Chapter 6
Eating and Feeding Disorders

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Introduction

Given the salient role that feeding (and eating) play in health and well-being, family functioning and relationship development, emotional regulation and the relatively frequent presentation of eating and feeding disorders, it is understandable that these difficulties are high priority in Pediatric Consultation-Liaison Psychiatry. Using case examples, this chapter reviews the clinical presentation of feeding and eating disorders throughout development, using DSM 5 diagnostic criteria, and suggests a potentially helpful assessment framework and a typical treatment plan.

Changes in the current DSM V diagnostic classification have (2014) included the addition of feeding disorders (FD), based on recognition that many individuals with eating disorders (ED) often had a previous history of childhood feeding difficulties (Uher and Rutter 2012) and that many mothers with either current or past ED have difficulty in feeding their infants (Agras et al. 1999), and the addition of binge eating disorder.

Feeding Disorders

DSM 5 lists three feeding disorders that, although typically presenting in childhood, can also present in adolescence or adulthood: (1) avoidant/restrictive food intake disorder, replacing DSM IV 'feeding disorder of infancy or early childhood', (2)

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Table 6.1 Classification of feeding disorders in infants and toddlers

Specific type	Typical age onset/ developmental phase	Main features
Feeding disorder of state regulation	Newborn period	Infant has difficulty regulating their arousal level to allow for effective feeding
Feeding disorder of reciprocity	2–6 months of age	Significant difficulties in social reciprocity and responsivity
Infantile anorexia	Often at transition to self-feeding	Lack of hunger or interest in food leading to inadequate food intake often accompanied by difficulties in infant-caregiver interactions
Sensory food aversions	Often at introduction of solid feeding	Avoidance often limited to specific food types, based on tastes, textures, appearance or smells. Normal ingestion of other foods
Feeding disorder associated with medical conditions	Can occur at any age	Concurrent medical condition often interfering with eating (cardiac) or leading to distress or pain (reflux)
Posttraumatic feeding disorder	Can occur at any age	Food refusal following traumatic medical (nasogastric [NG] feeding) or physiological (vomiting) events, leading to fear and avoidance

Chatoor (2002)

pica and (3) rumination disorder. This new classification shares many of the salient features present in the DSM IV criteria and also includes conditions proposed to occur at any age. Chatoor proposed several discrete syndromes, most occurring during infancy and childhood and associated with inadequate nutrition and growth failure (Chatoor 2002). Although these syndromes have not been universally accepted (Uher and Rutter 2012), elements are present in the current DSM V categories and highlight the interplay between infant and carer (Table 6.1). The salient role that feeding (and eating) plays in the development of interpersonal relationships and emotional regulation has been central to many of the proposed classifications, leading some researchers to conceptualise them fundamentally as a relational disorder and best termed 'Feeding Disorder Between Parent and Child' (Davies et al. 2006).

Case 6.1: Thomas

A 9-year-old boy, Thomas, was transferred from a local general hospital to a large paediatric hospital because of severe and rapid weight and an almost complete failure/refusal to eat. He was previously managed by his family doctor following a 1-week history of gastroenteritis with projectile vomiting and diarrhoea. He was slow to return to normal eating post recovery and remained fatigued and reluctant to attend school. After a 1-month period of failed treatment by his community primary care physician, he was admitted to his local hospital with continuing weight loss, increasing oppositionality around eating and increasing family discord. He accepted intravenous fluids but steadfastly refused to take any oral nutrition. After 2 weeks with further and significant weight loss, he was transferred to a paediatric hospital for the management of what was termed 'possible anorexia nervosa'.

You ponder how would you go about assessing this child?

'If-Me' Model of Assessment

For feeding disorders, it may be helpful to specifically assess (according to the If-Me or 'If it were Me going to a psychiatrist, what would I want in an assessment' mnemonic) individual, family, medical and environmental factors that contribute to the presenting complaint.

Assessment of Individual Symptoms The clinician needs to establish the child's food-related symptoms, their meaning, duration, development, associated impairment and other symptoms, including depression, anxiety or delusions.

Case 6.2
Thomas' food refusal was driven by an intense fear of vomiting rather than by fear of fatness, as would be typical in anorexia nervosa. Thomas described, because of the aversive experience of projectile vomiting while sick, an intense fear of vomiting that led to phobic avoidance of food, initially high-fat foods (which had been associated with most marked indigestion) but subsequently all foods other than clear fluids. His anxiety levels were pervasively high, and he dreaded the next attempt by family or medical staff to provide nutrition. This dread led to increasing bouts of oppositional behaviour, directed most prominently at his mother.

Assessing Family Factors and Parenting It is important to assess – general and especially more recent parenting styles – how parents have tried to intervene, how they generally relate to this child and their other children, membership of the family unit, family functioning and presence of any family psychopathology that might perpetuate the difficulty or interfere with treatment.

Case 6.3
In Thomas' case, a discussion about the family led the clinician to understand that the mother recently lost her father to pancreatic cancer 3 months prior to Thomas' gastroenteritis. Her father had many gastrointestinal symptoms, including vomiting, and she was aware of the salience of this symptom for Thomas. Mother was very close to her father and was bereft after his death. On reflection, she believes that Thomas's vomiting traumatised her by bringing flashbacks of her father's last weeks, and she opted out of her normal parenting role. Her husband, a successful but busy full-time farmer, was typically not involved in the homemaking or parenting and was unable to compensate for her withdrawal. Thomas' ongoing difficulty around eating, his reluctance to attend school, his demandingness on her time and the different parental perspectives on how best to manage these challenges led to significant inter-parental and family discord, which was uncharacteristic of the family functioning. Thomas' 15-year-old twin siblings, per mother's opinion, seemed relatively unaffected by the situation and had no history of mental health difficulties.

Studies of children with other feeding disorders have found certain caregiver risk characteristics, including tendencies of the mother to be more controlling and

coercive in the feeding situation, to use physical punishment or force feeding and to be more angry and hostile in child interactions (Chatoor et al. 1998, Stice et al. 1999, Allen et al. 2014). In nonfeeding situations parents have also been observed to be less flexible, less affectionate and less attuned to emotional signals from the child, in turn more likely to appear withdrawn (Feldman et al. 2004). Parents with their own eating difficulties were found, in comparison to controls, to display more negativity and conflict during meal times and to have more negative infants (Stein et al. 1994). This interplay between parent and child has led some researchers to propose that feeding disorders should be conceptualised as 'relational disorders' (Davies et al. 2006). This interplay highlights the importance of conducting a good assessment of parenting, examining the relationship between parent and child and enquiring about the caregiver's mental health along with their relationship with food.

