**Eating Disorders**

1 Anorexia nervosa

1.1 Symptoms, presentation and patterns of illness

Anorexia nervosa is a syndrome in which the individual maintains a low weight as

*a result of a pre-occupation with body weight*, construed either as a fear of fatness or

pursuit of thinness. In anorexia nervosa, weight is maintained at least 15 per cent below

that expected, or in adults body mass index (BMI) – calculated as weight in kilograms

divided by height in metres squared – is below 17.5 kg/m2. In younger people, the

diagnosis may be made in those who fail to gain weight during the expected growth

spurt of puberty, as they can become underweight without weight loss.

Weight loss in anorexia nervosa is induced by avoiding ‘fattening foods’, sometimes

supported by excessive exercising or self-induced purging (by vomiting or misuse of

laxatives). As a consequence of poor nutrition, a widespread endocrine disorder

involving the hypothalamic-pituitary-gonadal axis develops, manifest in women by

amenorrhoea and in men by a lack of sexual interest or potency. In prepubertal children,

puberty is delayed and growth and physical development are usually stunted.

The subjective experience of anorexia nervosa is often at odds with the assessment of

others. The conviction that weight control is desirable is usually strongly held,

particularly when challenged and others are seen as mistaken in believing the person

should gain weight, particularly where there is a marked disturbance of body image.

Weight loss is experienced as a positive achievement and, therefore, may be strongly

reinforcing to someone with low confidence and poor self-esteem. As a result, they will

often deny the seriousness of the condition. The essential role of ‘weight phobia’ is

increasingly being questioned however, and is believed by some to be culture specific.

The condition generally starts with dieting behaviour that may evoke no concern.

Indeed, some will experience reinforcing compliments. After a while, however, the previously been features of the person’s personality. A number of secondary difficulties

may develop including physical adverse effects, social isolation, compromise of

educational and employment plans and occupation in the areas of leisure, self-care, daily

living and productivity of employment and/or education. A smaller number will enter

anorexia nervosa through a pattern of purging behaviour without dieting, following a

viral illness, which resulted in weight loss that then became positively valued, or in the

context of a chronic illness such as diabetes or Crohn’s disease.

Typically individuals are persuaded to seek help by concerned family members, teaching

staff or general practitioners with whom they consult about physical consequences.

Sometimes, however, the person begins to appreciate the damaging effects of the

disorder and may seek help in their own right. Children and adolescents are almost

always brought to treatment, very rarely actively seeking help initially and can present

more complex diagnostic challenges (Bryant-Waugh *et al.*, 1992).

2.1.2 Diagnosis

The diagnosis of anorexia nervosa in its typical form is a relatively straightforward one in

older adolescents and adults. The diagnosis has good validity and reliability, the main

obstacle to diagnosis being the person’s own willingness or otherwise to disclose his or

her motives, symptoms and behaviours. Thus, engagement in a supportive, empathic

assessment interview is crucial in enabling the person to reveal fears around weight,

dieting behaviour and any purging or other maladaptive behaviour such as excessive

exercising. In the absence of this engagement, the individual may fail to reveal weightcontrolling

behaviours and collude with the doctor in pursuing physical investigations to

explain the weight loss. In women, the presence of secondary amenorrhoea (i.e.

cessation of menstruation after it has been established) or other physical features of

starvation should always alert the physician to the possibility of this diagnosis. Diagnosis

may be more problematic in children and younger adolescents, as the existing diagnostic

criteria are insufficiently developmentally sensitive (Lask & Bryant-Waugh, 2000).

The diagnosis is made on the basis of the history, supported where possible by a

corroborative account from a relative or friend. Physical examination, with measurement

of weight and height and calculation of body mass index (BMI), can reveal the extent of

emaciation. On occasion, clinical observation during a hospital assessment can enable

characteristic behaviours to be observed. Physical investigations are less useful in making

the diagnosis but are crucial in assessing the physical impact of the disorder and its

complications. Depending on the results of the physical examination, these may include

haematological tests, electrocardiography, radiological assessment and ultrasound

(Royal College of Psychiatrists, 2002).

