</td>
<td>0.64</td>
</tr>
<tr>
<td>Switzerland Willi &amp; Grossman (1983)</td>
<td>Hospital records</td>
<td>1956-1958</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1963-1965</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1973-1975</td>
<td>1.12</td>
</tr>
<tr>
<td>England/Wales Williams &amp; King (1987)</td>
<td>Hospital records</td>
<td>1972-1981</td>
<td>1.4-2.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1985-89</td>
<td>8.1</td>
</tr>
</tbody>
</table>

female population, being most prominent in the 15-24 age-group, but it may be in part explained by greater recognition and increased reporting of cases, due to better medical and public awareness of the disorder. Thus, whilst there probably has been a real rise in the number of cases, the official figures may exaggerate its extent, recording some that would have remained undetected earlier.

There appears to be a definite at-risk age-group, ranging from about 14 to 23 years. Theander (1970) reported an average age of onset of 17.2 years – a figure confirmed by Beumont et al (1978) amongst his patients, although he said that the age of onset depended upon when the illness is regarded as beginning. This could be when weight-losing behaviour started, when weight loss actually began, when menstruation stopped, or once the patient became emaciated. Touyz and Beumont (1984) found an average age of onset of 16.6 years in the patients seen by their service.

Prevalence rates of anorexia nervosa vary according to the age-range of the population studied, with higher rates for younger age-groups, e.g. if the sample ranges in age from 14 to 25. Crisp and McGuinness (1976) found a prevalence rate of 0.25 per cent amongst schoolgirls aged over 16 years in England, but girls in independent and boarding schools had a rate of one per cent, whilst 3.5 per cent of fashion students and 7.6 per cent of professional ballet students in Canada were found to meet the diagnostic criteria for anorexia nervosa (Garner and Garfinkel, 1980). One consistent finding is that anorexia nervosa is more common among females, with a ratio of around one male to every 12 females (Szmukler et al, 1986). If the prevalence of anorexia nervosa is assumed to be about one per cent of those women aged 15-29 years, about 70,000 women in the UK would be anorexia nervosa sufferers.

Anorexia nervosa has often been seen as a condition predominantly affecting white populations of higher socio-economic status. However, more recently evidence has emerged that the disorder is now present in a wider cross-section of the population, and this may in fact have always been the case. Those in lower socio-economic groups and ethnic minorities tend to under-utilise medical facilities, and this may be an explanation for their apparent omission from statistics of eating disorders in the past. It may also reflect the fact that many young people from ethnic minorities are second-generation, having been born in the UK. They may now be adopting British culture and values in a way that their parents did not, so that they are facing the same kind of psychological pressures as the dominant group in society.
Hoek (1991) suggests that ‘it is probably more difficult to detect eating disorders than other psychiatric disorders, because eating disorders are characterised by taboo and denial’. Fairburn and Beglin (1990), describe the importance of the nonrespondents in studies, since eating disorders are often deliberately hidden, thus a low response rate may hide many sufferers.

**Bulimia nervosa**

Since bulimia nervosa is a fairly newly identified condition, it is likely that many cases remained undetected in the past. It is difficult to know whether the incidence rates reported by recent studies are simply a reflection of greater knowledge and detection rates concerning the disorder or whether there has been a real rise in the number of people with bulimia nervosa. Only a small number of individuals fulfilled the diagnostic criteria for bulimia nervosa when Russell (1979) originally described the condition; indeed, it had taken him over six years to collect 30 cases.

One of the few incidence studies available for bulimia nervosa is based on a large Dutch sample (Hoek, 1991). The reported incidence rate was 9.9 per 100,000 population per year during the period 1985-86 and 11.4 during 1985-89. Only four per cent of the bulimia nervosa sufferers were male.

A considerable number of studies now exist concerning the prevalence of bulimia nervosa, and if DSM-III-R criteria are applied, they have yielded consistent findings (Fairburn and Beglin, 1990). Generally, the groups investigated have been easily accessible student populations and those communities in which bulimia nervosa is thought to be most common, e.g. Caucasian females aged 14 to 40 years. This has led to studies showing apparently high rates of bulimia nervosa, particularly in those which relied on self-reporting.

Those studies with a clinical assessment of respondents reveal that between one and two per cent of young women have bulimia nervosa (Johnson-Sabine et al, 1988; Cooper and Fairburn, 1983; Schotte and Stunkard, 1987), though estimates vary slightly, depending on the diagnostic criteria used and the age-group studied. If the lower rate were applied to the UK female population aged 15 to 45 years, it would imply that about 125,000 women would meet the criteria for bulimia nervosa. However, many such cases remain undetected in general practice: a study by Whitehouse et al (1992) of 540 women aged 16 to 35 years found a prevalence of 1.5 per cent, although 5.4 per cent were suffering from a partial syndrome. Of the patients selected for further assessment and
diagnosed as bulimic, half the cases had not previously been identified by the GP, but specialist referral was required to treat secondary complications of the disorder in only a couple of patients. One of the highest estimates has come from Beumont and Touyz (1987) who suggest that the prevalence of bulimia nervosa among student populations could range from four to as high as 19 per cent. They claim that 'there can be little doubt that the condition is very widespread among young women and adolescent girls in most developed societies of the Western type'.

Lacey (1993) carried out a catchment area study in two, outer, south-west London boroughs. The mean age of the women with bulimia nervosa was 24.8 years, with binge-eating commencing at an average age of 18.8 years. Over a quarter of these women gave a history of regularly using 'street' drugs and over 40 per cent reported a history of stealing. Twenty-three per cent reported having taken at least one overdose and 15 per cent had cut their bodies. However, such findings have not been reinforced by other studies and these results seem extremely unusual.

Partial syndrome subjects are defined as fulfilling some, but not all of the diagnostic criteria for anorexia or bulimia nervosa. The prevalence of partial syndrome bulimia is estimated to lie between 1.8 and five per cent of the female school and student population (Johnson-Sabine et al, 1988; Mann et al, 1983; Button and Whitehouse, 1981). However, references to the partial syndrome are open to subjective interpretation of whether or not the disorder is present, and thus cause definitional problems.

Reports of bulimia nervosa among men are rare. One of the most rigorous studies to date found a combined prevalence (bulimia nervosa and the partial syndrome) of 3.9 per cent among women and 0.5 per cent among men (King 1986, 1989).

Bulimia nervosa has been reported most frequently in Caucasians living in Western Europe, North America and Australasia, with few reports among non-Whites. However, Hsu (1987) suggests that the number of cases among Blacks is increasing. Mumford and Whitehouse (1988) looked at the prevalence of bulimia nervosa among Asian and white schoolchildren in Bradford, finding a higher prevalence among the Asian girls (3.4 per cent versus 0.6 per cent). This may indicate, as suggested above, that second generation ethnic minorities residing in the UK adopt Western attitudes to weight and shape, but until more studies have been carried out no firm conclusions can be reached.

