CHAPTER 14

EATING DISORDERS

“You can never be too rich or too thin.”

Duchess of Windsor (1896-1986)

Asceticism

Asceticism is a lifestyle in which the individual refuses worldly pleasures. The ascetic believes their chosen lifestyle is virtuous; their aim is usually to achieve greater spirituality. Asceticism has been practiced in all religions and by some non-religious individuals [who claim the practice increases the ability to think clearly and avoid destructive short term impulses].

St Catherine of Siena (1347-80) was an ascetic. Her story may be the first recorded case of anorexia nervosa (Bell, 1985; Reda & Sacco, 2001). St Catherine was an influential figure, convincing Pope Gregory XI to restore the papacy from Avignon (Fr.) to Rome. She was later in dialogue with Pope Urban VI.

Illustration. St Catherine of Siena. Possibly the first described case of anorexia nervosa.

At 16 years of age St Catherine was admitted to the Third Order of St. Dominic (but was allowed to remain living at home). For three years she lived on a spoonful of herbs per day and slept only two hours per night. When her mother insisted that she eat, St Catherine began to throw meat under the table. She lost half her weight. When the local priest, Don Tommaso of Fonte persuaded her “in the name of God” to eat at least once per day, she began to vomit. She was in the habit of prodding her throat with a stick of finnochio or a goose feather. St Catherine had been in conflict with her family over a proposed marriage, she was perfectionistic and was never satisfied with the results she achieved. She died at 33 years of age.

St Wilgefortis lived some centuries earlier. The case for her being the first recorded case of anorexia nervosa is less strong (Lacy, 1982). St Wilgefortis lived some time between 700 and 1000 AD; details are sketchy. She was the daughter of the King of Portugal. She had decided to enter the church. Her father arranged for her to marry the King of Sicily. To avoid the marriage St Wilgefortis prayed to God to be made unattractive. She became ascetic and ate little. She developed body hair and grew a beard. The King of Sicily withdrew his suit. He father was so outraged he had her crucified. Lanugo, (fine baby-like hair) is frequently observed on the face and limbs of patients with anorexia nervosa – but, not a beard.

Illustration. The statue of St Wilgefortis in Henry VIII's Chapel, Westminster Abbey, depicts her with a beard flowing down to her chest. In the above Polish depiction, she is beardless, but very thin.

Introduction to eating disorders

The DSM-IV recognises three eating disorders: 1) Anorexia nervosa, 2) Bulimia nervosa, and 3) Eating disorder not otherwise specified (which includes Binge-eating disorder).

Anorexia nervosa was first described in English as a medical condition by Sir William Gull in 1874. He drew attention to the diagnostic triad of 1) fasting, 2) amenorrhea and 3) hyperactivity. He described the disorder as "wasting without lassitude".

The diagnosis of bulimia nervosa first appeared less than 30 years ago (Russell, 1979) as a variant of anorexia nervosa, in which there is dietary restriction, episodes of overeating, vomiting or laxative use, and the maintenance of about normal weight.

Binge-eating disorder is yet to be fully characterized, but features recurrent episodes of binging in the absence of dietary restriction or other compensatory behaviours (except, in some cases, vomiting). It has been reported in 10-15% of female college students (Halmi et al, 1981).

ANOREXIA NERVOSA (AN)

DSM-IV Diagnostic criteria

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of a body weight less than 85% of that expected)
B. Intense fear of gaining weight or becoming fat, even though underweight.
C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
D. In postmenarche females, amenorrhea (the absence of at least three consecutive menstrual cycles).

AN is the most homogenous of all psychiatric disorders. There is a strong gender preference, and a stereotypic presentation and course of illness. There is resistance to eating, powerful pursuit of weight loss, but paradoxically, there is preoccupied with food and eating rituals. There is distorted body image, denial of being underweight, a practice of energetic exercise, a lack of insight and resistance to treatment.

The etiology is unclear, and the treatment options are of limited efficacy.

The **medical approach** to AN must be informed by the **socio-cultural** reality in which the disorder develops (the pressure to be slim and beautiful).

The **prevalence** of AN depends on the assessment tools and the population surveyed. The American Dietetic Association gives a prevalence of 0.3% for men and 0.9% for women, but that of 12-15 year olds females, 12% experienced some form of eating disorder.

