The Overweight Child

DEFINITION

Obesity implies the presence of excess body fat, and any increase in adipose mass is the result of increased storage of triglycerides. Thus, ideally, the diagnosis of obesity should be based on a direct demonstration of an increased amount of body fat. This is possible directly, for example with imaging techniques – computed tomography (CT), magnetic resonance imaging (MRI) and dual-energy X-ray absortptiometry (DEXA) scanning – and underwater weighing, and indirectly with ultrasonography, bioelectrical impedance analysis or skinfold thickness measurements. However, these technical measurements of body composition are not suitable for routine practice, and are limited to research protocols.

The use of skinfold thickness requires an experienced technician who knows how to use skinfold callipers appropriately (see **Figs 1.10–1.13**). Equations have been published to estimate fat mass from skinfold measurements, but their accuracy is limited, especially in individuals with very small or very large amounts of body fat.

Therefore, for clinical purposes, body weight is the usual proxy parameter for defining a child being thin (underweight), normal or fat (overweight). Overweight can also, by analogy to short or tall stature, be defined as a weight above the normal range, but in this case the situation is somewhat more complex. However, the cut-off limits for overweight and more extreme overweight (which is then labeled 'obesity') should be determined along with the reference data used to define the chosen cut-off limits for all age groups. It should be remembered that overweight can be caused not only by excess fat, but also by increased fat-free mass (e.g. muscle in well-trained athletes), and skinfold thickness measurements can help to differentiate in these cases.

There is a general consensus that body mass index (BMI, or Quetelet's index), calculated as weight (kg) divided by height (m) squared (weight/height^2), is the most suitable parameter to define overweight. It has a high correlation with fatness, is largely independent of height, and is the standard measure used in adulthood. Age- and-sex-related references are available from many countries. In several of these references the strongly skewed distribution of BMI for age has been transformed into a gaussian distribution, so that an individual BMI can be expressed as a standard deviation score (SDS) for age. Age reference values for BMI from the 1997 nationwide growth study in the Netherlands are shown in **Figs 5.1 & 5.2**.

Alternatively, weight can be plotted (on a logarithmic scale) versus height (on a linear scale) and compared with references for weight-for-height. An individual weight can then be expressed as a percentage of median weight for height. As the 90th centile is close to 110% of median weight-for-height, this percentage has also been used as cut-off for overweight. A comparison of weight-for-age percentile position with height-for-age percentile position has also been used in various countries, but this procedure systematically overestimates the target weight of adolescents.

With respect to the cut-off limits used to define underweight, overweight and obesity, an international task force of the World Health Organization and the European Childhood Obesity Group suggested pediatric centiles derived from the extrapolation of a BMI of 20, 25 and 30 kg/m^2 in young adults as the respective cut-off values. To establish the centiles, an analysis of six large population studies (from Brazil, the UK, Hong Kong, Netherlands, Singapore and the USA) resulted in two curves for each sex corresponding to 25 and 30 kg/m^2 in young adults (see **Figs 5.1 & 5.2**). These can now be used as the standard definitions for child overweight and obesity.

Although BMI SDS is the most practical index of overweight, it is still not a very accurate parameter of obesity for any given individual. In the absence of increased fat mass, body weight and hence BMI can be high due to a relatively high fat-free mass. On the other hand, excess fat mass will not be detected by increased BMI in cases of severe muscle wasting and osteoporosis.

BMI also gives no information about the distribution of body fat. In adults the regional distribution of body fat appears to be an important factor in determining
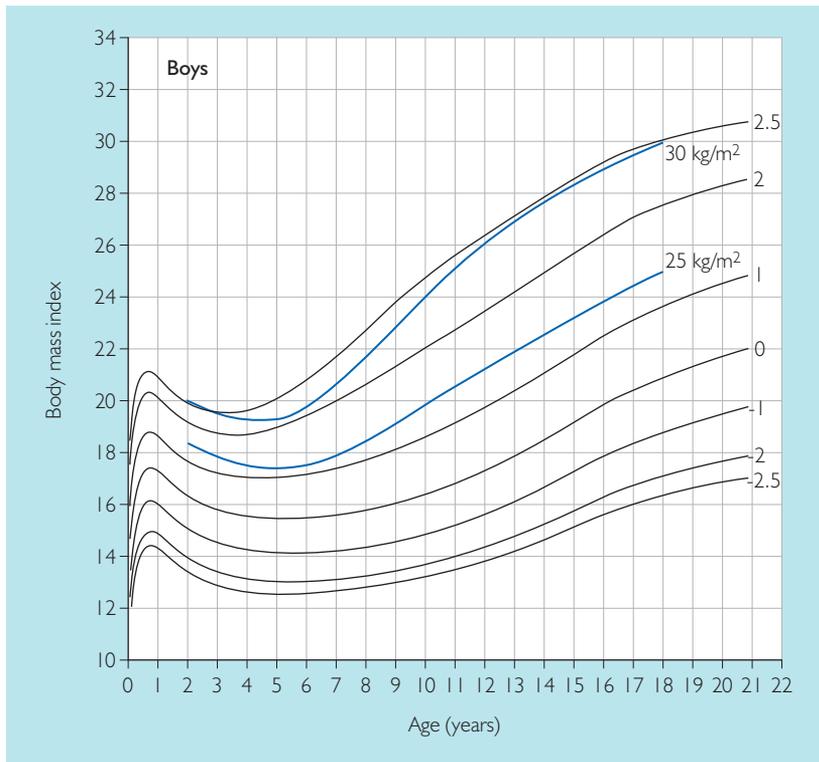
Fig. 5.1 Body mass index for age in Dutch boys compared with cut-off lines for overweight (extrapolated from 25 kg/m^2 at 18 years) and obesity (extrapolated from 30 kg/m^2) according to international growth references (Cole et al. *BMJ* 2000; 320: 1240–3). Lines indicate 0, ±1, ±2 and ±2.5 SDS. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.
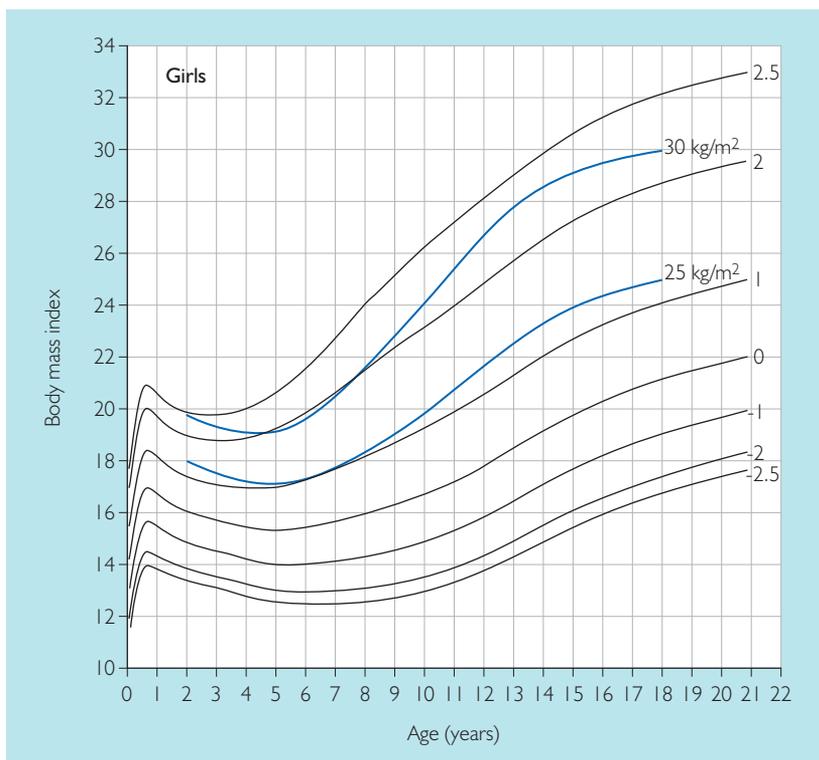
Fig. 5.2 Body mass index for age in Dutch girls, as in Fig. 5.1. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.