Assessing Medical Domain In Thomas' case, assessment of the medical domain includes assessment of immediate medical risk, along with medical developmental history. In general, a detailed dietary history must be collected, recording the exact amount and type of food being eaten, calculating the total daily calorie intake and estimating any deficits and duration of these deficits. Documenting a weight history by establishing the premorbid weight, recording actual weight loss and calculating the deviation from a mean body weight give an indication of the duration and severity of the nutritional deficit. Laboratory investigations will further refine the assessment of medical risk and will point to various interventions.

Case 6.4

Thomas presented after a 2-week period on predominantly IV dextrose, with fewer than 500 calories per day, placing him at a very high risk of refeeding. When Thomas was admitted, he weighed 55lbs (9th percentile), and his height was estimated at 4 feet 5 inches (37th percentile), giving him a current BMI (wt/ht2) of 13.8 (< 3rd percentile). A previous weight, 4 months prior to his illness, was 65lbs, (BMI 16.3, 50th percentile) representing a 15% drop in weight. Given that the mean male BMI at age 9 is 16.8, Thomas % mean BMW (also referred to as ideal body weight or IBW) was 82%, significantly lower. He appeared lethargic, had muscle wasting and hypotonia and had poor cardiac reserve with bradycardia (40 bpm), electrolyte disturbances (raised urea, low potassium and calcium) and hypothermia. Mother reported that Thomas met all his developmental milestones at the appropriate age and was medically well. He was perceived to be somewhat anxious, and 'measured', preferring familiarity to novelty and being slow to trying new foods and new activities. These traits had been accommodated by his family, and despite a somewhat selective diet and cautious personality, they were not viewed as impairing.

A clinical history is often enriched by observations of a feeding interaction, which might uncover developmental and medical factors and highlight any discordant relationships and coercive experiences. Clinical examination will assess general development, postural control, swallowing and sensory integration, which are all potential contributions to feeding disorders. The clinician may also wish to use a

caregiver questionnaire to supplement clinical history and observations and to offer a more holistic and multi-rater perspective. The psychometric properties and clinical utility of several feeding questionnaires were reviewed by Sanchez et al. (2015), who recommended the Behavioural Paediatric Feeding Assessment Scale.

Assessing Environmental Domain This domain will typically encompass the young child's academic and behavioural progress at school along with their peer relationships. Additionally, it includes any psychosocial adversities in the home and neighbourhood. Even in affluent nations, poverty and lack of food continue to be a significant factor in feeding problems (Casey et al. 2001).

Case 6.5

An assessment of Thomas' environment suggested minimal stressors linked with academic or peer activities. He was perceived as an able and willing student, albeit somewhat perfectionistic and high achieving. He was likeable and had a small number of close friends. His only post-school activities were weekly attendance at chess club and scouts.

A diagnosis of avoidant/restrictive food intake disorder was made.

Avoidant/Restrictive Food Intake Disorder (ARFID)

The salient feature in this diagnostic category is difficulty or severe disinterest in eating (or, in an infant, being fed) leading to inadequate nutritional intake and weight loss (or, in growing children, failure to maintain weight trajectory). The reasons leading to such difficulties are many but do 'not' involve a prominent fear of weight gain or body image distortion, as would be typical in anorexia nervosa (Bryant Waugh 2013). Additionally, these difficulties may present at any age across the lifespan, not just during infancy or childhood. Although specific aetiologies are not assumed or required, examples of putative aetiological causes include, as in the case of Thomas, a reaction to a previous aversive experience, where choking or severe vomiting induces anticipatory anxiety upon eating, which is subsequently avoided. Other aetiologies include idiosyncratic preferences for various food textures, appearances, smells and tastes, often linked with other sensitivities. In ARFID, the eating disturbance must not be because of a concurrent medical or psychiatric condition, such as depression leading to a loss of appetite.

Prevalence of Feeding Problems Transient, non-impairing and self-limiting food restriction is common and occurs in up to 25% of children (Jacobi et al. 2008). If general feeding problems are considered, the prevalence in typically developing children increases to almost 1 in 2, linked with temporary weight or relationship difficulties that fall short of a more formal 'feeding disorder' (Linscheid et al. 2003). General feeding problems account for the commonest reason for primary caregiver consultations in the preschool age (Arvedson 2008). It is only when the difficulty persists, linked with weight percentile trajectory deviations, nutritional deficiency

or significant interpersonal relationships, that it is considered pathological, with the child requiring additional high-calorie enteral nutritional supplements.

The prevalence of feeding problems is higher in children with developmental delay or attending specialists' clinics such as gastroenterology, speech and language or genetics clinics, where 8% met formal diagnostic criteria (Williams et al. 2009). When broader diagnostic criteria are applied, such as those suggested by Chatoor, up to 80% of children with developmental difficulties have feeding problems (1994). These may arise from structural or functional abnormalities, including oropharyngeal skills deficits, cleft lip or palate as may occur in 22q11 deletion syndrome or gastrointestinal reflux linking feeding and swallowing with pain and generating an aversive experience. Neurological issues such as cerebral palsy, leading to low muscle tone and dysphagia, may also be contributory and evident upon observation. Children with feeding problems also often have subclinical symptoms of obsessive-compulsive disorder, anxiety or autism (Schreck et al. 2004). Although some of these features were present in the case presented, as with many other cases, Thomas did not meet full diagnostic criteria for either autism or anxiety.

Additionally, aspects of the mother (carer)-child relationship may contribute, with suboptimal feeding experiences being linked with, for example, a mother being unaware of developmental stages or nutritional needs, unaware of child's inability to sit upright and self-feed or fearful of the infant choking, leading to delayed or shortened solid feeding and a transfer of anxiety to the infant. A mother with her own eating and body image-related concerns may control both food types and amounts offered to her child, along with meal time in general. Environmental factors, especially social adversity, poverty and lack of opportunity, may also play a part. The assessment of these difficulties requires a biopsychosocial approach, with an awareness of the often-combined contribution from several sources (Davies et al. 2006).