Fairburn and Beglin (1990) found that the majority of the 50 plus prevalence studies they identified relied on self-report
questionnaires. The samples often consisted of students enrolled in private prestigious universities. Fairburn and Beglin contend that such subjects are hardly representative of women in the community. They express doubts concerning family planning clinic samples, since low weight subjects are likely to have low or absent sexual appetite, and thus be underrepresented. General practice samples are likely to overrepresent bulimic women, however, due to the psychological and physical problems which usually accompany the disorder. Two-stage design studies, which use a self-report screening instrument, to identify individuals with probable bulimia nervosa, then interview them to obtain a definite diagnosis, demonstrate the short-comings of relying on self-reporting alone. The prevalence rates are markedly lower when an interview is added. 'These lower rates reflect the improved method for making diagnoses' (Fairburn and Beglin, 1990).

Fairburn and Beglin point out that studies have become increasingly sophisticated and the diagnostic criteria refined. Whilst a prevalence rate of one per cent among adolescent and young women appears to be consistently reported in the most sophisticated studies, 'the evidence that eating disorders are overrepresented among those who choose not to cooperate with prevalence studies suggests that the figures obtained may be an underestimate of the true rate' (Fairburn and Beglin, 1990). They also claim that only a small subgroup of those identified with bulimia nervosa are actually in treatment. Fairburn and Cooper (1982) found that less than one-third of the patients they identified with bulimia nervosa had ever mentioned their eating difficulties to a doctor, although more than half of them felt they definitely needed medical help.

**Mortality: anorexia and bulimia nervosa**

Crichton-Miller (1938) described anorexia nervosa as a compromise with suicide. The Eating Disorders Association state that anorexia nervosa has 'one of the highest mortality rates of all psychiatric illnesses – over 10 per cent of sufferers die either from the effects of starvation or by committing suicide', actual studies of mortality appear to have a wide range of results (see Table 3). Many of these variations can be explained by differences in length of follow-up, sample size, and diagnostic criteria.

The highest rates of mortality from anorexia nervosa are observed in those studies with the longest follow-up period. However, in some studies such as Theander’s (1985), with a mean follow-up period of 33 years, many of the sample will have died from causes
completely unrelated to their anorexia nervosa. The only study to use standardised mortality ratios was a comprehensive survey of 460 patients over 10 years (1971-81), which gave a mortality rate of six per cent (Patton, 1988).

Data concerning mortality in bulimia nervosa are sparse. Patton (1988) found a rate of 3.1 per cent (SMR) but further work is required to obtain a clearer overall finding on this issue.

Table 3  Mortality in anorexia nervosa

<table>
<thead>
<tr>
<th>AUTHOR/ YEAR</th>
<th>COUNTRY</th>
<th>NUMBER IN SAMPLE</th>
<th>MEAN FOLLOW UP (YEARS)</th>
<th>MORTALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hsu et al 1979</td>
<td>Britain</td>
<td>100</td>
<td>5.9</td>
<td>2 per cent</td>
</tr>
<tr>
<td>Isager et al 1985</td>
<td>Denmark</td>
<td>151</td>
<td>12.5</td>
<td>8.2 per cent</td>
</tr>
<tr>
<td>Theander 1985</td>
<td>Sweden</td>
<td>94</td>
<td>33</td>
<td>18 per cent</td>
</tr>
<tr>
<td>Patton 1988</td>
<td>Britain</td>
<td>460</td>
<td>10</td>
<td>3.3 per cent</td>
</tr>
<tr>
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<td>6* per cent</td>
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*standard mortality ratio (SMR)

Source: Adapted from Patton (1988).
AETIOLOGY

Anorexia nervosa

There are a plethora of theories regarding the cause(s) of anorexia nervosa. Table 4 provides a brief synopsis of some of the competing theories. Single-factor causal theories have been largely replaced by a view that anorexia nervosa is a multifactorial disorder. Three broad classes of predisposing factors emerge: individual (biological and psychological), familial and cultural (Garner, 1993). Individual predisposing factors would include psychological problems (however, it is difficult to know whether psychological problems caused, maintained or simply resulted from the disorder), depression (which may be secondary to starvation and coexisting

<table>
<thead>
<tr>
<th>EXPLANATION</th>
<th>FEATURES</th>
<th>WHY ANOREXIA?</th>
</tr>
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<tbody>
<tr>
<td>Emotional/ psychological (Garfinkel &amp; Garner, 1982)</td>
<td>Emotional crossroads - teens/early 20s. Threat of sex, pregnancy, leaving home, etc. Major life events probable as adulthood approaches.</td>
<td>Remove symbols of womanhood - hips, breasts, buttocks. Sufficient weight loss will prevent menstruation - denial of growing up process.</td>
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<tr>
<td>Emotional tribulation (Garfinkel &amp; Garner, 1982)</td>
<td>Parental divorce, alcoholic parents, physical/sexual abuse. Feeling unwanted, unhappy, lonely and shy.</td>
<td>Emotional tribulation leads to psychological problems leading to utilising anorexia as a coping mechanism.</td>
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<tr>
<td>Separations (from person or environment)</td>
<td>New circumstances require new demands which may involve a threat to ones self-worth and lack of control over the situation (Darby et al, 1983). Death of a parent before age of 16 (Halmi, 1974).</td>
<td>The rest of life feels out of control so decide to take control over own body by instituting a rigidly restrictive eating pattern.</td>
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Table 4 Competing explanations for the cause of anorexia nervosa
<table>
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<th>EXPLANATION</th>
<th>FEATURES</th>
<th>WHY ANOREXIA?</th>
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<tbody>
<tr>
<td>Family problems</td>
<td>Lack of parental love/support (EDA, 1992).</td>
<td>Route to anorexia not entirely clear.</td>
</tr>
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<td>Parents older than on average (Theander, 1970; Hall, 1978).</td>
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<td>However, difficult to distinguish between family conflicts resulting from</td>
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<td>anorexia and those leading to anorexia.</td>
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<tr>
<td>Psychiatric problems</td>
<td>Family history of mental illness (Kay and Leigh, 1954; Thoma 1967;</td>
<td>One hypothesis is that anorexia is a manifestation of an affective disorder</td>
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<td></td>
<td>Dally 1969; Hudson et al, 1983; Gershon et al, 1983).</td>
<td>e.g. a feature of a depressive disorder.</td>
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<td>However, definitions of family member and mental illness not always</td>
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<td></td>
<td>clear.</td>
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<tr>
<td>Exercise addiction</td>
<td>Excessive exercising develops as a major behavioural problem. Exercise</td>
<td>Clear relationship develops between caloric intake and physical</td>
</tr>
<tr>
<td></td>
<td>causes induced states of euphoria, relieves anxiety and depression –</td>
<td>exercise (debtting). Food intake only permitted if earned by burning</td>
</tr>
</tbody>
</table>

Sources: as indicated in the table.

complications), anxiety (obsessive-compulsive symptoms appear to be common) and personality disorder (which may take the form of self-mutilation, suicide attempts, stealing – probably more common among bingeing and purging anorectics). Psychological and physical trauma, possibly resulting from sexual abuse, would also fall into this classification as would genetic susceptibility to anorexia nervosa. 'Genetics may contribute to a specific vulnerability, or may operate indirectly by predisposing to obesity or personality traits that make restrictive dieting and anorexia nervosa more likely' (Garner, 1993).