A diagnostic challenge occurs in those with comorbid physical disorders, such as

diabetes, chronic bowel or thyroid disorder. In diabetes, the patient may be tempted to

restrict insulin intake in order to lose calories, whilst on occasions the symptoms of

organic intestinal disorder may mask the psychological condition.

The weight loss that occurs with the anorexia of depression can usually be distinguished

from that resulting from the dietary control of anorexia nervosa, but the condition can sometimes be difficult to distinguish from post-viral and other chronic fatigue

syndromes where food intake is poor. Weight loss and limited food intake secondary to

a brain tumour are also known to have been mistaken for anorexia nervosa.

1.3 Physical and social consequences

Although in the acute stages of anorexia nervosa subjective distress may be limited,

emotional disturbance is common, chiefly comprising anxiety and mood symptoms.

With time, emotional difficulties usually increase along with a range of physical and

social difficulties, including becoming unable to care for oneself adequately, reducing or

stopping leisure activities, interrupting educational goals and losing personal autonomy.

These affect the person’s quality of life and increase the reliance on and the importance

of the eating disorder.

Depression is a common comorbid diagnosis, with rates of up to 63 per cent in some

studies (Herzog *et al.*, 1992), while obsessive-compulsive disorder (OCD) has been found

to be present in 35 per cent of patients with anorexia nervosa (Rastam, 1992).

Physical problems can be classified as those due to the effects of starvation and the

consequences of purging behaviour. Starvation affects every system in the body. In the

musculo-skeletal system, this will be evident as weakness, loss of muscle strength (which

also affects heart muscle), loss of bone density and impairment of linear growth. Young

women with anorexia nervosa are at increased risk of bone fractures later in life (Lucas

*et al.*, 1999). The effects on the endocrine system have their impact on target organs,

causing infertility, a risk of polycystic ovaries and loss of bone mineralisation. Where

pubertal development has not been completed, incomplete development of secondary

sexual characteristics may occur (Goldbloom & Kennedy, 1995) and permanent stunting

of growth is common. Patients with anorexia nervosa have disorders in the reproductive

hormones (low LH and FSH), suppressed TSH, growth hormone resistance and raised

corticol levels. The effects of purging are described in Section 7.5.2, including long-term

disabilities such as erosion of tooth enamel sometimes amounting to destruction of the

whole dentition. Worn painful teeth can be a considerable concern to the patient in

terms of comfort, appearance and, therefore, self-esteem.

Brain volume is reduced in anorexia nervosa (Dolan, Mitchell & Wakeling, 1988; Kohn *et*

*al.*, 1997; Kingston, Szmukler, Andrewes, Tress & Desmond, 1996; Krieg, Pirke, Lauer &

Backmund, 1988; Swayze *et al.*, 1996). There are two small longitudinal studies, which

have examined the structural changes in the brain of adolescents after full weight gain

(Golden *et al.*, 1996; Katzman *et al.*, 1996). Both found persistent deficits in grey matter

(cell bodies of neurons and glial cells) although there was recovery of white matter

(mainly myelinated axons). This supports the finding of grey matter deficits in people

who have made a full recovery from their eating disorder (Lambe, Katzman, Mikulis,

Kennedy & Zipursky, 1997). One post-mortem study reported that there was a reduction

in basal dendritic fields and dendritic spine density (Neumarker *et al.*, 1997).

Many of the cognitive deficits in anorexia nervosa are restored after weight recovery.

However, some abnormalities in executive function remain after weight restoration. For

example, people with eating disorders have scores greater than one standard deviation

from the norms on tests of perceptual rigidity, perseveration and set shifting and the

neurological sign dysdiadokinesis (Tchanturia, Morris, Surguladze & Treasure, 2002). Although little is known of the effects short or long term of extreme weight loss on

brain development and function in children, it is possible that such weight loss may have

both short and long-term effects on cognitive functioning.