Treatment of Feeding Disorders

To date there are no evidenced-based guidelines for the treatment of ARFID, or indeed other feeding disorders, due to their recent conceptualisation. Given the differences in aetiological associations, it is likely that the treatment of feeding disorders, especially ARFID, will include cognitive, behavioural and psychological interventions and at times expert input from a multidisciplinary team, including mental health clinicians, paediatricians, dieticians and occupational and speech and language therapists. The body of evidence supporting the effectiveness of certain interventions appears to be growing, as indicated by publications in many varied journals (Strandjord et al. 2015).

The goal of treatment, based on a collaborative approach involving the young person, their family and the treating team, is to have the young person eating normally and adequately again. At a basic level, the focus is to have the child eat and subsequently to address other domains that may mediate or moderate their feeding

problems. Once the medical risk has been stabilised, treatment typically commences by establishing the young person's motivation to engage and establish their position in the stages of change model (Prochaska et al. 2008). Clinicians need to enlist the support of their parent to a degree based on the child's maturity and ability to engage yet giving the responsibility for the feeding and treatment plan to parents. Information about what constitutes a healthy diet, the food pyramid and energy requirements and changes linked with puberty are all possible motivators in an otherwise disinterested or scared child.

A cBT (cognitive behavioural therapy) approach is used, with the small 'c' representing the degree of cognitive work that is commensurate with a child's developmental age and capacity to use metacognition, i.e. thinking about thinking. The plan includes behavioural experiments where new foods are added based on a hierarchy, beginning with foods deemed easiest or 'most wanted', i.e. foods that allow social reintegration, such as popcorn and pizza. The management of food-related anxiety is important, as oftentimes the food restriction is in response to some aversive experience, and sometimes even the thought or presentation of food is enough to retraumatise the child. Anxiety symptoms may also be comorbid. Mindfulness allows for a more focused awareness of bodily sensations, including anxiety and autonomic instability with the approach of food or meal times. Practising relaxation, guided imagery and biofeedback may all be helpful. Family support is essential, as family may well have struggled heroically for long periods, particularly with AFRID, prior to seeking treatment (Forman et al. 2014). Parents may need help in reestablishing healthy meal time approaches and interactions with their child. They may need to examine their own prejudices regarding forbidden and unhealthy foods, and they may need to be reminded to separate their child from their illness through the process of externalisation (White and Epston 1990).

What factors do you think are relevant to target in Thomas' case?

Treatment of AFRID

Case 6.6

*Thomas was initially fed nasogastrically with small amounts of calories that were increased to his typical daily caloric intake by day 7. He was encouraged to take oral fluids in monitored amounts. The team used the 'IFME' framework. From individual assessment, there was no evidence that Thomas had any desire to be thin, there was no body image distortion and/or unwillingness to consider an appropriate range of calorie-dense foods. His current food preferences were not based on any sensory issues outside of the potential for a food item to get 'stuck in his throat' and to cause gagging and vomiting, his ultimate fear. He identified an acceptable range of 'less chokey foods'. The dietician introduced food items into his meal plan, starting with very small portions (*e.g. *one spoon of plain yoghurt, two spoons custard, one softened Weetabix) that were increased to more typical amounts with a corresponding reduction in the NG amounts. Thomas was coached in progressive muscle*

relaxation, deliberately starting with a focus on his feet, as far away from his mouth as possible. Guided mourning assisted the mother to overcome her grief and to be more available to the family unit. Family discussions led to a hierarchical shift, in which both parents were repositioned firmly at the top and in which siblings formed a supportive sibship group for Thomas. Attendance at hospital school allowed Thomas to experience social and academic engagement, which improved his mood. Consistent weight gain, normalisation of physical and laboratory tests and increasing food repertoire led Thomas to be more confident about trying increasing amounts of 'chokey' foods, and about trying these off the ward, starting with the hospital coffee shop and subsequently sites outside of the hospital. Following successful overnight leaves, Thomas was discharged from the hospital 4 weeks later with some food supplements. Thomas was followed up as an outpatient for ongoing review and growth monitoring, family-focussed work and individually focused cBT. His final review 18 months post-admission showed him to have regained his weight trajectory, with minimal residual anxiety symptoms and no food-related worries. His mother, who returned to work, reported more parenting competence and described a relaxed, happy household.

Picky Eating

Picky eating refers to a child's tendency to eat a preferred but restrictive diet, to be avoidant of trying new foods (food neophobia), to exhibit strong likes and dislikes and to insist of food being prepared or served in certain ways, in the context of normal weight. Adherence to this repertoire may severely restrict nutritional intake (Carruth et al. 2004), may cause growth retardation (Dovey et al. 2008) and may contribute to family conflict (Mascola et al. 2010). A child's refusal to eat certain foods, or to insist that parents prepare separate meals, has been linked with other behavioural problems and may represent an effort of the child to exert their autonomy. Other behavioural problems, such as temper tantrums, sleeping problems or oppositional-defiant behaviour, may coexist and may need to be the focus of treatment, with the management of picky eating being secondary (Jacobi et al. 2008; Mascola et al. 2010; Rydell et al. 1995). Successful treatment of clinically significant picky eating involves careful assessment of not only the food consumed but also the feeding and general relationship of the child and caregiver. An integrated treatment approach is often required.

Previously and occasionally still referred to as selective eating, fussy or faddy eating, or food avoidance, picky eating is common (Cardona Cano et al. 2015; Timimi et al. 1997), with prevalence ranging between 14% and 50% in early childhood (Dubois et al. 2007; Carruth et al. 2004) and decreasing with age (Mascola et al. 2010). Usually short lived and resolving by middle childhood, many prospective longitudinal studies have shown that a significant minority persist into adulthood (Jacobi et al. 2008). Some cases have their onset in adolescence (Van Tine et al. 2017). Concerns about the pathologising of picky eating as a psychiatric disorder (Kirkey 2012) have been raised and in part reflect the lack of standardised

definition and conflicting descriptions from studies that recruit different populations and that have heterogeneous samples (Norris et al. 2016). The inclusion of picky eating under the ED section might be understandable given the increased awareness, from several longitudinal studies, of the risk for subsequent disordered eating (Kotler et al. 2001, Van Tine et al. 2017, Mascola et al. 2010; Rydell et al. 1995). In the case presented above, although Thomas had many features of picky eating, i.e. preferred foods, his premorbid overall food intake was less restrictive, with severe selectivity being clearly linked with fear of choking or vomiting and resolving successfully following treatment.