Familial factors alludes to evidence of mothers who are dominant, intrusive, ambivalent and fathers who are passive and ineffectual. The family may seem overprotective and rigid.

Cultural factors refers to cultural pressures on women to diet and role conflicts experienced by women which may also be implicated. Rintala and Mustajoki (1992) described how the shape of shop
**BOX 2 Case studies**

**Case 1:** 'Susan' had a twin, as well as two brothers and a sister; all the children went away to boarding school at appropriate ages. The parents were workaholics and not physically affectionate towards the children. Susan felt that if she did well at school, her parents would be proud of her and therefore love her; this caused her to develop a perfectionist streak. However, 'A' levels were proving too much for her, and combined with the fact that she hid her feelings, in order to appear adult and in control, this led to crippling depression, resulting in a period of self-starvation and deliberate self-harm. The situation forced Susan to leave school, and she began seeing a psychiatrist. After some time, she managed to return to school and pass sufficient 'A' levels to get into art college, where although she loved the course, she again became anorectic. This led to two months in hospital, where for some time, she had to be fed through a nasogastric tube. Though at one time she was given two weeks to live, she made a good recovery and has since returned to art school.

**Case 2:** 'Mary' became anorectic in her early teens. What little she did eat was 'earned' through exercising: she would run over 100 miles a week and pedal an exercise bike for four hours a day. She described creating her own little world, which no-one else could enter – there, she was safe and untouched. Mary was sexually abused as a child. It was considered that abusing food and exercise was her way of blocking out feelings and memories that were too painful and frightening. She said that, 'they [exercise and anorexia] have been a way of denying my womanhood and my sexuality, and a way of punishing this body which I have hated so completely'. She is still struggling to come to terms with the past, but has discovered a world outside of food and exercise and is developing a fulfilling life.

**Case 3:** 'Carol' became anorectic when she was 13 years-old when she felt pressurized by her own excellent academic achievements. Approval from teachers and parents was gained through high attainment levels, so that perfection soon became the only acceptable standard and an obsession giving a great sense of worth.

Carol began to feel her life was 'out of control', with her parents dictating, for example, what options she should take for GCSEs – disregarding her own choice of subjects. Even her body felt 'out of control', as she started menstruating at this time: womanhood was perceived as a threat.

Carol's parents missed the anorexia nervosa symptoms for 18 months, until Carol's tutor contacted them about it. Carol said she had 'found it extremely difficult to explain to anyone that the art of my starvation was not in trying to kill myself, but in trying to keep my psychological self alive. I needed a coping mechanism, and I needed a means of somehow expressing 'me". She says desperately needed to know that she was loved and accepted.

When she had passed all 10 GCSEs with good grades, her parents tried to dictate to her what 'A' levels she should take. However, after a period in a self-help group and with a therapist, Carol began to take control of her life. She arranged to see a psychotherapist, changed her 'A' levels, and passed her driving test. She believes her greatest achievement is being able to say, 'I'm doing/not doing this because I want to and that's all'. Sources: Eating Disorders Association, Why should a teenager starve to death in an affluent society? Annual Report 1992. Eating Disorders Association, Signpost: Rattling the bars, February 1992.

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2 These case studies are, of course, anecdotal instances rather than those found to be typical in rigorous scientific study of patients. Caution should therefore be exercised in drawing any conclusions about causation from these cases.
mannequins (display figures) have become thinner with time suggesting that their proportions 'now differ considerably from those of normal young women'. In fact they claim 'a woman with the shape of a modern mannequin would probably not menstruate'. To be slim, fit and active is applauded in our society. In isolation social factors would be unlikely to lead to anorexia, but for young people, particularly girls, such social pressures when combined with personal or familial factors may encourage anorexia nervosa to be utilised as a coping mechanism in an attempt to avoid facing the underlying problems in their lives.

Box 2 provides three case studies which demonstrate some of the varied but often typical features which are associated with anorexia nervosa. These are, of course, anecdotal instances, and caution should therefore be exercised in drawing any conclusions about causation from them. However, the trait of high-achieving perfectionist is clearly evident in two cases, with the cause being unaffectionate or pressurizing parents. In the remaining case, sexual abuse is presumed to be the cause of the sufferer's anorexia nervosa.

It may be that a proportion of cases of anorexia nervosa emanate from each of these causes, or alternatively that some combination of these factors may be the cause. Since not everyone experiencing these situations develops anorexia nervosa, an element of personality or psychological/psychiatric make-up may well play an important role in determining the response to such situations.

**Self-perpetuating factors**
Some of the symptoms caused by dieting and starvation help to perpetuate the illness. Starvation both intensifies food preoccupations and affects the individual’s self-concept and sense of self-control, since impaired concentration, indecisiveness, mood lability, and sleep disturbance are all likely to occur. Just as starving people may feel hungrier after they have eaten something, so in anorexics this may exacerbate fears of loss of control. Thus, once initiated, anorexia nervosa can become a self-perpetuating illness.

Some of the vegetative features of depression – low self-esteem, sleep disturbance, loss of interest, reduced libido, and social withdrawal – are probably the result of the nutritional disturbances in anorexia nervosa, rather than a cause of them.

**Bulimia nervosa**
Since bulimia nervosa is a relatively newly defined illness, even less scientific evidence concerning its causes is available than with anorexia nervosa. However, as some cases of bulimia nervosa
develop from anorexia nervosa, it is reasonable to assume that many of the factors associated with that disorder will apply to bulimics. However, Casper (1980) suggested that bulimia nervosa sufferers differ from anorectics in that they are more extroverted, more interested in sex, and more likely to have had heterosexual experience. Beumont (1977) suggested that those anorectics who vomit and purge are more likely to have been premorbidly obese, sexually active, and extroverted, and to have histrionic personalities. These differences suggest that causes such as a fear of growing up and entering adult relationships are less likely to be factors in precipitating bulimia nervosa.

It is much less likely that the bulimic’s family will be as concerned as an anorectic’s with regard to the patient, since bulimics are often of normal weight, so that the threat to the patient’s life is not as perilous. Thus, a person with bulimia nervosa may succeed in keeping the illness secret from family members, whereas anorectics become very obviously ill. As a result, many of the patient/family interactions which may result from anorexia nervosa and possibly act as a contributory cause in perpetuating the illness, are less likely to occur in cases of bulimia nervosa.

There has been much literature regarding sexual abuse as a risk factor for bulimia nervosa. Pope and Hudson (1992) reviewed this issue and found six controlled retrospective studies. However, methodological problems were identified in most of these. Some studies failed to match the control group for sex, others used unsatisfactory definitions of sexual abuse and/or had poor evaluation of the abuse. In some cases sexual abuse began after the eating disorder. Four of the six studies found no significantly greater prevalence of sexual abuse among bulimic patients. Pope and Hudson concluded that, ‘current evidence does not support the hypothesis that childhood sexual abuse is a risk factor for bulimia nervosa’. However, Welch and Fairburn (in press) argue that ‘sexual abuse is a risk factor for the development of bulimia nervosa, [but] it does not appear to be specific to bulimia nervosa nor is it relevant to most cases’.