Social difficulties may result in continued dependence on family of origin into adult life

and often include difficulties engaging in intimate relationships. Employment prospects

may be adversely affected either because of the limitations of the disorder or the

disruption caused by lengthy hospitalisations.

1.4 Course and prognosis

The course of anorexia nervosa is very variable. There is no good evidence on the

prognosis for people with anorexia nervosa who do not access formal medical care

(Treasure & Schmidt, 2002). A summary of 68 treatment studies published before 1989

with a length of follow-up of one to 33 years, found that 43 per cent of people recover

completely, 36 per cent improve, 20 per cent develop a chronic eating disorder and

five per cent die from anorexia nervosa (Steinhausen, 1995). The overall mortality in

these long-term studies ranged from 0–21 per cent from a combination of physical

complications and suicide. The all-cause standardised mortality ratio anorexia nervosa

has been estimated at 9.6 (95 per cent Confidence Interval 7.8 to 11.5) Nielsen (2001),

about three times higher than other psychiatric illnesses. The average annual risk of

mortality has been calculated at 0.59 per cent per year in females averaged from 10

samples, with a minimum follow-up of six years (Neilsen *et al.*, 1998). The mortality rate

appears to be higher for people with lower weight during their illness and those

presenting between 20 and 29 years of age.

A number of those with anorexia nervosa progresses to other eating disorders, particularly

bulimia nervosa, but also binge eating disorder, highlighting the relationship between the

disorders. Movement in the other direction is less common, but a number of those with

anorexia nervosa gives a premorbid history of obesity in childhood or adolescence.

1.5 Anorexia nervosa in children and adolescents

Although the essential psychological features are similar, children and younger adolescents

may present with delayed puberty or stunted growth as well as weight loss. Parents or

teachers are generally the ones who raise concern and the young person may resist

medical attention. Some young people will voice anxieties around unwanted aspects of

development, particularly if they have experienced early puberty or feel unable to engage

with their peers’ increasing adolescent independence and social experimentation. In some,

bullying or teasing about weight may have provoked this concern.

Although the principles of making the diagnosis are the same as in adults and are often

straightforward, the greatest diagnostic difficulty occurs in the youngest cases. In

children between the ages of around eight and 12, the condition is less common than in

older individuals and should be distinguished from other types of eating disturbance

seen in middle childhood, such as selective eating and food avoidance emotional

disorder. By definition feeding disorder of infancy and childhood has onset below age

six. In pubescent cases with primary amenorrhoea, it can sometimes be difficult to judge

whether puberty has been delayed from the normal variation in timing of puberty.

Reference to height and weight centile charts is useful in evaluating weight in

comparison to height. It is particularly helpful to compare presenting centiles for weight

and height with historical values, as these may identify stunting of height (where the

young person has crossed height centile lines). The result of such stunting is that the

person may not appear unduly thin, though his or her weight may be considerably

below the projected level as indicated by premorbid height and weight. It is also helpful

to plot body mass index on BMI centile charts, as BMI norms are not stable over age.

Average BMI increases with age during childhood and adolescence, a BMI of 17.5 kg/m2

being close to the mean for a child at the age of 12 (Cole *et al.*, 1995).

In children and adolescents with atypical presentations of an eating disorder,

consideration should be given to the possibility of separate underlying physical

pathology. In these circumstances the involvement of a paediatrician should be

considered.

The prognosis for children and adolescents with anorexia nervosa is variable. Some

(particularly those with a rapid and early onset) will make a full recovery from a first

episode. This is most likely where early physical and psychosocial development has been

healthy and where there is an identified precipitating negative life event such as

bereavement (North *et al.*, 1997). In such cases and where onset is pre-pubertal, physical

consequences such as stunted growth and pubertal delay are usually fully reversible.

Others with a more insidious onset, with earlier social difficulties or abnormal

personality development, may go on to have a more chronic course into middle age

(Gowers *et al.*, 1991).