the risk profile for various diseases (i.e. the components of the metabolic syndrome or syndrome X). Abdominal obesity, which generates an 'apple' form of the body habitus (android obesity), is associated with insulin resistance, cardiovascular disease, non-insulin-dependent diabetes mellitus, stroke and hyperlipidemia. In the case of a body habitus with a peripheral fat distribution in the femoral area, called the 'pear' shape (gynoid obesity), these risks are much lower. Body fat distribution can be assessed by measuring the waist and hip circumferences, and calculating the waist : hip ratio. However, while in adults a ratio greater than 0.9 in males and 0.8 in females is associated with increased risk of insulin resistance and associated diseases, such standards have not been established in childhood obesity. Recent Dutch references for waist and hip circumferences, and the waist : hip ratio are presented in **Figs 5.3–5.8**.

EPIDEMIOLOGY

In all industrialized countries the percentage of overweight children has increased spectacularly in the past few decades. Percentages obviously depend on the definition used. Using the international definition described above, in the Netherlands the mean prevalence of overweight increased from 9.4% in 1980 to 13.4% in 1997, and in many other industrialized countries higher percentages have been reported. In children of Moroccan and Turkish origin, the percentages were close to 20%. The main reasons for this increase are probably changes in diet and activity patterns. The amount of fat and other calorie-rich food components in the diet of children has increased tremendously, together with the growing consumption of fast food and sweetened soft drinks. At the same time, children and adolescents nowadays spend much time watching television and playing computer games, so that physical activity has decreased. There is also an interaction effect, as children consume many snacks while watching television, and at the same time they are bombarded by TV commercials advertising fast food. Physical activity is probably also decreased by the fact that many children and youngsters have few opportunities to play outdoors and by fewer facilities in school. It has been shown that the prevalence of overweight and obesity is associated with living in a city and with lower socioeconomic status. The increase in the percentage of overweight children can be considered a prelude to a great increase in the prevalence of obesity in adults, as many obese children will become obese adults. This will have grave consequences for public health, especially cardiovascular health.

As overweight causes earlier puberty, possibly through the influence of leptin on pulsatile gonadotropin secretion, the population trend toward a higher prevalence of overweight causes a shift in the age references for pubertal stages, especially for girls. In obese male adolescents gynecomastia is relatively common, probably due to aromatase activity in adipocytes producing estrone from androstenedione.

PHYSIOLOGY

A simplistic view of the control of body fat is that a control system of appetite and satiety, localized in the hypothalamus, regulates dietary intake, in order to balance energy expenditure. However, the precise regulatory mechanisms have not yet been clarified. Part of the controlling system is a negative feedback loop, in which leptin, produced by adipose tissue, provides a feedback signal to the hypothalamic centers. Serum leptin levels are age dependent and correlated with fat mass. Age references are available, but the clinical value of measurement of serum leptin levels is not yet clear. Leptin circulates bound to a specific carrier protein and is actively transported across the blood–brain barrier to act on specific receptors in the ventromedial nucleus of the hypothalamus, probably along with other signals from glucose and insulin. Subsequent interactions with neuropeptides (e.g. neuropeptide Y) and corticotropin releasing hormone (CRH) and pro-opiomelanocortin (POMC) in the arcuate and dorsomedial nucleus act to control feeding behavior mediated through the paraventricular nucleus. Recently, it has also become clear in animal studies that adipocytes produce a host of other specific proteins, one of which, resistin, may induce insulin resistance, thus linking diabetes to obesity, although there are few data in humans. Ghrelin, secreted by the gastrointestinal tract in response to gastric dilatation, may modulate central growth hormone (GH) release and hence peripheral lipolysis. Some undefined genetic factors may have had survival value when famine was common, but now lead to obesity in people living in affluent conditions.