**Summary**

Anorexia and bulimia nervosa are triggered by difficult circumstances emanating from a number of possible sources, resulting in psychological disturbances. They may occur as the patients attempt to take control of their own bodies in circumstances in which they seem less and less in control. Becoming obsessed with diet and food removes the focus from those troublesome areas of
their lives where they feel unable to cope with or to control events. 'Different single-factor causal theories have been proposed, but the trend in recent years is to view the disorder as heterogenous and multifactorial, arising from the interplay of psychological, familial, cultural, and biological predisposing factors' (Garner, 1993).
TREATMENT

Anorexia nervosa
The main problem in treating anorexia nervosa appears to be that, whilst a return to normal weight is common, in the short-term, in the longer-term the relapse rate is high and the outcome therefore uncertain. Patient compliance is a major problem in the treatment of anorexia nervosa.

There are two fundamental purposes of treatment for both anorexia and bulimia nervosa. The first is to restore normal body weight, normal eating patterns, and physical health, while the second is to promote attitudinal change, psychological readjustment, and the resumption of emotional maturation and growth. The first stage will be crucial in restoring physical health and possibly in preventing death. The second is important if a successful outcome in the long-term is to be envisaged. Anorexia nervosa sufferers are usually treated through an out-patient programme involving a mixture of psychotherapy and nutritional counselling. The treatment mix is adapted to the needs of the individual patient. Pharmacological agents are rarely used except to treat cases of concomitant depression. However, a successful outcome often requires long-term treatment: Szmukler et al (1986) found that 14 per cent of patients required over four years of treatment. There appears to be no general consensus on the appropriate treatment mix for all anorexia nervosa patients and numerous types of therapy have been tried.

In the following discussion of treatment, we examine family therapy, behavioural therapy, psychotherapy, the problem of compliance, the need to achieve a therapeutic alliance, the role of nutritional counselling, whether hospitalisation is necessary, and common shortcomings in treatment studies.

Family therapy
For over a decade following Minuchin’s (1976) delivery of a paper at the first international conference on anorexia nervosa, family therapy was uncritically supported as the treatment of choice for anorexia nervosa (Rosman et al, 1977) and it remains the treatment of choice for younger patients. Minuchin’s impressive results were, however, questioned due to methodological weaknesses in the study.

Russell et al (1987) undertook a controlled trial comparing family therapy with individual supportive therapy among 80 patients, 57 of whom suffered from anorexia nervosa. The family therapy included all members of the household of the anorectic patient.
Children who had left home, as well as, separated or divorced parents were also encouraged to attend. The first task of the therapy was to gain the family’s cooperation. The therapist raised family anxiety towards anorexia by stressing the specific dangers of the malady, while countering their tendency to blame themselves for the patient’s illness. The second stage of therapy involved an assessment of the family organization. The way family members related to each other and alliances, especially, the quality of the marriage partnership and the family’s command and control techniques were assessed. It was assumed that the family was ineffective in helping the patient to eliminate her symptoms or might contribute to their maintenance, although this assumption was not based on an underlying belief that anorexia nervosa was caused by the family.

The final task was to make interventions to help the family change. The most common interventions involved parental management of the patient’s symptoms and other aspects of her life.

Individual therapy was used as the control therapy. The therapy consisted of frequent one-hour sessions involving supportive, educational, and problem-centred treatment. After one year the effects of family therapy and individual therapy were compared.

The main finding from the study was that family therapy is an effective treatment for a subgroup of patients whose illness commenced before 19 years of age and was of less than three years duration. Individual supportive psychotherapy appeared to be more effective in patients where the illness was chronic and had a later age of onset; although complete recovery was much rarer in the latter cases whatever form of treatment was chosen.

Hall (1987) suggests that individual therapy such as cognitive-behavioural therapy ‘can be undertaken concurrently with family therapy’. Hodes et al (1991) say that ‘there is growing empirical evidence that family therapy provides a helpful and effective model of outpatient treatment of anorexia nervosa in children and adolescents’. They add that it can also be a useful adjunct to ward management in those cases where hospitalisation is necessary.

**Behavioural therapy**

Behavioural programmes may be used in hospitals. They often make access to pleasurable activities contingent upon weight gain; in severe cases, the patient is usually confined to bed and most of her possessions removed. However, such regimes have been criticised on many grounds, not least that they impede subsequent psychotherapy. According to Agras and Kraemer (1984),
behavioural therapy is efficient in terms of the rate of weight gain, compared to other forms of therapy. This conclusion was based on the pooled data from 21 studies, encompassing 193 patients: eventual discharge weight was the same whether behavioural or medical therapy was used, but with the former the duration of treatment was shorter. In combination with family therapy, behavioural procedures have achieved impressive results, but mainly when applied to young patients with a short history, and it is well-established that such patients often have a good prognosis, whatever form of treatment is used (Garfinkel and Garner, 1982).

**Psychotherapy**

Casper (1987) says that ‘the main purpose of psychotherapy is the reorganisation of the dysfunctioning personality through individual and family therapy’. However, this treatment should only be initiated with the understanding that psychological changes need years, rather than months. Psychotherapy requires trust, honesty, and sincerity, but these very qualities are distorted by the eating disorder itself.

Bruch (1962) claimed that the cause could be early personality problems arising out of a faulty interaction with the mother. It was also her view that anorectic children were frequently overprotected and overcontrolled by their mothers (Bruch, 1973). Clearly, the nature the psychotherapy takes is likely to be dictated by what are perceived to be the cause of the illness and any perpetuating factors.

The patient is usually antagonistic towards any treatment which includes refeeding, and desperately attempts to defend her thin body. A sensitive approach is required, since if the pressure for weight gain and the speed of increase frighten the patient, she may become suicidal. Even gentle and kind persuasion to eat may be experienced as a compelling influence. Many months of patient work may be required before a therapeutic alliance is achieved, and only once this happens is there a chance for psychological recovery. Psychotherapy aims to lead the patient away from the defensive structure related to food-weight and exercise, towards the more fundamental issues such as, a sense of inadequacy, inner disorganisation, rebellion, and anger.

As patients gain weight, they may complain of distressing states of confusion, emptiness, and depression. The removal of their thin body – perceived as the only worthwhile goal in life – is perceived as a total loss of purpose, and they can become hopeless to the point of wanting to die. Thus, extreme sensitivity and caution are required when attempting to treat people with anorexia nervosa, in order to avoid any counter-productive outcomes.
Treatment resistance
A familiar and significant problem encountered in the treatment of anorexia nervosa is that the patients themselves are usually uninterested in, or resistant to treatment. Denial is the main obstacle which must be overcome before a therapeutic alliance with the patient can be achieved. The first step is to establish a good doctor-patient relationship as well as a positive alliance with the patient's family. The clinician has a fine line to tread - challenging any unrealistic statements that the patient makes, but without appearing aggressive or threatening, which is likely to be counter-productive.

Often, patients will be brought unwillingly by anxious parents who are concerned at their child's ailing physical state. In these circumstances, questions arise concerning the patient's right to refuse treatment and the right to enforce treatment on those considered to be a danger to themselves.