2 Bulimia nervosa

2.1 Symptoms, presentation and pattern of illness

Bulimia nervosa is characterised by recurrent episodes of binge eating and secondly by

compensatory behaviour (vomiting, purging, fasting or exercising or a combination of

these) in order to prevent weight gain. Binge eating is accompanied by a subjective feeling

of loss of control over eating. Self-induced vomiting and excessive exercise, as well as the

misuse of laxatives, diuretics, thyroxine, amphetamine or other medication, may occur. As

in anorexia nervosa, self-evaluation is unduly influenced by body shape and weight, and

there may indeed have been an earlier episode of anorexia nervosa. The diagnosis of

anorexia nervosa is given precedence over bulimia nervosa; hence in bulimia nervosa BMI is

maintained above 17.5 kg/m2 in adults and the equivalent in children and adolescents (see

Section 2.1.5). There is some controversy concerning whether those who binge eat but do

not purge should be included within this diagnostic category. The ICD10 criteria (WHO,

1992) stress the importance of purging behaviour on the grounds that vomiting and

laxative misuse are considered pathological behaviours in our society in comparison to

dieting and exercise. The DSM-IV criteria (APA, 1994) agree about the importance of

compensatory behaviour but distinguish between the purging type of bulimia nervosa in

which the person regularly engages in self-induced vomiting or the misuse of laxatives,

diuretics or enemas, from the non-purging type in which other inappropriate

compensatory behaviours such as fasting or excessive exercise occur but not vomiting or

laxative misuse.

People with bulimia nervosa tend to not disclose their behaviour nor to seek out

treatment readily although may be more likely to do so than those with anorexia

nervosa. The condition appears to be subjectively less ‘valued’ than anorexia nervosa;

indeed binge eating and purging are commonly associated with extreme subjective guilt

and shame. These emotions are sometimes reinforced by the pejorative language used

by relatives and others including some clinicians, who may refer to ‘confessing’ or

‘admitting’ to purging behaviour. A person’s ambivalence towards treatment often arises

from the fear that they will be stopped from vomiting and purging and then left to face

the consequences of their binge eating, i.e. excessive weight gain.

The condition usually develops at a slightly older age than anorexia nervosa (the mean

age of onset is 18 to 19, compared to 16 to 17 for anorexia nervosa). Bulimia nervosa

sometimes arises from a pre-existing anorexic illness. Where this is not the case the

development of the disturbance is often essentially similar to that of anorexia nervosa,

arising from a background of attempts to restrain eating. In bulimia nervosa however,

dietary restriction cannot be maintained and is broken by episodes of reactive binge

eating, which result from a combination of physiological and psychological factors.

Compensatory behaviours follow in order to counteract the effect of binge eating on

weight. The person, therefore, maintains a weight, usually within the normal range

despite overeating but commonly progresses into a vicious cycle of attempted dieting,

binge eating and compensatory purging, frequently on a daily basis. As these behaviours

dominate daily life, the person becomes preoccupied with thoughts of food and life may

be re-organised around shopping, eating and purging behaviour. Initially, those with

bulimia nervosa are generally secretive about their bulimic episodes, though some may

leave obvious signs of their disorder such as empty food packaging and occasionally

bags of vomit for other family members to discover.

Bulimic episodes are frequently planned, with food purchased or prepared in order to be

consumed without interruption. The individual may also avoid situations in which they

are likely to be exposed to food or will find it difficult to control their eating, such as

when eating out with others. This avoidance behaviour tends to add to any social and

relationship difficulties that may be present.

Mood disturbance is extremely common in bulimia nervosa and symptoms of anxiety

and tension are frequently experienced. Self-denigratory thoughts may develop out of

disgust at overeating or purging whilst low self-esteem and physical self-loathing may in

some be rooted in the past experience of physical or sexual abuse. Self-harm, commonly

by scratching or cutting, is common. A significant proportion of those with bulimia

nervosa have a history of disturbed interpersonal relationships with poor impulse

control. Some will abuse alcohol and drugs.