Pica and Rumination Disorder

'Pica' refers to a 1 month or longer period of nonnutritive or non-food substance ingestion that is neither culturally sanctioned nor developmentally appropriate. This behaviour is also not part of another mental health disorder or, if so, is sufficiently severe to merit attention. Medical complications, such as bowel obstruction or infections, are more likely to uncover this behaviour than any nutrient or vitamin deficiency, as often there is adequate nutritional intake. Although there are no community studies on the prevalence, it is more common in children and individuals with autism or intellectual disability and can occur temporarily in pregnancy. Treatment, although not well studied, typically involves behavioural interventions and environmental manipulations.

Pica is distinguished from eating as part of culturally sanctioned or socially normative practices, i.e. ingestion of clay, mud or lime in India may be viewed to be spiritually desirable. As with most FD, the onset is usually but not exclusively in childhood. Pica may occur in pregnancy or as part of autism spectrum disorder, but for a diagnosis to be given, it should be severe enough to warrant independent clinical attention.

'Rumination disorder' refers to the regurgitation of previous chewing or swallowed food, in an effortless manner and without disgust or distress. Regurgitated food may be subsequently swallowed or spat out. As with pica, a minimum duration of 1 month is required, with behaviour occurring most often daily and not part of an eating or other mental health disorder. Unlike pica, rumination disorder is more often associated with failure to thrive, failure to gain weight or hunger states, especially in younger children if food is not re-swallowed. In older individuals, social etiquette may also lead to an avoidance of social eating resulting in weight loss. As with pica, it is more common in individuals with autism or intellectual disability, but exact prevalence rates are not available. Although generally assumed to be more common in early childhood, some studies have suggested a mean age of onset in young adolescence (Chial et al. 2003). Treatment is usually behavioural, but further study is necessary.

Table 6.2 DSM V criteria for eating disorders

	Anorexia nervosa (AN)	Bulimia nervosa (BN)	Binge eating disorder (BED)
Weight/BMI	Significantly underweight		
Fear of wt gain/ fatness	Intense		
Body image/shape disturbance	Significant		
Recognition of seriousness of weight loss	Absent		
Emotions			Disgust, depression, guilt and marked distress
Feeling of control		Lack of control	Lack of control
Restricted energy intake	Persistent		
Self-evaluation		Linked with shape/weight	Linked with shape/weight
Binging	Less likely	Significant amount 1/ week × 3/12 months	Significant amount 1/ week × 3/12 months
Purging (vomiting, laxatives)	Only if binge-purge subtype	1/week × 3/12 months	
Excessive exercising	Can occur	Can occur	
Subtypes	Restricting		
	Binge purging		
Notes		Not exclusively during AN	Not exclusively in AN/BN

Eating Disorders in Children and Adolescents

Anorexia Nervosa

Originally described in 1873 by Sir William Gull, anorexia nervosa (AN) is the most commonly presenting eating disorder in the acute hospital setting. DSM V made two key changes that enhance its applicability to the child and adolescent age group. Firstly, it removed the criteria of amenorrhoea, so it can now fully apply to prepubertal girls, girls with primary amenorrhoea and boys. Secondly, it did away with a strict weight guideline of less than 85% and specifically includes the concept of developmental trajectory. The diagnosis of an 'atypical anorexia' is considered when not all of the AN criteria are met (see Table 6.2), e.g. a child's weight is within the normal range, but their weight loss is very significant as the child was in the obese range to begin with. Atypical anorexia nervosa is classed as an 'other specified feeding or eating disorder (OSFED)' within DSM V.

Epidemiology

The lifetime community prevalence of anorexia nervosa in adolescents has been estimated at 0.3% (Swanson et al. 2011) and as high as 1% in other studies. An even higher proportion of adolescents report subthreshold symptoms of anorexia nervosa (McNicholas et al. 2010), while within the younger paediatric population group (8–15 years), eating disorders and anorexia are less common (0.1%). However, anorexia nervosa is the most common eating disorder diagnosed in the younger paediatric population, and its median age of onset in the paediatric population is 12.3 years (Swanson et al. 2011). The female/male gender ratio in childhood is much less pronounced at 2:1 (CDC 2013 accessed 2016), with female preponderance then developing during adolescence. A comprehensive study of primary care databases in the UK reported incidence rates of 13.1 per 100,000 in the 10–14-year-old group and 26.7 per 100,000 in the 15–19-year-old group (Micali et al. 2013). Incidence appears to be rising.

Aetiology

Anorexia nervosa has complex biopsychosocial origins that are not yet fully understood. Several twin, adoption and family studies indicate that genetic factors play a key role in determining vulnerability to developing anorexia, with heritability estimates of 50–60% most commonly reported (Trace et al. 2013). Heritability is likely to involve several additive genetic factors, including phenotypic variations of noradrenergic, serotonergic and dopaminergic systems; endocrine, leptin and appetite centres; and obesity receptors. Epigenetic factors that may then influence phenotypic expression of anorexia nervosa may include the phenotypic effects of feeding difficulties, starvation and puberty (Yilmaz et al. 2015, Zerwas and Bulik 2011).

Psychological Factors

Certain premorbid child and adolescent temperamental and personality traits have also been associated with a higher risk of developing anorexia nervosa and include perfectionism, obsessive-compulsive traits, anxious temperament, low self-esteem and harm avoidance (Anderluh et al. 2003; Jacobs et al. 2009; Grylli et al. 2005; Fan et al. 2010). Premorbid body dissatisfaction has been noted in children as young as 8 years (Micali et al. 2015), who subsequently are more likely to overvalue their identity around appearance and control of food intake. Medical conditions such as diabetes mellitus and coeliac disease, where children have learned to count or restrict calories and avoid carbohydrates and other food types, and a high focus on athletic and gymnastic success also predispose genetically vulnerable children to developing an eating disorder, to manipulating their eating to cause weight change or to increase athletic performance (Bachle et al. 2016).