In ballet and modelling schools the prevalence is about 7% (Garner & Garfinkel, 1980).

AN is 10 times more common in females (not exactly consistent with the prevalence numbers above). The most common age of onset is 14-18 years, but has been reported in girls as young as 8 years. It is believed to be more common in the higher socio-economic classes. However, this has not been clearly demonstrated in epidemiological surveys. It is believed the incidence has increased over the last half century (Bulik et al, 2006; Hoek, 2006).

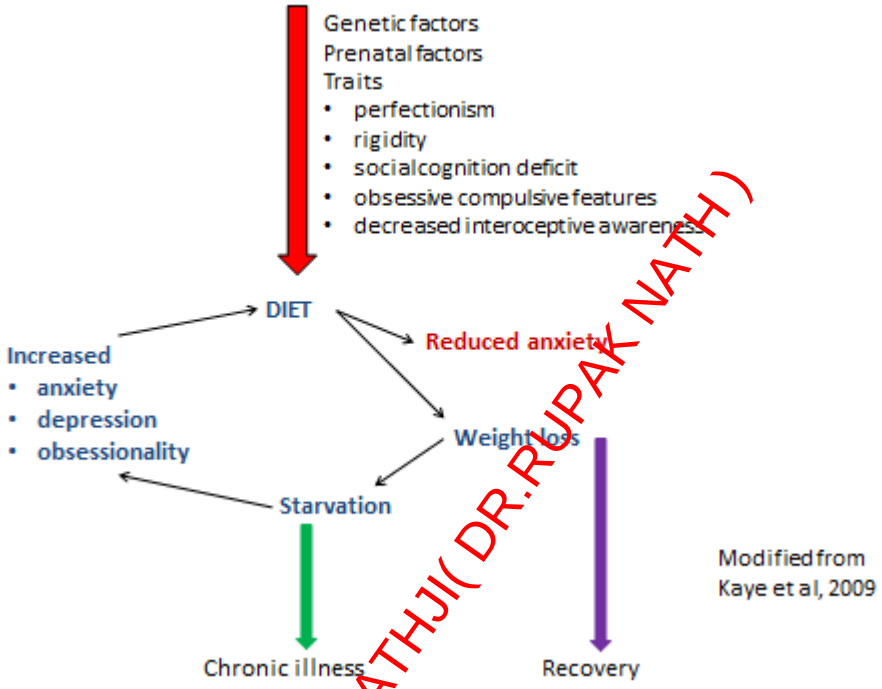

Illustration. See text for details. Dieting reduces anxiety in the short term, but begins a destructive cycle of increasing anxiety, depression and obsessionality, and further dieting.

Aetiology

As mentioned, the etiology of AN is unclear. Evidence suggests a genetic predisposition, and important contributions from culture, early life experience, personality type and the neurological and hormonal changes associated maturation and the stressors of adolescence (including increasing autonomy).

Genetic studies. The lifetime risk of AN is 7-12 times greater for individuals indicate a substantial genetic contribution to AN. Twin studies have estimated the heritability of AN to be 46-70% (Kendler et al, 1991) and 33-84% (Bulik & Tozzi, 2004). Recent twin studies found heritability due to genetics 56%, shared environment 5% and unique environment 38% (Bulik et al, 2006).

The gene-environment interaction model views strict dieting as sufficient “insult” for expression of the phenotype (Bulik, 2005).

Others argue that genetic factors could be “switched on” by the hormonal changes and the particular stressors of puberty (Herpertz-Dahlman et al, 2011).

Any biological factors which are "switched on" during puberty and adolescents arrive at a time when the brain is continuing to mature (dendrite pruning and myelination).

Pregnancy and **perinatal complications,** especially preterm birth are risk factors.

The evidence for **childhood sexual abuse** as an aetiological factor in AN is inconclusive (Wonderlich et al, 1997).

Adverse life events my precipitate AN. Severe life events are found in the majority of patients with onset after 25 years, and a minority of those with onset before 25 years.

Personality disorder is found in at least 70% of those with AN. Obsessive-compulsive personality disorder is the most common.