Obesity causes alterations in endocrine physiology. The strongest effect is seen on the somatotropic axis, and includes low levels of serum GH (also in response to provocation), high levels of GH binding protein (GHBP), normal (or raised) levels of insulin-like growth factor (IGF-1) and IGF binding protein (IGFBP-3), and low levels of IGFBP-1. Serum triiodothyronine (T_3) concentration is slightly increased, probably as a result of increased conversion of thyroxine (T_4) to T_3. The prolactin response to thyrotropin releasing hormone (TRH) may be blunted. Cortisol secretion rate is increased, but
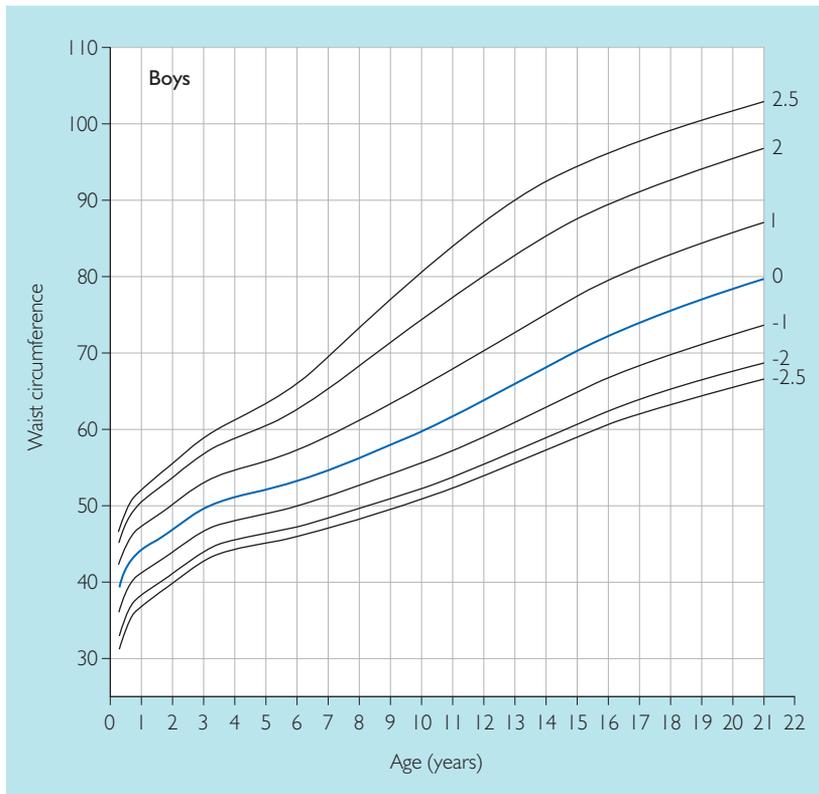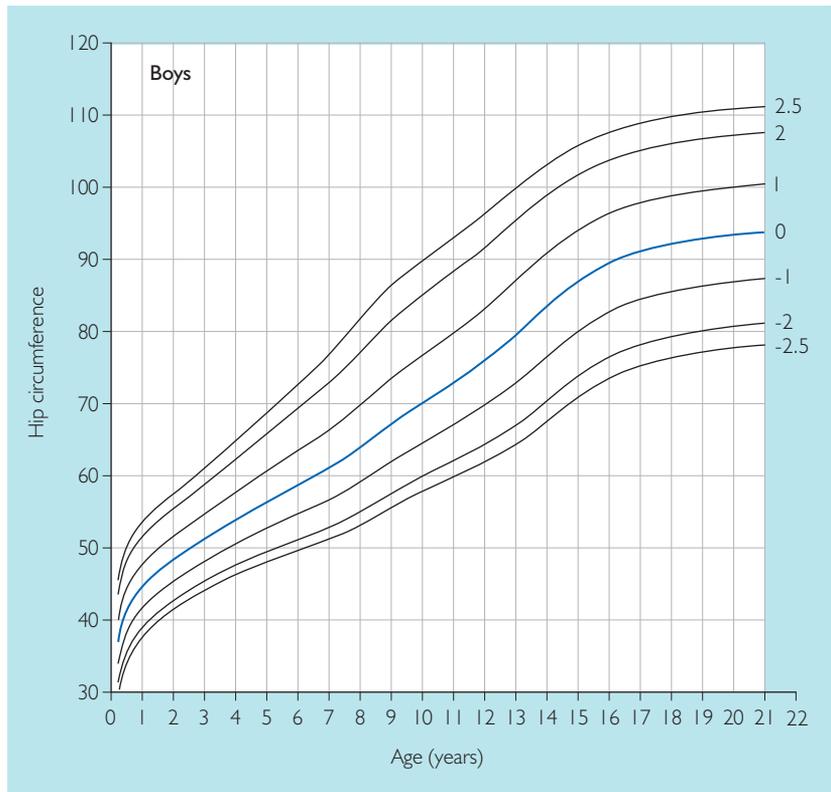
Fig. 5.3 Waist circumference in Dutch boys. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.
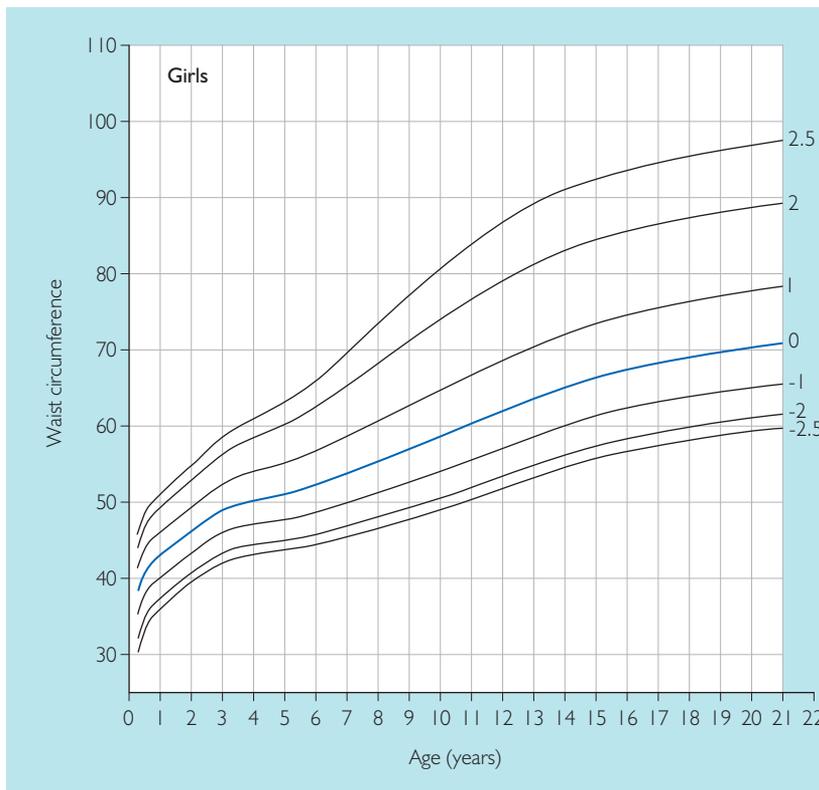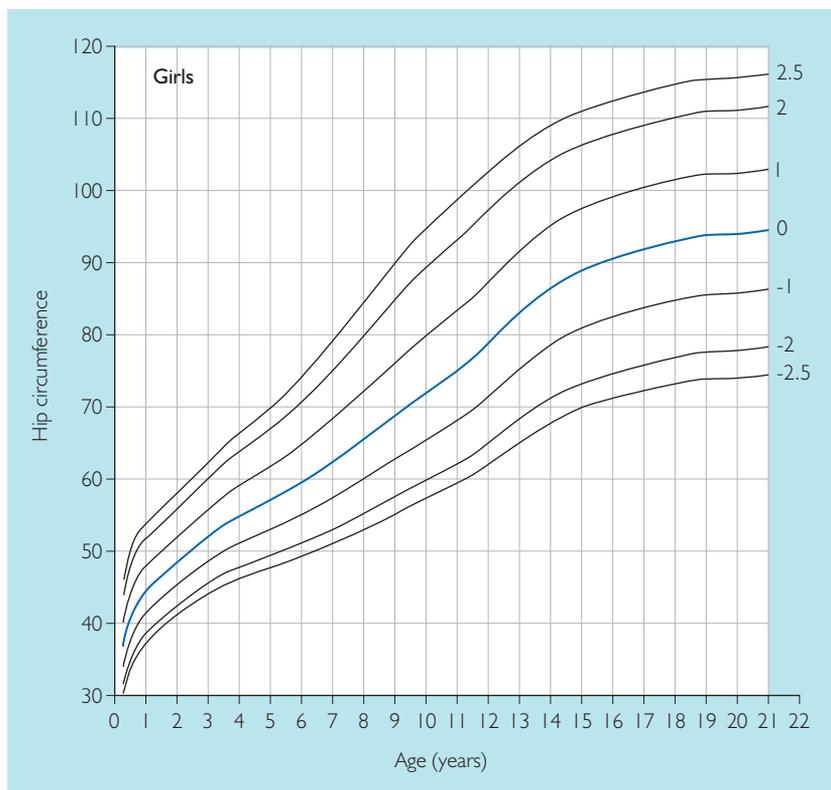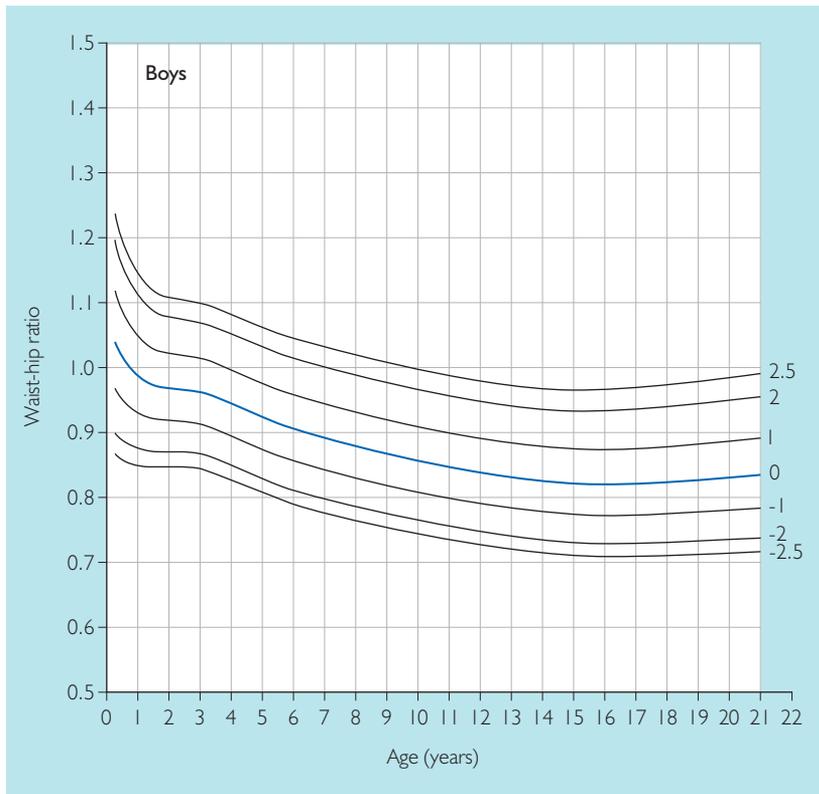