Achieving the therapeutic alliance
Restoring nutritional status is a first, necessary step. To encourage compliance, the patient can be told that a decrease in obsessive thoughts about food and body weight and relief from insomnia and depressive symptoms may be expected, once the treatment begins to work. In patients with extreme emaciation, reassurance that restoration of previous activity levels can be achieved may be helpful in persuading them to enter a treatment programme.

Treatment plans should have a high degree of transparency or openness for both patient and family: the patient should be fully informed and see herself as an active participant. The patient's feelings of ineffectiveness should be minimised and her control over what is happening in her life enhanced, increasing her responsibility for her own health. Since the family's support for a treatment programme is essential, they should be fully informed about its rationale.

Nutritional counselling in anorexia nervosa
Little has been published concerning either the role of the dietitian or the effectiveness of nutritional management as part of a treatment programme. This neglect may be partly explained by an assumption that the eating disorder is merely a secondary, albeit distinguishing feature of the illness. It is often presumed that the abnormal eating behaviour will correct itself, once the patient has received the necessary psychotherapy. In addition nutritionists and dietitians are more used to dealing with patients who eat too much, but are probably not much used to dealing with anorectics who must be encouraged to eat more.
The status of the dietitian may be less threatening than that of doctors, who may be viewed as authority figures collaborating with the family in coercing the patient to change behaviour. In their programme, Touyz and Beumont (1984) employed the dietitian as part of a multi-disciplinary team. The dietitian collected detailed nutritional and behavioural information in the form of an eating and dietary history, which all members of the team use to assess and manage the patient. The dietitian also prescribed and supervised daily meals. Direct advice regarding nutritional education to correct distorted ideas about food was given to the patient as well as a guide to ‘relaxed and healthy eating patterns’. Patients were asked to provide self-reports on food intake over 24 hour periods, this information being usually verified by other family members. The patient’s beliefs and fears concerning food were also collected, and her nutritional knowledge assessed.

Whilst it is fairly common to see weight gain after treatment is initiated, the abnormal eating behaviour and attitudes may not have been altered. Frequently, abnormal eating habits such as eating very slowly, cutting food into tiny portions, leaving the table frequently, agonizing over what to eat, and avoiding social contact or conversation whilst eating remain – mitigating against any long-term success. To avoid the patient covertly disposing of food after pretending to eat it, extensive pressure to eat is avoided. The amount of food prescribed is not increased until the patient is satisfactorily completing each meal. On attaining their target weight, patients select their own food, gradually reducing their daily input until their weight stabilises.

In order to reassure patients who may be concerned about overshooting their target weight, it should be emphasised that there is a normal weight range rather than a single normal weight. Also, patients are encouraged to eat in a variety of situations, e.g. at home, in restaurants, and in other social settings. Continuing friction between the patient and family regarding food and meals may exacerbate the situation, with the patient feeling constantly pressurised to eat more. If possible, the dietitian will help to defuse the situation, and counsel both the patient and family about food requirements and how these should be met.

The dietitian may also have an input into the care of patients who are never admitted to hospital, but only seen as out-patients. Such patients may be referred to a dietitian both for a detailed diet history and in order to provide a structured eating pattern which will promote weight gain. Meals are constructed to suit individual requirements, aiming at a weekly gain of 0.5 kg, but patients have to
learn to eat in a spontaneous manner and not stick ritualistically to the dietitian’s prescription of food.

Anorectic patients are often shocked when they see how much is required just for the maintenance of normal body weight. As soon as patients start eating normally, they are likely to gain weight as result of rehydration, but thereafter, weight gain should be more gradual. Patients should be warned and reassured on this aspect of changes in weight.

**Hospitalisation**

Treatment will be centred on an out-patient basis unless emergency situations call for admission to hospital. Beumont et al (1987) report that only 10 per cent of their patients are admitted to hospital, and then only for a short period during a phase of crisis. The decision to admit an eating disorder patient is usually based on a combination of criteria, which will include serious and potentially life-threatening deterioration of the patient’s general health. This may be due to unremitting extreme weight loss (e.g. more than 30 per cent of normal body weight), dangerous alterations in vital signs (postural hypotension, bradycardia – slowness of the heart beat and pulse, hypothermia), and psychotic reactions or suicidal tendencies or attempts. A patient in a seriously disturbed life situation, such as marked family disturbance, which may be a cause or consequence of the illness, may also require admission, as may patients with previous treatment failures or those who refuse to engage in outpatient therapy. The decision whether or not to admit a patient may involve certain dangers, e.g. it might increase the risk of suicide due to the abrupt change in the patient’s environment.

If hospital treatment is felt to be necessary, it must then be decided where the patient should be admitted. A medical setting may avoid the stigma attached to a psychiatric unit, but the decision will largely depend on the family’s attitude towards the illness – whether they see it as a somatic or a psychological condition – and on a particular hospital’s experience of treating eating disorders.

**Shortcomings of studies**

Hsu (1987) criticised the lack of clinical data and inadequate diagnostic criteria present in some studies, as well as their inclusion of atypical patients and omission of pertinent variables. Inadequate descriptions of the treatment, in terms of the treatment setting, method, and duration were also described. However, the most common methodological failings he identified were an insufficient follow-up period and/or a high failure rate in tracing subjects.
Hsu suggests that the following criteria should be used to assess the validity of studies on eating disorder patients. Firstly, the diagnostic criteria should be explicitly stated, so that atypical cases are excluded. Secondly, there should be at least 25 patients in the study. Thirdly, the follow-up period should be for a minimum of four years. Fourthly, there should be a failure-to-trace rate of no more than 20 per cent. Fifthly, direct interview at follow-up (rather than telephone or written contact) should be employed in over half the patients. Finally, multiple well-defined outcome measures should be applied.

Six studies in the period 1970-84 were identified which conformed to his criteria for a valid study. A good outcome was defined as body weight being maintained within 15 per cent of the average for height, weight, age, and sex, together with a regular menstrual cycle, where applicable. An intermediate outcome occurs when weight has only occasionally been within 15 per cent of the average and there is continuing menstrual disturbance. In cases with a poor outcome, weight has never approached the average minus 15 per cent and menstruation has been sporadic or absent.

The six studies in question demonstrated a good outcome in 44 per cent of patients, an intermediate outcome for 28 per cent, and a poor outcome for 24 per cent. Less than 5 per cent had died. A poor outcome was associated with a longer duration of illness or more severe illness, a lower minimum weight, a failure to respond to previous treatment, and a premorbid disturbed relationship with other family members. Some studies, but not all, found vomiting to be associated with a poorer outcome.

**Bulimia nervosa**

In contrast to anorectics, most bulimia nervosa sufferers recognise their eating disorder and desperately want help, though it may take years before they approach a GP for help. The Eating Disorders Association say that one of the greatest difficulties facing sufferers wanting help at present is the sheer lack of resources available. Some bulimia nervosa sufferers may be admitted to a hospital in-patient programme, but this is not the case for most.