Personality traits are characteristic, and may represent risk factors, or an underpinning pathological process. Herpertz-Dahlmann et al (2011) describe patients as "usually good and successful", however, often with "some peculiarities". They describe rigidity and perfectionism, depression, anxiety. Interpersonal function may be reduced with non-assertive, submissive interpersonal style, poor theory of mind, and autistic traits, while Ahren et al (2011) find that these patients characteristically evaluate themselves by comparison with others.

Patients with AN also appear to demonstrate diminished "interoceptive awareness". Interoception includes a range of sensations beyond taste, including the perception of pain, temperature, itch, tickle, sensual touch, muscle tension, air hunger, stomach pH and intestinal tension. Integration of these provides a sense of the entire body and the self – provides a link between cognitive and affective processes and the current body state.

Altered interoceptive awareness might be a precipitating and reinforcing factor in AN.

Many such personality features improve with remission of the condition. However, tendency to negative emotional states, harm avoidance, perfectionism, desire for thinness and mild dietary preoccupation persist – suggesting these are underlying traits which contribute to the pathogenesis of AN (Kaye et al, 2009). At the same time, it must be remembered that such features are exaggerated by starvation (Keys et al, 1950).

Anxiety and dysporic mood. Evidence suggests that individuals with AN experience anxiety, and that dietary restraint reduces this unpleasant state, and that eating stimulates dysphoric mood (Kaye et al, 2009). (This may be underpinned by altered serotonin function.) Others state that dieting counteracts feeling of worthlessness (Herpertz-Dahlmann et al, 2011).

Socio-cultural factors have shaped "the modern cult of thinness" in Western societies. It is believed individuals respond by dieting, and that genetically vulnerable individual's progress through dieting to AN (Bulik, 2005).

Evidence suggests a culture bound syndrome, as AN is rare in Asia and developing countries - although this pattern is probably changing (Jennings et al, 2006).

Socio-economic status. Earlier reports found that the prevalence of AN was higher in higher socio-economic schools, but the evidence is now equivocal. Recent studies, however, indicate the risk for hospitalization for NA is related to mothers level of education (higher risk is associated with higher maternal education; Ahren et al, 2011). Perhaps such mothers are more demanding of their daughters.

Maintaining factors

Mentioned under etiology, the relief of anxiety by dieting and dysphoric mood cause by eating, may also serve as a maintaining factor.

Starvation is another maintaining factor, inducing complex physiological and psychological reactions involving central and peripheral mechanisms. Such mechanism may have had evolutionary value, allowing animals to survive periods of food shortage, but in the current setting they serve only to perpetuate a vicious cycle of weight loss.

Neuropsychological testing

Neuropsychological testing reveals **cognitive deficits** which are related to severity of the disorder, and may play a role in cause and outcome (Zakzanis et al, 2009). Executive control is impaired with problems in set-shifting, attention and decision-making (Treasure & Russell, 2011). A link has been demonstrated between amenorrhoea, brain structure and deficits in cognition, including recall, verbal fluency, working memory, visual reproduction, reading, maths and oral language (Chui et al, 2008).

Neuroimaging

Neuroimaging studies demonstrate structural and functional abnormalities. However, more work is needed to characterize them definitively.

There is global reduction of grey (GM) and white matter (WM) during the acute stage. GM is reduced by 5-20%, and WM is reduced to a lesser extent.

GM loss is found in the anterior cingulate, hippocampi and the temporal, parietal and prefrontal regions. GM loss correlates with serum cortisol. With recovery GM is restored, but most studies find small residual deficits remain. In one study, there was 60% restoration after 15 weeks of successful treatment.

A recent diffusion tensor imaging study (DTI; Kazlouski et al, 2011) revealed WM abnormalities in the fornix, fronto-occipital fasciculus and the posterior cingulum.

Magnetic resonance spectroscopy (MRS), which gives information on nerve cell damage by assessing brain metabolites, indicates altered cell membrane turnover which is reversible with recovery.

Functional magnetic resonance imaging (fMRI) using visual stimuli of food or body image suggest involvement of the prefrontal, parietal and cingulate cortices. fMRI combined with a set shifting task (Zastrow, 2009) demonstrated hypo-activity of the ventral anterior cingulated-striato-thalamic loop, and hyper-activity of frontoparietal networks. fMRI combined with an emotional words test (Pringle et al, 2011) demonstrated hypo-activity in parietal, occipital and limbic structures (including the amygdala).