Fig. 5.4 Waist circumference in Dutch girls. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.

Fig. 5.5 Hip circumference in Dutch boys. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.
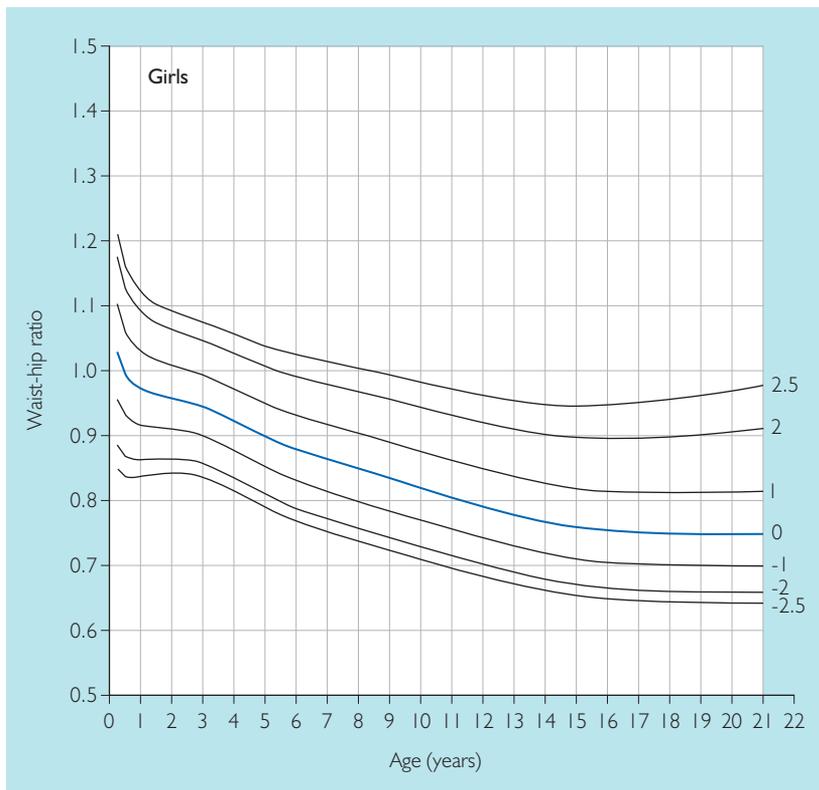
Fig. 5.6 Hip circumference in Dutch girls. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.

Fig. 5.7 Waist : hip ratio in Dutch boys. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.

Fig. 5.8 Waist : hip ratio in Dutch girls. From Fredriks et al. Fourth Dutch growth study. *Arch Dis Child* 2000; 82: 107–12.

serum cortisol levels and urinary free cortisol excretion are normal. In boys serum testosterone and sex hormone binding globulin (SHBG) are decreased and estrogen (especially estrone) concentration is increased. In girls levels of both estrogens and androgens are increased, SHBG concentration is low, and puberty is advanced. Insulin resistance is usually present.

CAUSES

SIMPLE OBESITY

In most cases, obesity in childhood is caused by a combination of genetic and environmental factors. There is frequently a family history of overweight, which can be an expression of the genetic influences as well as the influence of family dietary practices. A dietary history usually indicates excessive calorie intake, although the actual intake is often underreported. This is usually called 'simple obesity', although its pathophysiology, as well as its treatment, is far from simple. Growth velocity is slightly increased, so that the majority of overweight and obese children have a height in the upper half of the population reference, and higher than the target height. Bone age is often slightly advanced, as well as the onset of puberty. Adult height is generally close to target height; however, those with the earliest progression into puberty may be several centimeters below their target height as adults. Psychologic disturbances are often present, and may play a role in the development of obesity. However, obesity also leads to psychologic problems and social stigmatization.

Examination

- The excess subcutaneous fat is often the only abnormality (**Figs 3.29 & 5.9**).
- Some children show high cheek color, mimicking Cushing syndrome (see **Fig. 3.30**). Striae (usually quite narrow) may also be present.
- Hypertension may be present (but a large sphygmomanometer cuff must be used).
- Hirsutism from polycystic ovary changes and the low SHBG levels secondary to insulin resistance (see **Figs 6.33–6.35**).
- Limited rotation of the hip may indicate slipped femoral epiphyses (**Fig. 5.10**).
- Acanthosis nigricans and skin tags may indicate insulin resistance (see **Fig. 1.140**).

Investigation

There may be a number of metabolic changes:

- Hyperinsulinemia and impaired fasting glucose, which can lead to glucose intolerance and type 2


Fig. 5.9 Gross infantile obesity.

Fig. 5.10 Slipped capital femoral epiphysis.

diabetes mellitus. The incidence of this type of diabetes in childhood and adolescence has increased greatly in the past two decades (see Ch. 10).
- Hyperlipidemia is associated with obesity.
- Sleep apnea can occur with right heart failure in extreme cases (**Figs 5.11 & 5.12**).
- Non-alcoholic steatohepatitis (NASH) may result in progressive abnormalities of liver function (**Fig. 5.13**).
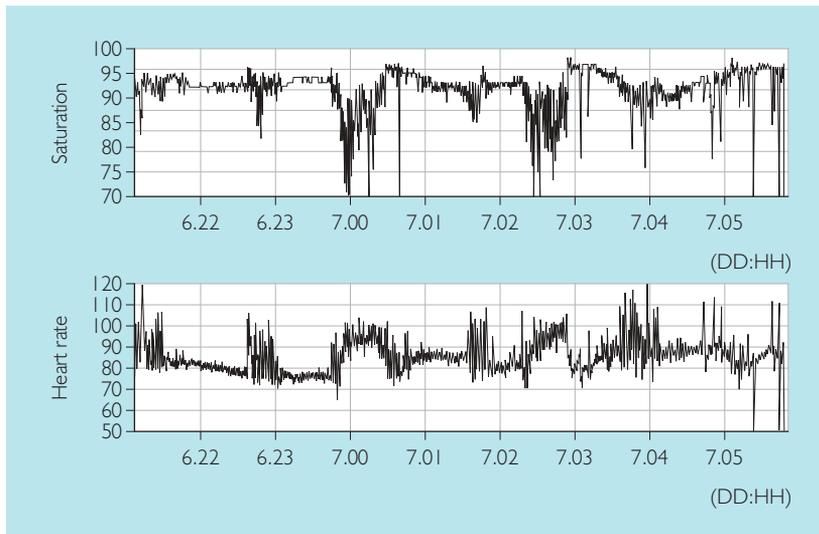
Fig. 5.11 Oximetry trace from the patient in Fig. 5.9 demonstrating sleep apnea with dips in oxygen saturation to less than 80%.