2.2 Diagnosis

As in anorexia nervosa, the diagnosis depends on obtaining a history supported, as

appropriate, by the corroborative account of a parent or relative. This will require an

empathic, supportive, non-judgemental interview style in which the person is enabled to

reveal the extent of his or her symptoms and behaviours. Although those with bulimia

nervosa generally have fewer serious physical complications than those with anorexia

nervosa, they commonly report more physical complaints when first seen. They may

complain of fatigue, lethargy, or feeling bloated, and they may suffer constipation,

abdominal pain and on occasions swelling of the hands and feet or irregular

menstruation. There may also be erosion of dental enamel in which the lingual surface

of the upper teeth is mainly affected, and it has been argued that this is virtually

pathognomonic of vomiting (Mitchell, 1995).

Physical examination is often normal, though the salivary glands (particularly the parotid

glands) may be enlarged. Calluses on the back of the hand may be found; these result

from the use of the hand to stimulate the gag reflex and induce vomiting. Oedema is

common in those who have used laxatives or diuretics whilst these behaviours also lead

to fluid loss and subsequent dehydration, which in turn may result in a metabolic

alkalosis. This is generally accompanied by hypochloraemia and hypokalaemia. Overall,

about 10 per cent of those with bulimia nervosa have electrolyte abnormalities detected

on routine screening (Mitchell, 1995). Metabolic acidosis can also occur in patients who

are abusing laxatives as a result of the loss of bicarbonate from the bowel. Less

commonly hyponatraemia, hypocalcaemia, hypophosphataemia and hypomagnesaemia

may develop.

In the gastrointestinal system, oesophagitis may occur. Gastric dilatation that poses the

risk of gastric rupture and death occurs rarely but may be the most common cause of

fatality (Mitchell, 1997). Constipation is extremely common, mainly due to dehydration;

steatorrhoea and protein losing gastroenteropathy have also been reported secondary to

laxative abuse. Severe use of laxatives has been reported to cause cardiomyopathy and

other types of myopathic disorder as a result of storage of Ipecac in muscle tissue

(Mitchell, 1995). Electrocardiography may indicate heart conduction abnormalities and

possibly an increase in the risk of mitral valve prolapse.

Abnormal electroencephalographic (EEG) findings have been reported probably

secondary to fluid and electrolyte abnormalities; there does not seem to be an

association between bulimia nervosa and epilepsy.

Endocrine abnormalities are variable. These include abnormalities in the menstrual cycle

and blunting of the thyroid stimulating hormone and growth hormone response to

thyroid releasing hormone.

2.3 Impairment and disability

There is considerable overlap between the long-term disabling consequences of bulimia

nervosa and those of anorexia nervosa. Mood and anxiety symptoms are very common.

These symptoms, low self-esteem and body image disturbance can all have a negative

effect on social relationships, which in turn may be damaged by a lifestyle that may be

chaotic and characterised by impulsivity.

The adverse physical consequences of purging behaviour have been identified in Section

7.5.2. In addition, those with bulimia nervosa may be at risk from the effects of

alternating weight loss and weight gain.

Lissner *et al.* (1991) in a series of 3130 participants found that both all-cause and

coronary heart disease mortality were increased significantly in both men and women

with high levels of weight variability. Morbidity from coronary heart disease was also

increased.

Estimates of the prevalence of the diagnoses of personality disorder in people with

bulimia nervosa have ranged from 21 per cent to 77 per cent. Obsessive-compulsive and

avoidant personality disorders (Cluster C) have been described frequently (Braun *et al.*,

1994). The relationship of borderline personality disorder to bulimia nervosa has been a

source of considerable debate (Wonderlich, 1995) with reported rates ranging from

two per cent to 47 per cent, these rates apparently influenced by subject and

measurement variability across studies, thus BPD probably occurs no more frequently

than other PDs in bulimia nervosa.