These data can be difficult to comprehend. The brain has been described as containing a ventral [limbic] neuro-circuit that includes the amygdala, insula, ventral striatum, ventral regions of the ACC and the orbitofrontal cortex, which is important in identifying the emotional significance of stimuli and for generation an affective response to these stimuli. And a dorsal [cognitive] neuro-circuit that includes the hippocampus, dorsal regions of the ACC, dorsolateral prefrontal cortex and parietal regions, which is important in modulating selective attention, planning and effortful regulation of affective states.

In AN there is hypo-activation of the ventral circuit, which would be consistent with difficulty in appropriate emotional response to immediately salient stimuli, and hyper-activity in the dorsal circuit, which would be consistent with increased concerned with planning and consequences.

The insula may be of particular importance (Kaye et al, 2009), as it integrates interoceptive information – confirmation is awaited.

Neurotransmitters and cells

Receptor imaging studies show reduced 5HT-2A receptor binding in the acute stage, which persists after recovery (Frank et al, 2004). The 5HT-1A receptor binding potential is increased in AN (decreased in depression), suggesting a disordered serotonin system in this disorder.

On the topic of neurotransmitters, dopamine (DA) dysfunction in AN, particularly in striatal circuits, may contribute to altered reward centre responses.

Loss of GM volume is probably due to reduction in the size of neuronal and glial cell bodies, and reduction in the density of dendrites and synapses – as restoration of volume occurs with remission.

The clinical picture

The clinical picture is embodied in the DSM-IV diagnostic criteria listed above.

The patient is usually a teenage female, brought in by her parents. There has been weight loss, cessation of the menses, fine hair growth on the face and limbs, refusal to eat in the manner expected for her age and family circumstances, particular avoidance of carbohydrate and fatty foods, frequent weighing, often vomiting and excessive laxative use, insomnia, irritability, sensitivity to cold, and withdrawal from friends.

The hands and feet are cold, the skin is dry, the pulse is slow (50-60/min) and the blood pressure is low (e.g., 90/60). There may be calluses on the dorsum of the second and third digits through frequent contact with the front teeth and erosion of tooth enamel due to regurgitated gastric acid. There may be disorder of hormones, including cortisol, gonadal and thyroid hormones.

History taking and subsequent management may be difficult. 'Denial of the illness, lies, cheating, manipulation, are characteristic of the behaviour of anorectics', states an author cited by Russell (2000).

The patient frequently believes that overweight is indicative of greed and deserving of social ostracism. She frequently maintains that she is overweight, in spite of evidence to the contrary. There is a fear of gaining weight. Traditionally, terms such as 'distorted body image', 'over-valued ideas' and 'irrational beliefs' have been used, although similarities to delusional thinking have been noted. [A recent study (Steinglass et al, 2007) found that 20% of 25 AN patients were delusional, and suggested a sub-group of AN patients whose concerns reach delusional proportions may be more refractory to treatment. This view may not be widely accepted.]

Depression is present in over 40%, and obsessive-compulsive disorder in over 20% of cases.

Gonadotrophins and oestrogens are low or undetectable. Pelvic ultrasound reveals reduced ovarian size. The 24 hour urinary cortisol is elevated and plasma tri-iodothyroxine is low (Krassas, 2003).

The complications of starvation include fluid and electrolyte disturbance, peripheral oedema, hypoglycaemia, myopathy, osteoporosis and thrombocytopenic purpura. A recent account (Sheu et al, 2007) describes nutritional deficiency affecting both respiratory muscle strength and lung parenchyma, leading to emphysema. Death from medical causes may results from starvation or purging-related arrhythmias.

Outcome

The annual mortality rate is 0.6%.

Full recovery includes return to appropriate weight and continued growth and development, restoration of menstruation, and normal eating behaviour and attitude with regard to food and body shape. A Swedish study (Theander, 1985) followed

patients up over 33 years and reported 18% had died. 29% recovered in less than 3 years, another 35% recovered by 6 years. Recovery after 12 years was rare. An Australian study (Wade et al, 2006) followed patients after almost 15 years and found that while 75% had a good outcome, less than 50% were asymptomatic.

At follow up, 20% are unable to support themselves independently (Treasure & Russell, 2011).