Social Influences

While social factors alone do not cause anorexia nervosa, they no doubt are associated with development and maintenance of AN particularly in adolescents. The socio-cultural pursuit of thinness, idealised by magazines, media and social media, has significantly contributed to body dissatisfaction in all ages, especially during the critical developmental stage of identity formation and socio-emotional growth (Harrison 2000, Goncalves et al. 2012). In a time of unparalleled access to social media in younger adolescents, this is important as both a risk and maintaining factor. Such influence may be mediated though the family's idealisation of thinness and attitudes towards eating, social interests and peer competition (Ferguson et al. 2012). Children and adolescents with anorexia nervosa often identify a precipitating critical life event, often bullying, peer conflict, family stressor, childhood trauma and abuse or negative change event. Such social factors may trigger a cycle of eating disorder behaviour and psychopathology that then becomes embedded. While much of the literature on anorexia nervosa is based in westernised cultures, this is an eating disorder that occurs globally, across culture, ethnicity and geographical boundaries.

Case 6.7: Amy

Amy is a 15-year-old teenager who sees her family doctor, Dr. Johnson, for a history, as reported by the mother, of noticeable weight loss (about 20 pounds) over the last 6 months. Previously of average weight and with good health, Amy has dropped three clothes sizes and has become irritable and tired. She has also withdrawn from family and friends. Amy announced 4 months ago that she had become vegan, and she is 'obsessed' with healthy eating magazines and Instagram pictures of models. She is a keen gymnast, but her mother is very concerned that her physical activity has become extreme. On two occasions recently, she became faint at school and was sent home, and her performance at gym has suffered. Amy is a bright student and has always pushed herself to do well, so this setback upset her greatly. She thinks she might need a multivitamin to help her concentrate and have more energy at school. Her mother thinks she may have an eating disorder: a few friends have had eating issues, as did an aunt.

How would you screen and assess Amy using the 'If-Me' framework, as described above?

Screening for Eating Disorders

Children and adolescents with eating disorders usually present initially to their family doctor, and so screening of Amy begins here with careful history taking and physical examination. The SCOFF screening tool (Morgan et al. 2000) is a useful framework for Dr. Johnson. He will also need to inquire about Amy's eating patterns, weight loss trajectory, exercise, physical symptoms and pubertal, growth and menstrual status. Amy will also require a physical examination with attention to

height; weight; BMI and BMI percentile; sitting, lying, and standing pulse and blood pressure; temperature; any signs of starvation or medical instability; and an ECG

Case 6.8
Dr. Johnson concludes that Amy may have an eating disorder with significant risk: her heart rate is 50, she is losing 0.5 pounds per week, and she looks malnourished. He therefore refers her to the paediatric hospital and psychiatry team.

Assessment of Eating Disorders

When a patient presents to an eating disorder or child liaison team, she will meet with a child psychiatrist/paediatrician and therapist and will receive a full history, with a focus on thoughts and feelings about food, eating, weight and shape and a psychosocial history, mental status and physical examination and risk review.

Assessment of Individual Symptoms

Case 6.9
As well as school gymnastics practice, Amy disclosed that she participates in school gymnastics practice, goes to the gym three times per week for up to 2 hours at a time and runs each day to improve her performance and to be lean and 'healthy'. She checks her weight daily and skips breakfast and lunch on most days. While she plans to eat lunch at school, she tends to join friends in their decision to skip lunch. She admits to avoiding 'carbs'. She is upset that she tires easily and is moody. Amy is now very anxious and worried about missing school, and her doctor notes that she is irritated with her mother for making her go through an appointment. The doctors see that Amy's self-esteem is very low. She feels that the only thing that is going well for her is her eating plan, and she is worried that if she loses control of the situation, she will not reach her target of 100 pounds and will not make the gymnastics team. As Amy's insight seems to be poor, her doctor obtains collateral history from the parents and asks about symptoms of depression, suicidality, anxiety, obsessive-compulsive disorder, psychosis, autism as well as other medical disorders which might present similarly or complicate her treatment, e.g. *coeliac disease, diabetes, hyperthyroidism, other endocrine disorders,* etc. *The doctor also obtains a detailed medical and developmental history, including feeding history.*

Use of Supplementary Questionnaires

Especially when patients have difficulty discussing feelings about shape and weight, standardised questionnaires and instruments can supplement the clinical interview (Hay et al. 2014; Lock et al. 2015). For a 15-year-old patient like Amy, the

children's eating attitudes questionnaire (ChEAT, Maloney et al. 1988) or the eating disorder examination-questionnaire (EDE-Q, Fairburn and Beglin 1994) are good options to explore thoughts, feelings and behaviours and to track progress during treatment.

Case 6.10
Amy finds the process of completing a questionnaire useful in helping her to realise how out of balance her life has become. She is now feeling scared and overwhelmed and recognises that she may need some help. Amy also completes a quality of life measure (the Clinical Impairment Questionnaire, Bohn et al. 2008) and realises how her problem has taken over her friendships, family and activities. The doctor is hopeful that these insights might help with motivation and identification of recovery and treatment goals.

Assessment of Family Factors

This step includes assessment of family composition, communication, relationships, major events, eating patterns, history of eating disorders or other mental health concerns and strengths and resilience factors. It also includes assessment of impact of eating disorder symptoms on the family.