2.4 Course and prognosis

There have been few studies with a lengthy follow-up period of the course and outcome

of bulimia nervosa in the community. Many people with bulimia nervosa are not

receiving any form of help (Hsu, 1995). Of these, the majority will suffer chronicity or a

relapsing course, maintained by over-valued belief in the importance of appearance and

thinness in particular (Fairburn *et al.*, 2000). With the most effective treatments about

50 per cent of people with bulimia nervosa can be expected to be asymptomatic two to

10 years after assessment. Twenty per cent are likely to continue with the full form of

bulimia nervosa whilst the remainder (30 per cent) have a course of illness characterised

either by remissions or relapses or persistent but subdiagnostic bulimia (Hsu, 1995).

One 10-year follow-up study of 50 people with bulimia nervosa found that 52 per cent

had fully recovered and only nine per cent continued to experience symptoms of bulimia

nervosa (Collings & King, 1994). A larger study of 222 followed-up for a mean of 11

years revealed that 11 per cent still met criteria for bulimia nervosa whereas 70 per cent

were in full or partial remission (Keel *et al.*, 1999).

There are few consistent predictors of longer-term outcome, though a number of studies

have shown strong associations between weight fluctuation (which commonly occurs in

bulimia nervosa) and negative health outcomes. Patients with the uni-impulsive form of

the illness without additional control difficulties are also thought to do better. The

mortality rate associated with bulimia is uncertain but may be higher than in the

matched general population (Hsu, 1995).

2.5 Bulimia nervosa in children and adolescents

The full syndrome of bulimia nervosa is rarely seen in young people under the age of 14

(Bryant-Waugh & Lask, 1995). Indeed in these authors’ uniquely specialised clinic fewer

than five per cent of children under the age of 14 presenting with eating disorders were

diagnosed with bulimia nervosa. Where these presentations occurred the clinical features

were the same as found in the older age group. Young people with this disorder were

also, in common with adults with the same diagnosis, depressed and suffered poor self

image (Bryant-Waugh & Lask, 1995). There is no substantial literature on treatment or

outcome in this age group. Adolescents with bulimia nervosa may sometimes be

considered to be suffering incipient personality disorder, though caution should be

exercised in diagnosing personality disorder before development is completed. In these

cases it can be difficult to judge whether the eating disorder is contributing to abnormal

personality development or conversely, if the personality difficulties have acted as a risk

factor for the development of bulimia nervosa.

Since bulimia nervosa is very rarely seen in children and uncommonly in younger

adolescents, and there has been no research on the treatment of adolescents with bulimia

nervosa, in line with much current clinical practice and taking into account the above

issues, the GDG took the view that, subject to adaptation to their age, circumstances and

level of development, adolescent patients with bulimia nervosa should receive the same

type of treatment as adults with the disorder along with appropriate family involvement.

3 Atypical eating disorders (eating disorders not

otherwise specified; EDNOS) including binge eating

disorder

3.1 Symptoms, presentation and pattern of illness

A number of people suffer from eating disorders that closely resemble anorexia nervosa

and bulimia nervosa, but which are considered atypical, as they do not meet the precise

diagnostic criteria for these conditions (Fairburn & Harrison, 2003; Turner & Bryant-

Waugh, 2003; Ricca *et al.*, 2001). In Europe, these are often termed ‘atypical eating

disorders’ (Fairburn & Harrison, 2003), the equivalent American term being ‘eating

disorders not otherwise specified’ (American Psychiatric Association, 1994). For example,

the patient’s weight might be just above the diagnostic threshold for anorexia nervosa

or she might still be menstruating. Binge eating and purging may occur less frequently

than specified for a diagnosis of bulimia nervosa. Over concern with weight and shape is

generally present in these disorders, although in some the primary focus is on

maintaining strict control over eating. Although the diagnostic criteria may not be met,

many atypical eating disorders are as severe and long lasting as anorexia nervosa and

bulimia nervosa. Patients’ treatment needs and prognosis may be virtually identical.