Fairburn et al (1992) identified four phases in the evolution of the treatment of bulimia nervosa. Phase I (1976-81) was the period in which bulimia was recognised as a distinct psychiatric disorder, although it was not until 1979 that Russell proposed the term 'bulimia nervosa'; Boskind-Lodahl had used the term 'bulimarexia' in 1976. There was a conflict of opinions between those who believed the condition was intractable (Russell, 1979) and those who reported
good results with brief interventions (Boskind-Lodahl and White 1978, 1981). Phase II was a short phase (1981-82) in which three promising approaches to treatment were described. Fairburn (1981) outlined a cognitive behavioural approach, designed to modify patient’s disturbed eating habits and their dysfunctional attitudes to shape and weight. Rosen and Leitenberg (1982) described a treatment known as exposure with response prevention. Finally, reports were beginning to emerge on the use of antidepressant drugs to treat the condition (e.g. Pope and Hudson 1982).

Phase III (1983 to present) has seen the scientific evaluation of these approaches to treatment. Other forms of treatment have also been described, but these have not been thoroughly tested. Phase IV is just beginning and has several strands. Studies have been designed to ascertain which elements of psychological therapy are effective and which are redundant or countertherapeutic. Comparative studies have been developed to compare and contrast treatment approaches, both in isolation and in combination. There is also interest in comprehensive treatment programmes, in which a variety of approaches are offered according to need.

In the following discussion of treatment we look at cognitive behavioural approaches, exposure with response prevention, self-help, and psychopharmacological interventions.

**Cognitive behavioural approaches**

This therapy is based on the assumption that a key factor preventing people with bulimia nervosa from spontaneously recovering is their extreme concerns about weight and shape. Cognitive behavioural therapy aims to modify concerns about weight and shape. Administered on a one-to-one basis this is the treatment of choice for bulimia nervosa. This therapy, on an out-patient basis, usually lasts for about 20 sessions over four to five months. The first stage is to establish some control over eating, and the techniques used include self-monitoring, establishing a regular eating pattern and weighing once a week. Fairburn (1985) suggests that patients weigh themselves only once a week, to avoid over-concern about their precise weight: in this way they are able to keep a check on their weight, without becoming preoccupied with it. To avoid feeling uncomfortably full after meals, it is advised that loose clothing should be worn. Patients are also provided with information about regulating body weight, dieting, and the adverse effects of vomiting and/or purgatives as a means of weight control. This stage will probably last about one month, on a twice-weekly basis.

The second stage is more explicitly cognitive. Here, an attempt is
made to reduce the tendency to diet and to train the patient in problem-solving using cognitive restructuring techniques. This stage lasts about two months, based around once-weekly appointments. In order to maintain any progress made, a ‘maintenance plan’, usually consisting of three fortnightly appointments, is initiated.

Beck et al (1979) proposed an approach where dysfunctional thoughts are identified by self-monitoring. The therapist may ask ‘what would you think if...’ type of questions, to elicit thought patterns. Patients are also asked how others may respond in the same situation, and whether they are applying one set of standards to themselves and another to other people. Their dysfunctional style of thinking may need correcting, as well as dysfunctional values and beliefs. These must be inferred from the patient’s behaviour, since the patient cannot look at them objectively, being so much a part of the person. The therapist tries to get the patient to examine the validity and significance of their underlying attitudes.

Assertiveness and social skills training may be a useful part of treatment, in dealing with problems not specific to eating disorders such as low self-esteem and social isolation.

Table 5 provides a brief synopsis of some of the studies which have been reported concerning non-pharmacologic therapy, but some of these suffer from one or more drawbacks, often relating to small sample sizes or lack of adequate follow-up. However, Lacey (1983) carried out a fairly large-scale (30 patients plus controls) controlled study in the UK, using both behavioural and cognitive techniques. Weekly sessions consisted of one hour divided equally between individual therapy and group therapy, over 10 weeks. Twenty-four patients were completely remitted by the end of the programme, whereas no changes were seen in the control group; 20 remained completely remitted at two years’ follow-up. Generally, studies concerning psychological treatments for bulimia nervosa have been of a higher quality than those of antidepressants. Although not all studies have produced such positive treatment benefits as Lacey, a reduction in the frequency of bingeing and purging can be expected, accompanied by an improvement in social functioning and general psychopathology. Whilst few studies have a follow-up period of over one year, the findings suggest that maintenance of change is good with less tendency to relapse than is seen when using antidepressants.

Fairburn et al (1992) state that, ‘other than cognitive-behaviour therapy, the only established psychological treatment that appears promising is interpersonal psychotherapy’. For less symptomatic
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<th>AUTHOR/ COUNTRY</th>
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<td>TROLLED</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schwartz USA</td>
<td>30</td>
<td>No</td>
<td>Psychotherapy</td>
<td>20 binge/purging less than once a month; 6 improved; 4 no change</td>
<td>16 months: 20 remained well; 2 of 6 improved failed to maintain improvement</td>
</tr>
<tr>
<td>et al (1981)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lacey UK (1983)</td>
<td>30</td>
<td>Yes</td>
<td>Individual and group therapy</td>
<td>24 totally remitted; no change in controls</td>
<td>2 Years: 20 remained totally remitted; 2 not traced</td>
</tr>
<tr>
<td>Pyle et al USA (1984)</td>
<td>104</td>
<td>No</td>
<td>Group therapy</td>
<td>49 totally remitted; 26 1-3 episodes per month; 11 four or more episodes; 11 dropped out; 6 non-compliance problems</td>
<td>None</td>
</tr>
<tr>
<td>Fairburn England</td>
<td>24</td>
<td>No</td>
<td>Cognitive behavioural technique and short-term focal psychotherapy (STP)</td>
<td>Binge/vomiting dramatically reduced. STP slightly less effective than CBT</td>
<td>12 months: most patients totally remitted</td>
</tr>
<tr>
<td>et al (1986)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Agras USA</td>
<td>77:</td>
<td>Wait list control</td>
<td>Self-monitoring; cognitive behavioural therapy; response prevention</td>
<td>77% reduction in purging - CBT; 53% reduction - response prevention</td>
<td>6 months: 59% totally remitted in CBT group; 20% in response prevention group; 18% in self-monitoring</td>
</tr>
<tr>
<td>et al (1989)</td>
<td>18 wait list; 16 self-monitoring; 17 cognitive behavioural therapy; 16 response prevention</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
patients, Fairburn et al suggest that educational treatment, as an initial intervention, might be sufficient for uncomplicated cases.

**Exposure with response prevention**

This technique involves exposing the patient to anxiety-eliciting clues (perceived or actual overeating) and then preventing vomiting. Patients bring to each treatment session, foods upon which they would typically binge, and then eat them to the point at which they would normally vomit. They are encouraged not to vomit but to learn to cope. However, studies using exposure-prevention in combination with cognitive-behaviour therapy do not indicate that it enhances the effectiveness of cognitive-behaviour therapy.

**Self-help**

Cooper (1993) has produced a book detailing a self-help version of the cognitive behavioural approach. The programme he sets out can be used by a therapist as part of a ‘guided self-help’ strategy or as a straight forward self-help manual by the patient without professional help. It will only work for those patients very highly motivated to resuming normal eating patterns and stopping bingeing. Cooper suggests that it would take about six months to work through the six stage self-help programme. However, if there is no progress after six weeks he advises patients to consult their GP. A GP or practice nurse could become a ‘helper’ to aid particularly during times of demoralization to prevent the patient giving up the programme, but in any event the ‘helper’ should be seen once a week. A friend or relative could also take on the ‘helper’ role.