Case 6.11
Amy lives with her mother; her father, who works long hours; and her sister, who is 11 years old and who has diabetes. Amy's close maternal aunt had an eating disorder when she was 19 years old. She is now recovered but, as a PE teacher, focuses significantly on fitness and shape. She spends a lot of time with Amy while the father is away at work. Amy's younger sister, Susan, spent two long periods in the hospital in the last 3 years. Susan is on insulin, monitors her blood sugars and follows a careful diet, which all the family are aware of. She and Amy are quite competitive. Amy has been frustrated at times when her sister has needed high levels of support and feels terribly guilty about being frustrated. The Parent Versus *Anorexia Scale (Rhodes et al. 2005) helps the team to recognise how un-empowered her parents feel they are in terms of helping Amy with her eating disorder on top of all the other stressors they face as a family.*

Assessment of Medical Factors

Case 6.12
The team reviews a 4-day eating diary and notes that there are long gaps of 6 hours between meals, and when Amy does eat, it is a mainly plant-based diet with little protein and no fat or complex carbohydrates. They estimate that she is eating 800 calories per day and using up 400 alone though exercise. They are relieved that there is no history of binging, purging or use of laxatives. They review the primary care physician's growth and weight charts and note that these were normal until

2 years ago. Amy met all her developmental milestones early and has no other significant medical history. Her history of fainting, occasional palpitations, skin deterioration and tiredness concern them.

Assessment of Environmental Factors

Case 6.13
Amy has been finding it hard to balance her school work with getting on the gym team, and this pressure has been building up now that exam time is approaching. There has also been a falling out among her group of friends in the last year, and Amy feels caught in the middle. She admits being an avid follower of two bloggers who have eating disorders. Academically, Amy has always achieved high grades and continues to do so with additional effort due to her fatigue.

At this point, construct a biopsychosocial formulation for Amy and include predisposing, precipitating, perpetuating and protective factors.

Physical Examination

The child psychiatrist and family doctor/paediatrician must collaboratively establish level of immediate physical risk. Unlike in adults, the healthy BMI range for children and adolescents varies over childhood, so it is essential that BMI is plotted onto an appropriate BMI percentile chart and that % BMI be calculated (RCPsych 2012). One study found that one-third of children under 13 years with early-onset eating disorders were medically unstable at presentation despite not being significantly underweight, and half of them required hospital admission (Hudson and Court 2012). Therefore, BMI % alone is inadequate in assessing risk, and a broad and systematised approach to physical risk assessment is required.

Case 6.14
Amy's doctors noted her cachectic appearance, lanugo (fine) hair on her face, dry skin, acrocyanosis in her fingers, brittle hair and cold intolerance. They reviewed the results of Dr. Johnson's investigations, with special attention to vital signs. Her height is 165 cm and her weight is 46 kg, giving her a BMI of 17 and a BMI % of 82.7. She is slightly dehydrated, but her heart rate is 55 with normal ECG, and there is no significant drop in postural systolic or diastolic blood pressure. She has proximal leg muscle wasting and scores a 2 on the sit up and squat test (SUSS) (RCPsych 2012). In line with the history, there are no signs of self-induced vomiting: no enlarged parotid glands, Russell's sign (knuckle calluses) or eroded or loose dentition.

Physical Investigation

Laboratory studies should screen for any nutritional deficiencies and the physiological impact of the eating disorder (Table 6.3). These will need to be repeated at regular intervals as part of regular risk monitoring.

Table 6.3 Physical investigations

Blood tests: Urea and electrolytes, calcium, liver function tests, coagulation, glucose, full blood count, white cell count differential, vitamin B-12, folate, vitamin D	Electrocardiogram
Endocrine thyroid function tests, follicle-stimulating hormone/luteinizing hormone/testosterone	Abdominal ultrasound (if no menstruation for >6/12 months)
Coeliac screen as history indicates	Dexa scan (if no menstruation for >6/12 months)

Case 6.15

The team concludes that Amy has anorexia nervosa, restricting subtype, and discusses the most appropriate treatment setting.

In which setting would you treat Amy?

What do you think that the key components of her treatment plan be?

Three principles to consider in the paediatric age group are that (1) early intervention is most effective (2). Psychosocial treatment involving families is most effective (3). Treatment begins with patient safety, and in AN refeeding is the most important first treatment.

Medical Admission

If physical risk assessment indicates physical instability or food intake less than 400 kcal per day, a paediatric inpatient medical admission is warranted to enable safe supervised refeeding and medical stabilisation. Two key frameworks that enable clinicians to take a systematic approach to decision-making are the Junior MARSIPAN guidelines, MARSIPAN checklist (RCPsych 2012, 2015) and Society of Adolescent Health and Medicine guidelines (2015). The goal is to minimise the risk of refeeding syndrome, a rare but potentially fatal consequence of the body's sudden shift from catabolic to anabolic state, which can result in electrolyte imbalance, multi-organ failure, seizures, coma and death (Mehanna 2008). Communication around eating disorders can be complex, as anorexia nervosa crosses the medical and mental health interface, and multiple clinicians and teams are involved. Suggestions to enhance communication, safety and care are included in Table 6.4. Patients may feel very anxious and angry at the idea of weight gain, which will need to be explained and enforced firmly, consistently and collaboratively by clinicians and parents, so that refeeding is effective and safe.

Outpatient Treatment

As soon as the patient is deemed medically stable, with improvement of the physical parameters that led to her hospitalisation (e.g. MARSIPAN checklist, RCPsych 2016 and a pattern of some restoration of her oral intake), she should be treated in an outpatient setting so that her family can become empowered quickly to manage

Table 6.4 Inpatient admission guidelines

Initiation of the refeeding plan immediately; food is the most important treatment
Formal weekly meeting of paediatrics, liaison psychiatry, nursing and dietary team
Collaborative written treatment plan by this multidisciplinary team
Clear communication, by a nominated person, of the plan, once formulated, with Amy and her family
Twice weekly weights (first thing in the morning while wearing hospital garments)
Daily physical and review of blood K, P, mg and white cell count for 5–7 days (highest refeeding risk)
ECG monitoring
Supervision of meals and use of toilets
Involvement of parents where possible early on to empower them (e.g. during snack time)
Possible supplementation with thiamine and phosphate
Activities and activity level determined by psychiatry team consultation and advice
Goal of 0.5–1 kg weight gain per week in hospital
(Very rarely) bolus NG feeding may be needed as a short-term intervention
Admission of ED patients to the same ward to allow staff to build experience

the eating disorder (Lock 2013). In this age group, specialist outpatient eating disorder treatment is as effective as, and less costly than, specialist inpatient care (Gowers et al. 2010). For a small number of patients for whom outpatient care has failed or for whom parents are unable, a more structured programme of refeeding and psychological treatment may be needed, and an inpatient or day patient eating disorder programmes may be appropriate. A recent randomised open study comparing inpatient and day patient treatment in Germany for adolescents with moderate/severe anorexia following brief psychiatric admission found that day programmes were as effective as long-term inpatient admission for weight restoration, weight maintenance and safety and that they were less costly (Herpertz-Dahlmann et al. 2014). Adolescent day programmes in the UK such as multifamily therapy for anorexia nervosa are also options (Eisler et al. 2016).