Binge eating disorder (BED) is a recently described condition, first defined as a research

category in DSM-IV (APA, 1994), though there is some overlap with the ICD 10 category

‘Overeating associated with other psychiatric disturbance’ (F50.4) (WHO, 1992). In BED,

individuals engage in uncontrollable episodes of binge eating but do not use

compensatory purging behaviours. These binge eating episodes are associated with

three or more of the following:

● Eating much more rapidly than normal

● Eating until feeling uncomfortably full

● Eating large amounts of food when not physically hungry

● Eating alone through embarrassment at the amount one is eating

● Feeling disgust or extreme guilt after overeating.

Marked distress regarding binge eating is present and social avoidance is common.

3.2 Diagnosis

Atypical eating disorders are conditions of clinical severity that do not conform to the

diagnostic criteria for anorexia nervosa or bulimia nervosa. An example would be someone

with extreme dietary restraint, who exercised excessively to control weight, which was

maintained in the low normal range. This condition would also include those with the

features of anorexia nervosa at low weight who are still menstruating. Many people with

atypical eating disorders have suffered with anorexia nervosa or bulimia nervosa in the past.

In comparison with anorexia nervosa and bulimia nervosa, far less is known about binge

eating disorder (Fairburn & Harrison, 2003). Apart from binge eating, its systematic

profile overlaps little with the other eating disorders. Defining binge eating can be

problematic and there may be a discrepancy between the subjective experience and

clinical assessment of a binge. The onset of binge eating disorder is usually in the

teenage years or early 20s, but people tend to present later, typically in their 30s or 40s,

when they have become overweight or obese. The sex ratio is more even and binge

eating generally occurs against a background of a tendency to overeat rather than of

dietary restraint. Many people with binge eating disorder are obese. By definition selfinduced

vomiting and laxative misuse are not present or only occasionally present.

Depressive features and dissatisfaction with shape is common, though over-evaluation of

the importance of weight and shape is less marked than in bulimia nervosa.

As with the other eating disorders, the diagnosis of binge eating disorder is made on the

basis of the history, with physical investigations being used to assess any physical

consequences. Observation and assessment in hospital is rarely indicated, though where

available as part of a day programme, assessment of meal preparation and eating may

be a useful adjunct to treatment planning.

3.3 Impairment and disability

Where atypical eating disorders are similar to the full syndromes of anorexia and bulimia

nervosa, the physical dangers and psychosocial impairments closely resemble those of

the diagnostic conditions. Many of those with BED will suffer similar physical

complications of binge eating to those with bulimia nervosa, though purging carries

greater physical risk than binge eating. Those who are obese are at risk of the

psychological and physical disabilities associated with this condition, namely low selfesteem,

diabetes, heart disease, hypertension and stroke.

3.4 Course and prognosis

The prognosis for those disorders that resemble anorexia nervosa and bulimia nervosa

depends on the severity of the associated physical and psychological features. Those

with binge eating disorder typically give long histories of proneness to binge eating but

these may alternate with extended periods free from binge eating. The spontaneous

remission rate may be high (Fairburn *et al.*, 2000). Short-term response to treatment

appears better than for anorexia nervosa and bulimia nervosa but there are as yet no

studies of long-term course or outcome. There is a certain amount of movement from

one diagnosis to another, thus those with atypical eating disorders may go on to

develop bulimia nervosa, or more rarely anorexia nervosa.

3.5 Atypical eating disorders in children and adolescents

Atypical eating disorders are relatively commonly diagnosed in childhood, in part because

of the difficulty in strictly applying existing diagnostic criteria for anorexia nervosa and

bulimia nervosa, and in part because of an ongoing lack of clarity about the classification

of eating disturbances in this age group. Children and younger adolescents may present

with a range of other types of clinical eating disturbance, which may be different in terms

of psychopathology to anorexia nervosa/bulimia nervosa presentations (Cooper *et al.*,

2002). They frequently develop in those who have suffered feeding disorders of childhood

and sometimes the distinction (particularly in middle childhood) can be a difficult one.

Some have suggested that atypical eating disorders probably occur more commonly

(Bryant-Waugh, 2000) and some have suggested modification to the ICD10 criteria for the

diagnosis of eating disorders in children is required. Binge eating disorder has not been

systematically investigated in this age group.