40% of AN deaths is by suicide (Papadopoulos et al, 2009). AN has the highest suicide rate of all mental disorders. However, people with AN do not have a higher attempted suicide rate than people with other eating disorders or the general public - they select more lethal means (suggesting a stronger desire to die; Guillaume et al, 2011).

Those who recover may do well in life – they have the ability to plan ahead, control their impulses and avoid harm.

Recovery form AN is much less likely the longer the illness has persisted.

Treatment

As with all eating disorders, a collaborative approach and a multidisciplinary team is essential in the management of AN.

Provocatively, Herpertz-Dahlmann et al (2011) state, “To date, there is no evidence based therapy for AN”. And, there is no FDA approved medication for this condition. Accordingly, a pragmatic approach is taken, frequently with good results.

As mentioned above, early treatment is most important.

Inpatient treatment restores weight most rapidly (usually within three months). There may be difficulty in persuading the patient to remain in an inpatient program. Benefits include the omnipresence of skilled nursing staff who are able to provide psychotherapy and supervision. The patient is encouraged to take nutritious meals at regular meal times. It may be necessary to commence with smaller meals. Once eating is re-established the aim is to gain 200-300 grams per day.

Some experts find that initial in-patient treatment has no advantages over out-patient treatment, and that out-patient failures do poorly on transferred to in-patient facilities (Gowers et al, 2007). It seems where the patient is best managed depends on local factors.

Day hospital treatment is offered in some centres; weight gain is not as rapid as with inpatient care, but the outcomes are probably similar in all other respects.

Family therapy is usually offered and is effective in helping to correct miscommunication and misunderstandings within the family, and restore healthy eating habits (especially for patients less than 19 years of age).

When treatment is available early, family therapy provides and advantage over individual therapy. However, when treatment is delayed, at follow-up, there no advantage of family over individual therapy.

Individual psychotherapy is usually offered, the type being influenced by the theoretical inclinations of the treating centres/clinicians (Hay et al, 2003). The patient remains psychologically inaccessible as long as starvation continues, thus, productive psychotherapy may not begin until there is at least some weight restoration.

Cognitive behaviour therapy (CBT) aims to restructure cognition, but limited success in AN (American Dietetic Association, 2011). A recent unexpected finding (McIntosh et al, 2005) is that non-specific supportive clinical management was superior to specialized psychotherapy (cognitive behaviour therapy and interpersonal psychotherapy).

Dialectical behavior therapy (DBT) is used in the management of borderline personality and focuses on emotional dysregulation and new coping skills. There is now interest in evaluating DBT in AN.

Drug treatment. As mentioned, there is no FDA drug approved for the treatment of AN. Chlorpromazine or olanzapine may reduce distress and stimulate the appetite.

SSRIs are of no value during the weight restoration period, but may be helpful during the maintenance phase.

Antidepressants are indicated for the treatment of major depression, but they are ineffective in the presence of malnutrition.

Compulsory treatment and **naso-gastric feeding** depend on the local mental health legislation. This is generally avoided as it disrupts the trusting patient-clinician relationship and acceptance by the patient of responsibility for her/his actions. However, it become necessary should malnutrition pose an immediate threat to life.

BULIMIA NERVOSA (BN)
DSM-IV diagnostic criteria

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
    1. eating, in a discrete period of time an amount of food that is definitely larger than most people would eat during the same time and in the same circumstances
    2. a sense of lack of control over eating during the episode.
B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications, fasting, or excessive exercise.
C. The binge eating and inappropriate compensatory behaviour both occur, on average, at least twice a week for 3 months.
D. Self-evaluation is unduly influenced by body shape and weight.
E. The disturbance does not occur exclusively during episodes of Anorexia nervosa.

"Bulimia" is derived from the Greek words, bous, for ox, and limos, for hunger – indicating the appetite of an ox.

BN was first described by Russell (1979), and first appeared in the DSM (III) in 1980. The diagnostic features include binge eating followed by abnormal behaviour to avoid weight gain, including vomiting, purging and use of diuretics, fasting and excessive exercise. There are many similarities with AN, such as sustained periods of fasting. The main diagnostic difference is that with BN the weight is maintained about normal. Should the weight fall and remain below 85% of the ideal body weight (IBW) and amenorrhoea appear, the diagnosis becomes AN.