Evidence-Based Psychosocial Treatment of Anorexia Nervosa

Families do not cause adolescent anorexia nervosa, but it is overwhelmingly clear from the literature that they are key to recovery and that systemic family therapy treatments are superior to individual therapy (Robin et al. 1994, Lock et al. 2010). Family-based treatment for anorexia nervosa (FBT) is the most systematically researched treatment to date and is regarded as the first-line treatment for adolescents with anorexia nervosa (Hay et al. 2014; Lock et al. 2015; Herpertz-Dahlman 2016).

Case 6.16

Amy's father will adjust his work to attend the family-based treatment sessions. The psychiatrist explains that this is a manualised treatment that lasts up 20 weeks and that will empower Amy's parents to take charge of refeeding, with weekly specialist

support and monitoring of weight and physical parameters. They are told that this treatment is associated with BMI recovery rates of 40–50%, with a further 30% of adolescents having a partial recovery (Lock and Le Grange 2012). They are encouraged to approach her illness as they would a serious, life-threatening medical condition requiring a programme of treatment. They are all clear that a goal of 0.2–0.5 kg weight gain per week is expected with outpatient treatment.

If the patient were too obsessional, or if FBT were not possible for family reasons or otherwise ineffective, a broader systemic family therapy approach would be recommended (Lock et al. 2015). Second-line interventions include multifamily therapy for anorexia (Eisler et al. 2016), enhanced cognitive behavioural therapy for eating disorders (CBT-E) for older adolescents (Fairburn 2008) and day/inpatient programmes. To date, there is no evidence to suggest that psychopharmacological treatments are effective in treating Amy's anorexia nervosa, and so except for nutritional supplementation, medication would be used only for comorbidity (e.g. anxiety or depression) (Herpertz-Dahlman et al. 2015). Throughout her psychosocial treatment, Amy's psychiatrist/paediatrician will continue to monitor her physical parameters to ensure that her medical risk is monitored.

Prognosis of Paediatric Anorexia Nervosa

A recent meta-analysis of 35 studies found, for AN in the 15–19 age group, a 10% mortality rate, which is the highest for the mental disorders. Prompt engagement in treatment is therefore essential. For a significant subgroup, AN will become a chronic or life-limiting illness (Arcelus et al. 2011). On a positive note, adolescents appear to have a better prognosis than adults with anorexia nervosa, with one study reporting, for severe anorexia nervosa, a 75% full recovery rate at 10–15 years (Strober et al. 1997). Key to good outcomes is access to early specialist intervention and a focus on weight and behavioural outcomes.

Bulimia Nervosa

Epidemiology Bulimia nervosa (BN) is more common in childhood than anorexia nervosa, with a lifetime prevalence of 1–1.5% in paediatric community samples. Like with AN, a higher proportion of the paediatric population has subthreshold symptoms, but, in comparison to AN, BN presents less frequently to liaison services. There is a significant age effect, with an incidence of 2.9/100,000 in the 10–14-year-old group and 25.9/100,000 in the 15–19-year-old group. At 2–3:1, the female to male ratio for BN is lower than that for AN (Micali et al. 2013, Swanson et al. 2011).

Diagnostic Criteria

The main change in the DSM V for BN is that binging and purging cycles need only occur once weekly rather than twice as was the case in DSM IV-TR (Table 6.2).

Aetiology

The different eating disorders overlap substantially, and over time they can develop from one into the other. Indeed, a transdiagnostic approach to understanding and treating all eating disorders has been developed (Fairburn 2008).

Biological Factors Most twin and family studies estimate BN's heritability to be between 0.55 and 0.62 (Trace et al. 2013). Multiple candidate genes include those related to gender, obesity, weight and appetite and neurotransmitters such as serotonin and dopamine. Studies also indicate that early pubertal timing is a biological risk factor for the development of BN in girls (Klump 2013).

Psychological Adult studies associate BN with certain personality traits. A child or adolescent, who has difficulty in regulating their emotions, who is impulsive and who engages in risk behaviours such as substance misuse and self-harm, is more likely to develop BN (Fischer 2007; Pearson et al. 2015; Myers et al. 2006). The CBT-E formulation of BN identifies how low self-esteem leads to an overevaluation of shape and weight and subsequent dieting and compensatory behaviour. In a vulnerable adolescent, this process develops into binge behaviour and a cycle of escalation (Murphy et al. 2010).

Sociocultural Factors Adolescent social pressure around thinness and body dissatisfaction is associated with BN. Adolescents who want to become athletes, especially those interested in aesthetic sports (gymnastics, aerobics, dancing), modelling and bodybuilding (especially for males), are all at increased risk. With a 5–16% prevalence in such subgroups, BN is the most common eating disorder in athletes (Currie 2010; Joy et al. 2016). Other social risk factors for BN include a history of childhood trauma, abuse, poly trauma (Utzinger et al. 2016) and criticism.

Screening and Assessment of Bulimia Nervosa

Screening and assessment by the family doctor and specialist liaison or eating disorder team is identical to that described for Amy above. The EDE-Q can be particularly helpful for uncovering hidden bulimia-type behaviours such as laxative abuse and self-induced vomiting, which are often under-reported or not known to parents. Such diagnostic accuracy is particularly critical to decision-making around treatment.

Specifically, for BM, it is important to explore:

- Individual factors: comorbidity of depression, substance use, deliberate self-harm and suicidality.
- Family factors: adverse situations that may be a consequence to parental attempts to manage the ED and that may perpetuate the ED. Parental attitude to food, body shape and exercise are also important perpetuating factors that will need to be carefully reviewed.
- Medical factors: frequency of purging, which may lead to cardiac abnormalities, potassium depletion, metabolic alkalosis or acidosis, palpitations, fainting, breathlessness or haematemesis
- Environmental factors: such as reckless social behaviour and relationships, to ensure that safety is maintained.