Fig. 5.12 Cor pulmonale with gross obesity (note subcutaneous fat thickness).
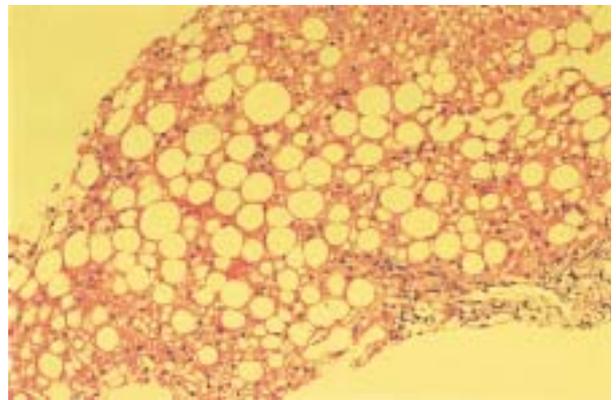
Fig. 5.13 Liver biopsy of non-alcoholic steatohepatitis (NASH) in simple dietary obesity. Note large fat-filled spaces. Liver function deranged with raised transaminases and γ-glycosyltransferase.

■ In adulthood, persistent obesity can lead to the same disorders, as well as coronary heart disease, stroke, gallbladder disease, psychosocial disability and osteoarthritis.

ENDOCRINE CAUSES OF OVERWEIGHT

While simple obesity is associated with *increased* growth velocity, most endocrine causes of obesity are associated with a decreased growth rate. Although an endocrine cause can be found in only a small minority of obese children, it is important to perform additional investigations to diagnose or exclude an endocrine cause. The main clinical reason is for treatment but another, less medical, reason is that many parents know of the association between obesity and 'hormones or glands' and expect endocrine causes to be investigated (see Ch. 2).

Juvenile hypothyroidism

This is characterized by slow growth, delayed bone maturation and (mostly mild) obesity (see Chs 2 & 9) (see **Fig. 9.5**). Substitution therapy with L-thyroxine normalizes the clinical picture completely, although adult height may be less than the target height (see **Fig. 2.91**).

Glucocorticoid excess

Glucocorticoid excess, due either to exogenous administration or to an increase in endogenous production, also causes slow growth and obesity. Even a small excess of steroids above physiologic substitution can cause obesity.

For the clinical characteristics of Cushing syndrome, see Chapter 2. The classic 'buffalo hump' and the centripetal fat distribution are not always seen in children. A 24-h urine free cortisol and a diurnal cortisol rhythm are probably the most sensitive screening tests. If clinical concern persists, an overnight dexamethasone suppression test is often performed followed by low- and high-dose dexamethasone suppression tests and imaging studies as appropriate (see Appendix). If Cushing syndrome or disease is treated successfully, BMI decreases, although often not completely (see **Figs 2.42 & 2.43**).

Growth hormone deficiency

GH deficiency is characterized primarily by slow growth, but in severe cases there may be truncal obesity (see **Fig. 2.9**). The truncal fat may have a dimpled appearance (see **Fig. 1.124**).

The BMI is usually in the normal range, except when a hypothalamic disorder (e.g. craniopharyngioma) is the cause of GH deficiency. GH treatment not only causes catch-up growth, but also normalization of body composition, with disappearance of truncal obesity.

Mauriac syndrome

This condition, which occurs when diabetes mellitus is poorly treated, is now rarely seen in industrialized countries (see **Figs 4.17 & 4.18**). Poor growth is combined with peripheral wasting and abdominal distension, leading to a superficial appearance of overweight.

There are also two endocrine conditions in which obesity is not associated with poor growth:

- Insulinoma, which is characterized primarily by hypoglycemia (see Ch. 11).
- Polycystic ovary syndrome (PCOS), a syndrome of dysregulation of the hypothalamo-pituitary–gonadal axis, of which the pathophysiology is still unclear (see Ch. 6). It is associated with obesity, insulin resistance, hypertension, hirsutism, menstrual irregularity and high serum levels of luteinizing hormone (LH). Its prevalence appears to have risen in adolescents along with the rising prevalence of obesity. A history of intrauterine growth retardation is said to be a risk factor.

CENTRAL NERVOUS SYSTEM CAUSES

Tumors, malformations, infiltration (histiocytosis) and damage by infections (meningitis, encephalitis) or trauma (birth trauma or another causality) in the

Fig. 5.14
Moderate obesity secondary to large arachnoid cyst, shown in Fig. 5.15.
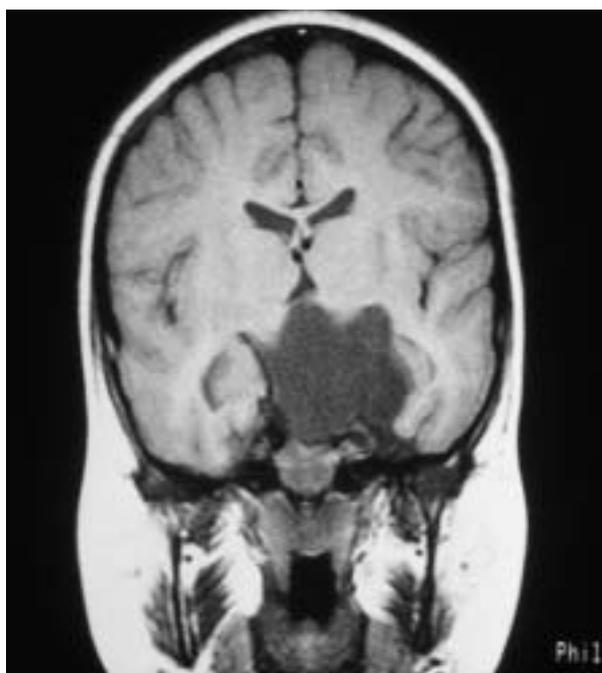
Fig. 5.15 Large arachnoid cyst.