Binge eating (up to 10 000 calories; usually processed carbohydrates and fatty foods) may occur following prolonged fasts or in response to adverse emotional states, including low mood or feelings of rejection. Evidence also indicates that exposure to certain foods can trigger binges (Staiger et al, 2000), suggesting a similarity with drug use behaviour. Patients frequently describe a sense of pleasure during binging, which may indicate an anxiolytic effect. Concurrently, there may be a sense of loss of control, which is unpleasant. Purging (used here to include vomiting) occurs immediately, and may be associated with a sense of relief (erroneous) at having avoided weight gain. Purging is soon followed by a sense of self-disgust, frustration and regret.

BN is more common than AN, with a life-time **prevalence** in Western regions of 0.5% for males and 1.5% for females. Ten times more females than males present for treatment. BN typically develops in late adolescence and early adulthood. It is much more common in countries "where palatable food is plentiful yet thinness is esteemed" (Klein &Walsh, 2003).

Aetiology

As with AN, **socio-cultural factors** are important. In the west, thinness in women has been prized for most of the last century. The "Flappers" of the 1920s are a prime example, and the female "mannequins" and "models" of all eras have been thin. That is not to deny the desirability of the voluptuous "pin-up girls" of the 1950, and the well rounded form of the sensuous type throughout history. Many of those with eating disorders have difficulty with sexuality and this may inspire some to seek the aesthetic form of the mannequin rather than the voluptuous form of the sex idol.

It is not only the female whom Western culture encourages to have a particular shape. In recent decades the ideal male depicted in underpants advertisements is shown to have abdominal muscles which are just as unattainable to the average male as the stick-figure is to the average female.

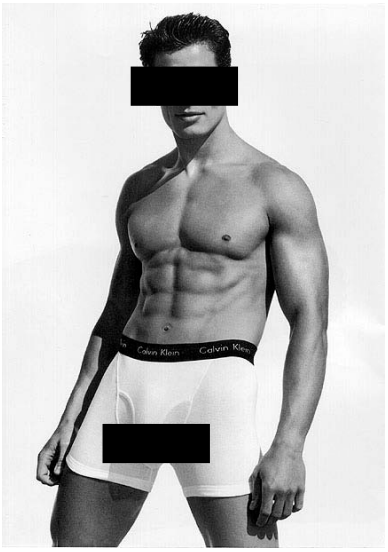

Illustration. Western culture encourages the female to aspire to thinness and the male to be thin but muscular. Both are almost unattainable to people with day-jobs.

Childhood sexual abuse is more common in people with BN than normal control populations. However, it is not specific for BN, and does not occur more commonly in people with BN than in people with other psychiatric disorders. The majority of people with BN have not been exposed to this trauma.

Personality disorder is common in BN, particularly borderline personality disorder. In the absence of frank personality disorder, the temperament frequently features elevated novelty seeking and impulsivity. There is a high prevalence of drug and alcohol abuse, self-injurious behaviour such as cutting, and suicide attempts (Paul et al, 2002).

Genetics
Studies in twins have estimated the heritability of BN as 28-83% (Bulik et al, 2000). There is familial aggregation of AN and BN which suggests a shared vulnerability.

Neuroimaging
Neuroimaging shows similar changes to those of AN: reduced global reduction of GM and WM, and 5HT-2A receptor binding abnormalities (Frank et al, 2004; Goethals et al, 2006).

A recent fMRI study using a food stimulus demonstrated reduced activation of the lateral prefrontal cortex and the anterior cingulate (Joos et al, 2011). (BN is a dysregulation syndrome and the lateral prefrontal cortex is a region of impulse control. BN is often associated with anxiety and distress, and the anterior cingulate plays a role in mood regulation.)

The clinical picture

The clinical picture can be extrapolated from the diagnostic criteria. In contrast to the patient with AN, the patient with BN usually self-presents seeking help. Weight loss is not a prominent feature. Thinness and physical appearance are of great importance to her/him, and self-esteem is judged by this cultural yard-stick. There may be calluses on the dorsum of the second and third digits, erosion of dental enamel, and hypertrophy of the parotid glands. There is rarely disturbance of body chemistry. Arrhythmias occasionally occur secondary to electrolyte disturbances. Menstrual abnormalities are not uncommon, even when the body weight is normal.

Depression and low self-esteem are common. Anxiety disorders and obsessive-compulsive disorder are not uncommon.