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**Table 5 Continued**

<table>
<thead>
<tr>
<th>AUTHOR/ COUNTRY</th>
<th>PATIENTS</th>
<th>THERAPY</th>
<th>OUTCOME</th>
<th>FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freeman et al (1988)</td>
<td>112: Randomised behavioural control therapy; 30 trial (waitlist controls); 30 group therapy; 20 wait list controls</td>
<td>Cognitive behavioural therapy; behavioural therapy; group therapy</td>
<td>Binge/vomiting; reduced vomiting; by all methods; but group therapy</td>
<td>2 years</td>
</tr>
</tbody>
</table>
Psychopharmacological intervention

Antidepressants have been the most extensively investigated pharmacologic approach. Overall, the results of studies (see Table 6) seem to suggest that antidepressants are more effective than placebo in reducing the frequency of bingeing and purging and the intensity of some of the other symptoms. Some studies suggest that this is true, whether or not the patients are actually depressed (Hughes et al, 1986; Walsh et al, 1988). Relapse appears to be common, whether or not the patient remains on the antidepressant. However, problems concerning the short duration of the studies and the assessment of unsophisticated and narrow measures detract from the studies.

Fairburn et al (1992) say that, 'the initial enthusiasm for antidepressants drugs can no longer be justified. It seems that they have a selective effect on the psychopathology of bulimia nervosa, which is transitory in many cases'. Cognitive-behaviour therapy has been shown to be more effective than antidepressants in studies which have compared the two treatments (Agras et al, 1990; Mitchell et al, 1990).

Table 6  Psycho-pharmacological intervention

<table>
<thead>
<tr>
<th>AUTHOR/COUNTRY</th>
<th>PATIENTS</th>
<th>CONTROLLED</th>
<th>THERAPY</th>
<th>OUTCOME FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pope et al (1983)</td>
<td>USA</td>
<td>21: Double-blind placebo</td>
<td>Imipramine</td>
<td>2 withdrew due to side-effects</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11 treated; 10 placebo</td>
<td>for six weeks</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2-8 months: 3 totally remitted; 6 reduced bingeing by &gt;50%; Placebo group unchanged</td>
</tr>
<tr>
<td>Pope et al (1983)</td>
<td>USA</td>
<td>9 from placebo group</td>
<td>No</td>
<td>Imipramine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>above</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sabine et al (1983)</td>
<td>UK</td>
<td>50: Double-blind placebo</td>
<td>Mainserin</td>
<td>No change in binge/purge for either group; depression/ anxiety reduced</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20 treated; 30 placebo</td>
<td>for eight weeks</td>
<td></td>
</tr>
</tbody>
</table>
Table 6 Continued

<table>
<thead>
<tr>
<th>AUTHOR/ COUNTRY</th>
<th>PATIENTS</th>
<th>CONTROLLED</th>
<th>THERAPY</th>
<th>OUTCOME FOLLOW-UP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitchell and Groat (1984)</td>
<td>32: 16 treated; 16 placebo (8 depressed patients in each group)</td>
<td>Double-blind placebo controlled</td>
<td>Amitriptyline + behavioural programme for all patients</td>
<td>Both groups improved, drug treated group moreso. Depressed patients poorer prognosis</td>
</tr>
<tr>
<td>Kennedy et al (1988)</td>
<td>18</td>
<td>Double-blind placebo controlled crossover study</td>
<td>Isocarboxazid</td>
<td>Over 50% reduction in binge/vomiting. Some side-effect problems continued due to side effects</td>
</tr>
<tr>
<td>Mitchell et al (1990)</td>
<td>171: 31 placebo; 54 imipramine; 34 placebo plus group therapy; 52 group therapy plus imipramine</td>
<td>Randomised control trial</td>
<td>Imipramine and group therapy</td>
<td>Imipramine reduced binge/vomit episodes by half; but group therapy most effective treatment</td>
</tr>
<tr>
<td>Walsh et al (1991)</td>
<td>78: 40 desipramine; 38 placebo</td>
<td>Double-blind placebo controlled</td>
<td>Desipramine (MAOI or fluoxetine if failed to respond to desipramine)</td>
<td>47% reduction in binge frequency; 12.5% totally remitted</td>
</tr>
</tbody>
</table>

Sources: As indicated in the text
Conclusion
There is no easy cure for either anorexia or bulimia nervosa. Recovery is a long, hard process, which often requires the patient to face painful issues she or he has perhaps been avoiding by being anorectic or bulimic. Stephenson et al (1986) stated that ‘at the present time, no single therapeutic technique is predictably effective in the treatment of all patients who have an eating disorder. Nor does any one profession possess the skills and energy necessary to meet the needs of these patients’.

One problem which lessens the validity and credibility of much of the findings from studies of eating disorders is the fact that many of these are uncontrolled pilot programmes with small samples. However, some good studies of anorexia nervosa do indicate that family therapy is the treatment of choice, although patient compliance and long-term recovery are often difficult to achieve. Young patients with a short history of anorexia nervosa usually have a good prognosis, indeed family therapy is particularly recommended for those under 19 years of age with a duration of illness of less than three years. If the condition is chronic and occurred at a later age of onset treatment is more challenging. Larger studies with longer follow-up periods are required to increase confidence in the effectiveness of particular treatment regimes.

The consensus from studies of bulimia nervosa support Fairburn et al (1992) who state that, ‘cognitive-behaviour therapy must be regarded as the treatment of choice for bulimia nervosa’. Nevertheless, they go on to point out that, ‘the cognitive-behavioural approach is neither necessary nor sufficient for all patients with bulimia nervosa... for some patients, the cognitive-behavioural approach constitutes overtreatment. For these patients, simple brief interventions are appropriate. For other patients, cognitive-behaviour therapy is insufficient’. A variety of treatment options according to the patients needs should be available, ranging from simple education and advice through to inpatient treatment for severe cases. However, there is a need for development and evaluation of simple initial interventions.

Antidepressants may have a reasonable transitory effect on bulimia nervosa but relapse is common in the long-term. Self-help or ‘guided self-help’ programmes may be of use to patients where their bulimic behaviour is not chronic.
COSTS

Anorexia and bulimia nervosa are conditions that can be long-term, with many potential complications and a high relapse rate. Both usually require a multi-dimensional approach to treatment, with consequent implications for cost: Stephenson et al (1986) state that, ‘an eating disorder is frequently a protracted and expensive illness’.

The most readily quantifiable component of the financial burden of eating disorders is the direct expenditure borne by the National Health Service. Data on the extent of treatment in the primary setting can be obtained from the 3rd National Survey of Morbidity in General Practice (OPCS, 1986). This study covered a 12-month period during 1981/2, and found that one per 1,000 females and 0.6 per 1,000 males consulted their general practitioner for anorexia nervosa (rates for bulimia nervosa were not available). Application of these rates to the 1990 UK population suggest that a total of 46,806 individuals consult their GP each year concerning anorexia nervosa.