hypothalamus often result in abnormal weight gain. If a hypothalamic lesion is suspected, brain imaging (MRI) is indicated (**Figs 5.14–5.16**). Obesity may be extreme (morbid) in children with hypothalamic tumors, such as craniopharyngioma, particularly after
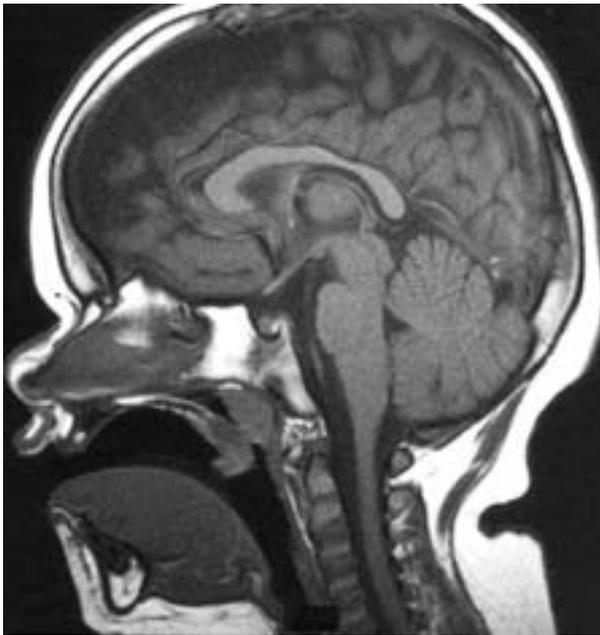
Fig. 5.16 Normal MRI of pituitary and hypothalamus in suspected central disorder of eating behaviour in child with absent thyroid stimulating hormone (TSH) response to thyrotropin releasing hormone (TRH) and central hypothyroidism. Note the massive subcutaneous skinfold seen at the neck.

Fig. 5.17
Prader–Willi syndrome.

surgery. Compulsive eating and lack of satiety may be the most important etiologic factor, but it is likely that changes in energy expenditure are also involved. These forms of obesity are very difficult to treat.

CHROMOSOMAL DEFECTS

Chromosomal disorders with trisomy or deletion and duplication of the X chromosome are associated with mild obesity (such as Turner, Klinefelter and Down syndromes).

OTHER GENETIC DEFECTS

Prader–Willi syndrome

After a period of hypotonia and poor feeding in the first year, obesity typically develops between 1 and 4 years of age. There are a number of dysmorphic features, intellectual impairment, slow growth and hypogonadism (**Fig. 5.17**) (see also Ch. 2 – **Table 2.2, Figs 2.15 & 2.16**). The dietary treatment of obesity is usually very difficult, although with special behavior modification reasonable results can be obtained, particularly if started early. However, prevention of obesity in early childhood is most important.

Other

Other, less well known, syndromes associated with obesity are Beckwith–Wiedemann syndrome (see **Figs. 3.17–3.19**), Laurence–Moon–Biedl syndrome (see **Figs 1.117 & 5.18**), Alstrom syndrome, Cohen syndrome, Carpenter syndrome (see **Fig. 1.31**) and pseudohypoparathyroidism (see **Figs 1.28, 1.29, 1.81, 2.51**).

GENETIC DISORDERS OF CENTRAL APPETITE REGULATION

These abnormalities are rare, but potentially treatable. They offer some insights as to the genesis of obesity in the general population.

A few families with massive obesity due to leptin deficiency or leptin receptor defect have been described. There is often consanguinity and a family history of hypogonadism. Here, treatment with recombinant leptin can produce massive weight loss and restoration of pulsatile gonadotropin secretion (**Fig. 5.19**).

A small number of other rare abnormalities of hypothalamic signals of satiety have been described, including the red-haired human homolog of the Agouti

Fig. 5.18
Laurence–Moon
–Biedl syndrome.

Fig. 5.19 Treatment of congenital leptin deficiency with recombinant leptin. In addition to weight loss there was a reversal of central hypogonadism. From Farooqi S et al. *N Engl J Med* 2000; 341: 879–84.Copyright © 2000 Massachusetts Medical Society. All rights reserved.

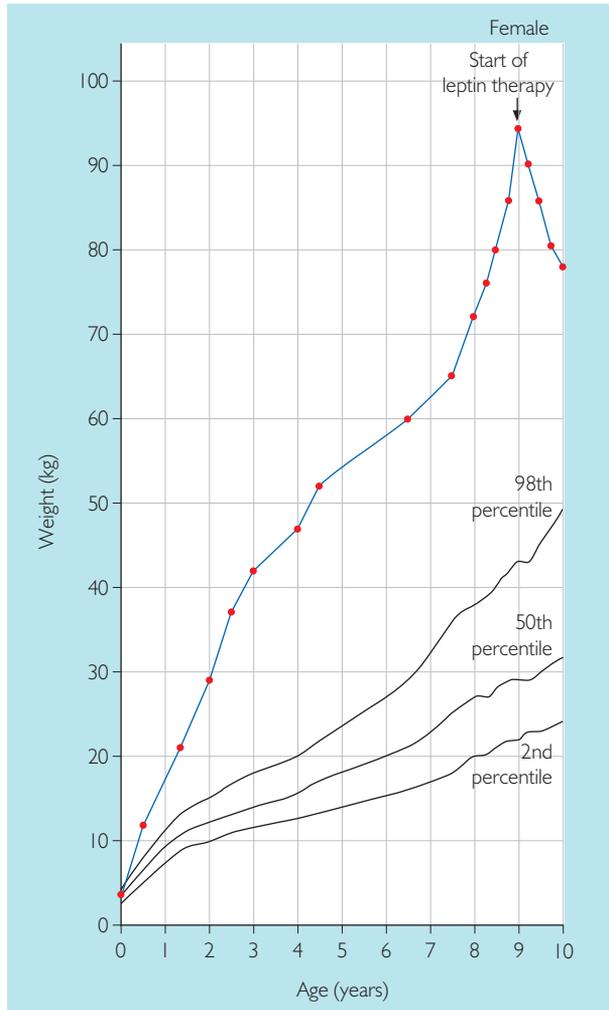mouse due to POMC deficiency. More such rare defects will undoubtedly emerge with further research.