Outcome

5-10 years following presentation, 50% have achieved full recovery and 20% continue to meet the criteria for BN. This leaves 30% who experience relapses (Keel & Mitchell, 1997).

Treatment

Psychotherapy is the first line therapy. CBT is used to restructure maladaptive thoughts which underpin the maladaptive behaviour. Dialectical behaviour therapy (DBT) may also have a place.

Antidepressants are the second line of therapy. The selective serotonin reuptake inhibitors (SSRIs) are usually chosen (fluoxetine is the only FDA approved drug for this disorder). They are used in high doses (e.g., fluoxetine 60mg daily) and help reduce the frequency of binging, irrespective of the presence of depression (Zhu &Walsh, 2002).

Hospitalization is rarely required, but may be indicated when psychotherapy and antidepressants fail to help.

BINGE-EATING

Individuals tend to be middle ages, rather than adolescents or young adults. The genders are more evenly represented. Commonly co-occurs with obesity, and tends to be intermittent (Fairburn et al, 2011). The prevalence is 2% for males and 3.5% for females.

References

American Dietetic Association. Position of the American Dietetic Association: Journal Am Dietetic Association 2011; 111: 1236-1241.
Bell R. Holy Anorexia. Chicago: University of Chicago Press. 1985.
Bulik C. Exploring the gene-environment nexus in eating disorders. Journal of Psychiatry and Neuroscience 2005; 30:335-339.
Bulik C, Sullivan P, Wade T, Kendler K. Twin studies of eating disorders: a review. International Journal of Eating Disorders 2000; 27:1-20.
Bulik C, Tozzi F. Genetics in eating disorders: state of the science. CNS Spectrums 2004; 9:511-515.
Bulik C, Sullivan P, Tozzi F, Furberg H, Lichtenstein P, Pedersen N. Prevalence, heritability, and prospective risk factors for anorexia nervosa. Archives of General Psychiatry 2006; 63:305-312.
Chui H, Christensen B, Zipursky R, et a. Cognitive function and brain structure in females with a history of adolescent-onset anorexia nervosa. Pediatrics 2008; 122:e426-e427.
Fairburn C, Cooper Z. Eating disorders, DSM-5 and clinical reality. British Journal of Psychiatry 2011; 198:8-10.
Frank G, Bailer U, Henry S, Wagner A, Kaye W. Neuroimaging studies in eating disorders. CNS Spectrums 2004; 9:539-548.
Goethals I, Vervaet M, Audenaert K, Jacobs F, Ham H, Van Heeringen C. Does regional brain perfusion correlate with eating disorder symptoms in anorexia and bulimia nervosa patients? Journal of Psychiatric Research 2006; October 17 (Epub ahead of print).
Gowers S, Clark A, Roberts C, Griffiths A, et al. Clinical effectiveness of treatment for anorexia in adolescents. British Journal of Psychiatry 2007; 191:427-435.
Guillaume S, Jaussent I, Olie E, et al. Characteristics of suicide attempts in anorexia and bulimia nervosa: a case-control study. PLoS ONE 2011; 6(8): e23578. doi: 10.1371/journal.pone.0023578
Gull W, Anorexia nervosa (apepsia hysterica, anorexia hysterica). Transactions of the Clinical Society of London 1874; 7:22-28.
Halmi K, Falk J, Schwartz E. Binge eating and vomiting: a survey of a college population. Psychological Medicine 1981; 11:697-705.
Hartman A, Zeeck A, Barrett M. Interpersonal problems in eating disorders. International Journal of Eating Disorders 2009. DOI: 10.1002/eat.20747.
Hay P, Bacaltchuk J, Claudine A Ben Tovim D, Yong P. Individual psychotherapy in the outpatient treatment of adults with anorexia nervosa. Cochrane Database Syst Rev 2003; 4:CD003909
Hoek, H. Incidence, prevalence and mortality of anorexia nervosas and other eating disorders. Current Opinion in Psychiatry 2006; 19:3879-394.
Jennings P, Forbes D, McDermott B, Hulse G, Juniper S. Eating disorder attitudes and psychopathology in Caucasian Australian, Asian Australian and Thai university students. Australian and New Zealand Journal of Psychiatry 2006; 40:143-149.
Joos A, Saum B, Zeeck A et al. Frotocingular dysfunction in bulimia nervosa when confronted with disease-specific stimuli. European Eating Disorders Rev. 2011. Aug 2 [Epub ahead of print].
Kazlouski D, Rollin M, Tregellas J et al. Altered fimbria-fornix white matter integrity in anorexia nervosa predicts harm avoidance. Psychiatry Research 2011; 192:109-116.