The consultation rates for the female patients observed by the study was 1.5 per 1,000 for anorexia nervosa, the respective figure for men being 0.7 per 1,000. The average cost of a GP consultation was estimated at £9.85 (OHE, 1991). This yields an estimated cost for anorexia nervosa consultations of over £500,000 per annum.

The morbidity survey also provides information on the extent to which general practitioners refer patients to outpatient departments of general hospitals. Anorexia nervosa referrals are not reported separately, but are incorporated into a category which includes nausea and vomiting, heartburn, digestive gas problems, and hepatomegaly and splenomegaly. For that category the total figure is 1.3 per cent. It is probable that anorexia nervosa referrals are higher than this figure, but since no data are available regarding psychotherapy and other out-patient treatments, it is impossible to produce a reliable costing.

A large proportion of direct health care costs result from treatment on an inpatient basis. The Hospital Inpatient Enquiry estimates that in 1985, there were 1,000 admissions for females with anorexia nervosa in hospitals in England, and the mean duration of their inpatient stay was 21.5 days. Anorexia nervosa thus accounted for 21,500 hospital bed days in 1985 in England. Adjusting these figures pro rata to include the rest of the United Kingdom suggests that a total of 25,748 bed days were accounted for in the UK by anorexia nervosa. Taking an average inpatient bed day cost of £137.73\(^3\), it may

\(^3\) The 1990 DoH figure has been inflated by the RPI for 1991/92.
be estimated that hospital inpatient treatment for anorexia nervosa gives rise to annual NHS expenditure of £3.55 million.

The pharmaceutical costs are relatively low, since medicaments have to date played only a limited role in the treatment of anorexia and bulimia nervosa. However, appetite stimulants are used in the treatment of anorexia nervosa, although it is questionable whether this is justifiable. Anorexia (ICD 783.0) and anorexia nervosa (ICD 307.1) account for over half the prescriptions written for appetite stimulants in general practice and amount to 40,000 prescriptions per annum. Tonics are also occasionally used to stimulate appetite, but only 17,000 prescriptions for these were made out for people with anorexia. The combined value of retail chemist and hospital sales of tonics and appetite stimulants used in the treatment of anorexia nervosa totals about £150,000 per annum. Over-the-counter spending on tonics amounts to almost £4.5 million per annum; but self-medication for anorexia nervosa is unlikely, as most sufferers will not admit they are ill and hence do not actively seek treatment.

Antidepressants have been used in the treatment of both anorexia and bulimia nervosa. Fluoxetine is the only antidepressant on the UK market which is registered for use in both depression and bulimia nervosa ‘for the reduction of binge-eating and purging activity’ (ABPI, Data Sheet Compendium, 1991-2). Fluoxetine is a relatively expensive antidepressant, but only a negligible proportion of total prescriptions written for antidepressants are accounted for by people with anorexia or bulimia nervosa, so that the cost of this treatment nationally is therefore minimal.

In total, we estimate anorexia nervosa costs the National Health Service (NHS) over £4 million per annum but this omits the costs involved in providing psychotherapy, group, family, behavioural, and cognitive therapy. Data concerning consultations, in- and out-patient numbers and pharmaceutical use for patients with bulimia nervosa are not available.

Taking a broader perspective, there are other costs to consider to the health service and to society. Some anorectics and bulimics are known to be suicide risks. There are many more attempted suicides than actual suicides and medical treatment is usually necessary for uncompleted attempts, often involving accident and emergency departments, with further implications for costs. Lost production may also result from people with anorexia or bulimia nervosa being unable to work at some points during their illness. When suicide tragically occurs the loss of a young life is also of considerable economic significance.
It must be remembered that many anorexia nervosa sufferers are likely not to be in contact with treatment services, but may be a cost to society in a number of ways. The omission of bulimia nervosa from the costings, due to lack of data, is a serious one since bulimia nervosa is more common than anorexia nervosa and affects a wider age range. The treatment of choice for bulimia nervosa sufferers is a specific form of cognitive behavioural therapy administered on a one-to-one basis over several months, which would produce a large burden for the NHS if most people with bulimia nervosa presented for treatment.

Table 7 The cost of anorexia nervosa to the National Health Service

<table>
<thead>
<tr>
<th>HEALTH SERVICE SECTOR</th>
<th>ANOREXIA NERVOSA £MN</th>
</tr>
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<tbody>
<tr>
<td>General practice</td>
<td>0.58</td>
</tr>
<tr>
<td>In-patient</td>
<td>3.55</td>
</tr>
<tr>
<td>Out-patient</td>
<td>N/A</td>
</tr>
<tr>
<td>Pharmaceutical services</td>
<td>0.1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>4.23</strong></td>
</tr>
</tbody>
</table>

*Source: IMS, OHE estimates.*
CONCLUSION

This paper has highlighted the threat to health posed by anorexia and bulimia nervosa. These disorders cause considerable morbidity and mortality, and unfortunately, we are currently witnessing what appears to be a worrying upward trend in the reported prevalence of both conditions.

There is a general view that anorexia nervosa has a basic psychological causation. Societal pressures towards thinness probably encourage anorexia nervosa as a coping mechanism for certain, mostly young, female, individuals with underlying psychological disturbances. Bulimia nervosa also appears to originate from psychological problems, although there seems to be a broader range of person affected. A history of sexual abuse has been identified as a common feature in many sufferers backgrounds, but connection between the two has yet to be proved.

In terms of treatment, these conditions pose difficult problems. Nevertheless, cognitive-behaviour therapy is regarded as the treatment of choice for bulimia nervosa. Family therapy is the treatment of choice for anorexia nervosa, particularly in cases where the patient is under 19 years of age and the illness is of less than three years duration. Not all patients will respond to the same therapy. If the disorder is of recent origin, minimal brief intervention may be sufficient, whereas if the condition has remained undetected and untreated until it becomes chronic and perhaps life threatening, hospitalisation may be required.

A change in attitudes across our society which may help prevent both anorexia and bulimia nervosa, would be to place less value on slimness. This might help to prevent some vulnerable young people from turning to anorexia or bulimia nervosa as a way of dealing with their psychological problems.

The growing publicity surrounding anorexia and bulimia nervosa is a positive step, in that, sufferers may become identified medically at an earlier stage. Currently many anorexia and bulimia nervosa sufferers are not receiving medical treatment. Whilst greater levels of detection and treatment would seem to imply a greater burden on the NHS this may not necessarily be the case. If sufferers can be detected when the condition is at an early stage, a simple brief intervention may be sufficient, avoiding expensive hospitalisation or psychotherapy that might be required at a later stage in the illness. While clinicians are confident about the effectiveness of such brief interventions, it will be important to conduct formal evaluations of these new forms of treatment.
Self-help groups:
Eating Disorders Association
Sackville Place
44 Magdalen Street
Norwich NR3 1JU
0603-621414
(0603-765050 Youth helpline, for 18 years and under)

Anorexia and Bulimia Nervosa Association
Women’s Health Centre
Tottenham Town Hall
London N15 4RB

Anorexic Aid
The Priory Centre
11 Priory Road
High Wycombe
Bucks
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