Physical Examination and Investigation

In addition to following the same format as outlined for AN (Table 6.2), the examining doctor will look for signs of dehydration and purging (enlarged parotids, loose teeth), check urea and electrolytes and assess for hypokalaemia-induced cardiac changes on ECG patients with BN who are also at high risk of oesophageal tears or rupture. Dental referral may be required to identify and treat dental erosion and loose teeth. Even adolescents with bulimia who are not anaemic or underweight may be malnourished due to purging, and nutritional screening tests will also be needed.

Case 6.17

Michelle is a 15-year-old female with a % BMI of 105% who is referred by her family doctor for eating disorder assessment. In the last year, she describes having lost control of her eating, and she feels frustrated and hopeless. She makes herself sick 2 times per day on most days, particularly in the evenings after dinner when she feels at her lowest. She often skips breakfast and lunch, but then later in the day is overwhelmed by hunger and will eat anything she can find. Last night she had up to a loaf of bread, chips, a tub of ice cream as well as dinner and cookies. She finds it hard to remember what she eats or feels at the time. Afterwards however, she feels awful and usually makes herself sick. She has also cut her arms and thighs on several occasions. Michelle is frustrated as her weight is 68 kg, and she knows she is overweight, but she can't lose weight despite the purging and fasting. She goes to the gym in bouts of 2–3 h when she exhausts herself. She is then starving, irritable, 'all over the place' and angry. Her mother thinks that she is 'big' like her family and that Michelle is influenced by magazines and friends not realistic about how she can look.

What is your diagnosis and why?

Construct a formulation of Michelle.

Outline your physical and risk assessment of Michelle.

What investigations will you order?

Treatment of Bulimia Nervosa in Adolescents

Adolescents with BN rarely require medical admission unless they have hypokalaemia or ECG changes, in which case telemetry may be required, and so they are less likely to present to a paediatric liaison service. Once cardiac and gastrooesophageal issues are resolved, treatment is generally on an outpatient basis. Hospital management principles are like those recommended for AN (Table 6.4), with individualised determination of weight goals and vitamin supplementation.

There is a very limited evidence base around the psychological treatment of adolescent BN. CBT-E has been recommended because of its evidence base in adults (Murphy et al. 2010; Lock et al. 2015). Like FBT, CBT-E is also a manualised treatment that is delivered by a skilled therapist and that involves a 20- or 40-session programme focused on normal eating pattern restoration through real-time recording, regular meal structure, weekly weights, psycho-education and formulation and eventually discussions of body shape evaluation, body checking and management of emotional triggers. It is adapted for adolescents and emphasises parents' important role in the treatment process. More recently, three randomised studies of older adolescents suggest that an adapted form of family-based therapy (FBT-BN) may be superior to individual therapy for BN (Le Grange et al. 2015). Guided self-help may also be considered for older adolescents. There is no evidence base around BN treatment in adolescent males. Outpatient psychosocial treatment is always complemented by regular meal planning, nutritional supplementation and physical monitoring.

As with adolescent AN, psychopharmacological treatment is reserved for comorbidity, and despite evidence of effectiveness for high doses of fluoxetine (60 mg) in adults with BN, there have been no robust trials in adolescents (Goldstein et al. 1995).

Prognosis Bulimia nervosa has a standardised mortality rate (SMR) of 2.22 based on 12 studies (Arcelus et al. 2011). Risk factors associated with poorer outcome in adolescents include family history of obesity, longer duration and poor early response to treatment. There is also a risk of developing AN.

Binge Eating Disorder

Binge eating disorder (BED) was previously classified as an atypical disorder under 'Eating Disorder Not Otherwise Specified' (ED-NOS) in DSM 4-TR but now has full eating disorder status. As with BN, the only change is a decrease in the threshold of binge-purge frequency from twice to once per week (Table 6.2). Due to its relatively recent inclusion as a full disorder (it remains under 'other' disorders in ICD 10), BED is relatively less studied, particularly in the paediatric population.

Epidemiology Previous inclusion under ED-NOS enables us to estimate that the lifetime prevalence of BED in community-based samples of 13–18-year-olds is 1.6%, making it the most common of the adolescent-eating disorders. It is also more

common in females. The onset of BED is in later adolescence or early adulthood, with an average age of onset in the early to mid-twenties (Kessler et al. 2013).

Aetiology In the absence of studies of BED in adolescents, research in adults indicates that this disorder has a complex aetiology similar to the other eating disorders, where a combination of genetic factors, dieting, body dissatisfaction, low self-esteem, emotional eating and social factors all play a role. A key cognitive process involves binging leading to feelings of disgust, depression and shame, which further reinforces the adolescent's low self-esteem and cyclical pattern of binging.

Screening and Assessment

Screening and assessment for BED follows a similar framework to AN. Comorbid depression is common. Particularly in the case of binge eating disorder, adolescents may be significantly overweight or obese, and the medical history and examination may need a paediatric obesity-focused workup to include screening for: (1) developmental syndromes (e.g. Prader-Willi) with karyotyping if necessary; (2) metabolic syndrome and diabetes (lipids, blood glucose); (3) endocrine problems involving thyroid, growth or reproductive hormones; (4) gastrointestinal diseases; and (5) other anatomical abnormalities visible on imaging studies (e.g. polycystic ovaries, brain lesions or other pathology).

Treatment

Patients with BED are less likely to be physically compromised and so generally present to primary care services. Paediatric admission is rare. To date, there have been no RCTs that specifically address the treatment of BED in the adolescent population, and it is recommended that treatment planning is extrapolated from what works for an adult population, or for the adolescent-eating disorder, it most closely resembles. CBT-E or guided self-help may be useful approaches (Hay et al. 2014). However, given adolescents' developmental stage and relative vulnerability compared with adults, an adapted CBT-E approach with parent support is the most scaffolded and safest approach for the treatment of BED in adolescents. Group formats also hold promise, particularly for older adolescents, but this modality requires further research in this age group.

Conclusion

Feeding and eating difficulties frequently present in childhood and adolescence and need prompt identification and expert management to preserve physical and psychological development and avoid life-threatening situations.

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