Kaye W, Fudge J, Paulus M. New insights into symptoms and neurocircuit function of anorexia nervosa. Nature Reviews Neuroscience 2009; 10:573-584.
Keel P, Mitchell J. Outcome in bulimia nervosa. American Journal of Psychiatry 1997; 154:313-321.
Kendler K, McLean C, Neale M, Kessler R, Heath A, Eaves L. The genetics epidemiology of bulimia nervosa. American Journal of Psychiatry 1991; 148:1627-1637.
Keys a, Brozek J, Henschel A, et al. The Biology of Human Starvation. Minneapolis: University of Minneapolis Press, 1950.
Klein D, Walsh B. Eating disorders. International Review of Psychiatry 2003; 15:205-216.
Krassas G. Endocrine abnormalities in anorexia nervosa. Pediatric Endocrinology Review 2003; 1:46-54.
Lacy J. Anorexia nervosa and a bearded female saint. British Medical Journal 1982: 285:1816-7.
McIntosh V, Jordan J, Carter F, et al. Three psychotherapies for anorexia nervosa: a randomized controlled trial. American Journal of Psychiatry 2005: 162:741-747.
Paul T, Schroeter K, Dahme B, Nutzinger D. Self-injurious behaviour in women with eating disorders. American Journal of Psychiatry 2002; 159:408-411.
Papadopoulos F, Ekbom A, Brandt L, Ekselius L. Excess mortality, causes of death and prognostic factors in anorexia nervosa. British Journal of Psychiatry 2009; 194:10-17.
Pringle A, Ashworth F, Hamer C, Norbury R, Cooper M. Neural correlates of the processing of self-referent emotional information in bulimia nervosa. Neuropsychologia 2011; Aug 5. [Epub ahead of print].
Reda M, Sacco G. Anorexia and the holiness of Saint Catherine of Siena. Journal of Criminal Justice and Popular Culture 2001; 8:37-47.
Russell G. Bulimia nervosa: an ominous variant of anorexia nervosa. Psychological Medicine 1979; 9:429-448.
Sheu C, Tsai J, Chong I, Hwang J. Nutritional emphysema. Internal Medicine Journal 2007; 37:578-9.
Staiger P, Dawe S, McCarthy R. Responsivity to food cues in bulimic women and controls. Appetite 2000; 35:27-33.
Steinglass J, Eisen J, Attia E, Mayer L, Walsh B. Is anorexia nervosa a delusional disorder? An assessment of eating beliefs in anorexia nervosa. Journal of Psychiatric Practice 2007; 13:65-71.
Theander S. Outcome and prognosis in anorexia nervosa and bulimia: some results of previous investigations, compared with a Swedish long-term study. Journal of Psychosomatic Research 1985; 19:493-508.
Treasure J, Russell G. The case for early intervention in anorexia nervosa: theoretical exploration of maintaining factors. British Journal of Psychiatry 2011; 199:5-7.
Wade T, Bergin J, Tiggemann M, Bulik C, Fairburn C. Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. Australian and New Zealand Journal of Psychiatry 2006; 40:121-128.
Wonderlich S, Brewerton T, Jocic S, Dansky B, Abbott D. Relationship of childhood sexual abuse and eating disorders. Journal of the American Academy of Child and Adolescent Psychiatry 1997; 36:1107-1115.
Zakzanis K, Campbell Z, Polsinelli A. Quantitative evidence for distinct cognitive impairment in anorexia nervosa and bulimia nervosa. Journal of Neuropsychology 2009; July 16 [Epub ahead of print]

Zastrow A, Kaiser S, Stipppich C, et al. Neural correlates of impaired cognitive-behavioral flexability in anorexia nervosa. American Journal of Psychiatry 2009; 166:608-616.
Zhu A, Walsh B. Pharmacologic treatment of eating disorders. Canadian Journal of Psychiatry 2002; 47:227-234.