OBESITY ACCOMPANYING IMMOBILITY, MENTAL DISTURBANCE, SOCIAL AND CULTURAL PRESSURE

In children with muscular dystrophy, spina bifida, achondroplasia, immobilization after trauma and/or orthopedic surgery, and intellectual impairment, obesity is secondary to a combination of decreased energy expenses due to immobility, overprotection of parents, and abnormal food intake resulting from mental disturbance or social/cultural pressure. In such cases it may be useful to assess body composition, with auxological or technical means.

IATROGENIC OBESITY

Obesity can be caused by various medications:

- Corticosteroids (see endocrine causes).
- Overmedication with insulin in diabetes, which may lure adolescents into a vicious circle of insulin overdosage–hypoglycemia–overeating–obesity–insulin resistance and back to insulin overdosage.
- Sodium valproate, carbamazepine, phenothiazine, tricyclic antidepressants and cyproheptadine.

EVALUATION OF THE OVERWEIGHT CHILD

HISTORY AND EXAMINATION

Although in most obese children no pathologic cause can be found, to permit the label of 'simple obesity' to be given, a full history and physical examination have to be performed in all cases.

Growth should be plotted along with previous measurements of height and weight.

Data on height and weight of parents and siblings provide insight into possible genetic factors and familial dietary patterns.

A detailed history of diet (not forgetting non-diet soft drink use) and physical activity is essential, although

Fig. 5.20 Severe dietary obesity: weight +7.7 SDS.
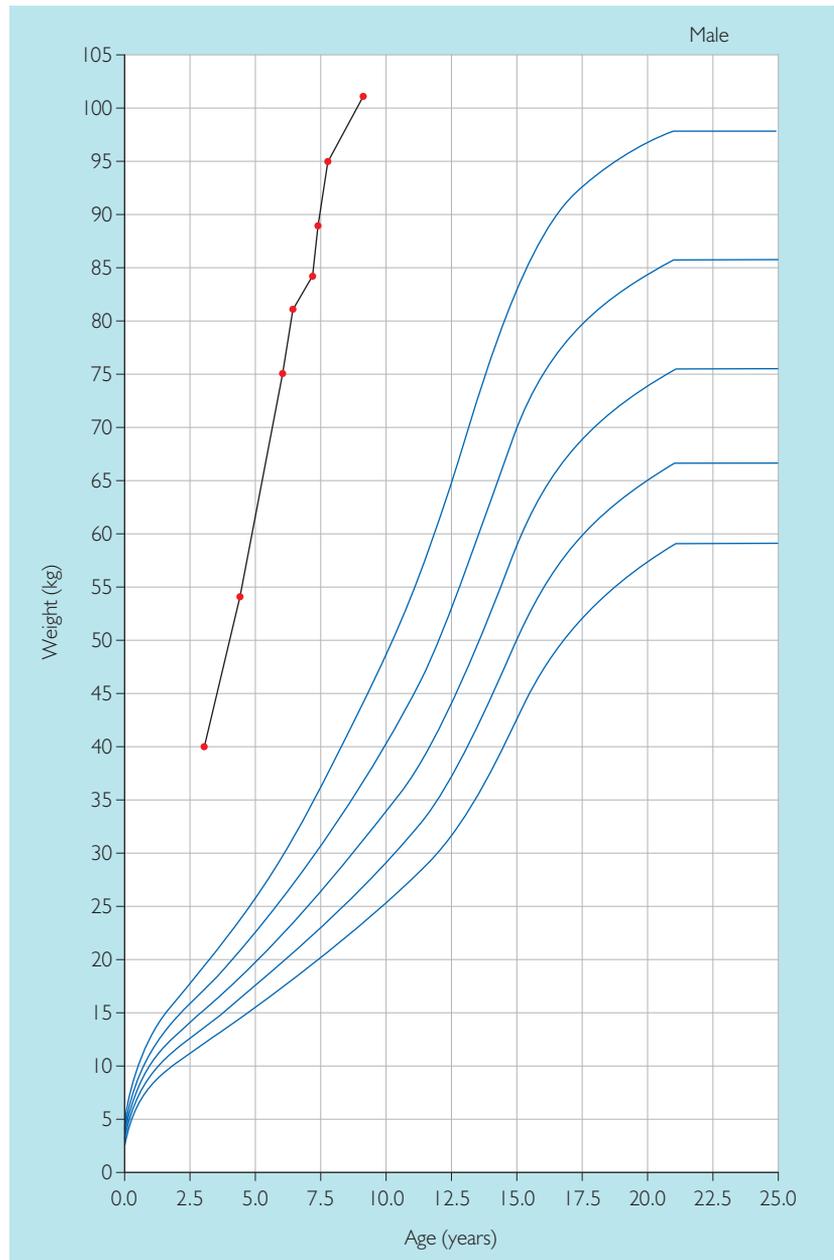
it may be difficult to get reliable information. More detailed and reliable information can be obtained by asking the child and family to fill a dietary diary and activity log, although compliance and accuracy may still be a problem.

A detailed psychosocial history is needed to understand the development of obesity, as well as the severity of the psychosocial consequences of obesity.

Interpretation of the clues

■ Low height velocity points to hypothyroidism, Cushing syndrome or GH deficiency (see Ch. 2).

■ A growth curve above the target height centile with mildly increased height velocity excludes these three endocrine causes and is compatible with 'simple' obesity (**Figs 5.20 & 5.21**).

■ Calculating the weight gained in relation to height gained over a specified period can allow an estimation of the excess calorie intake; 1 kg fat is deposited by 9000 kilocalories (kcal) of excess intake (and releases 7000 kcal energy (28 MJ) when utilized). Therefore a gain in weight of 5 kg more than expected for the height gained over 1 year is equivalent to

Fig. 5.21 Same patient as in Fig. 5.20 with height +3.5 SDS, compared with target height of 0 SDS. Advanced bone age and early pubertal changes at 9 years mean that final height is likely to be within the normal centile range but above target height.
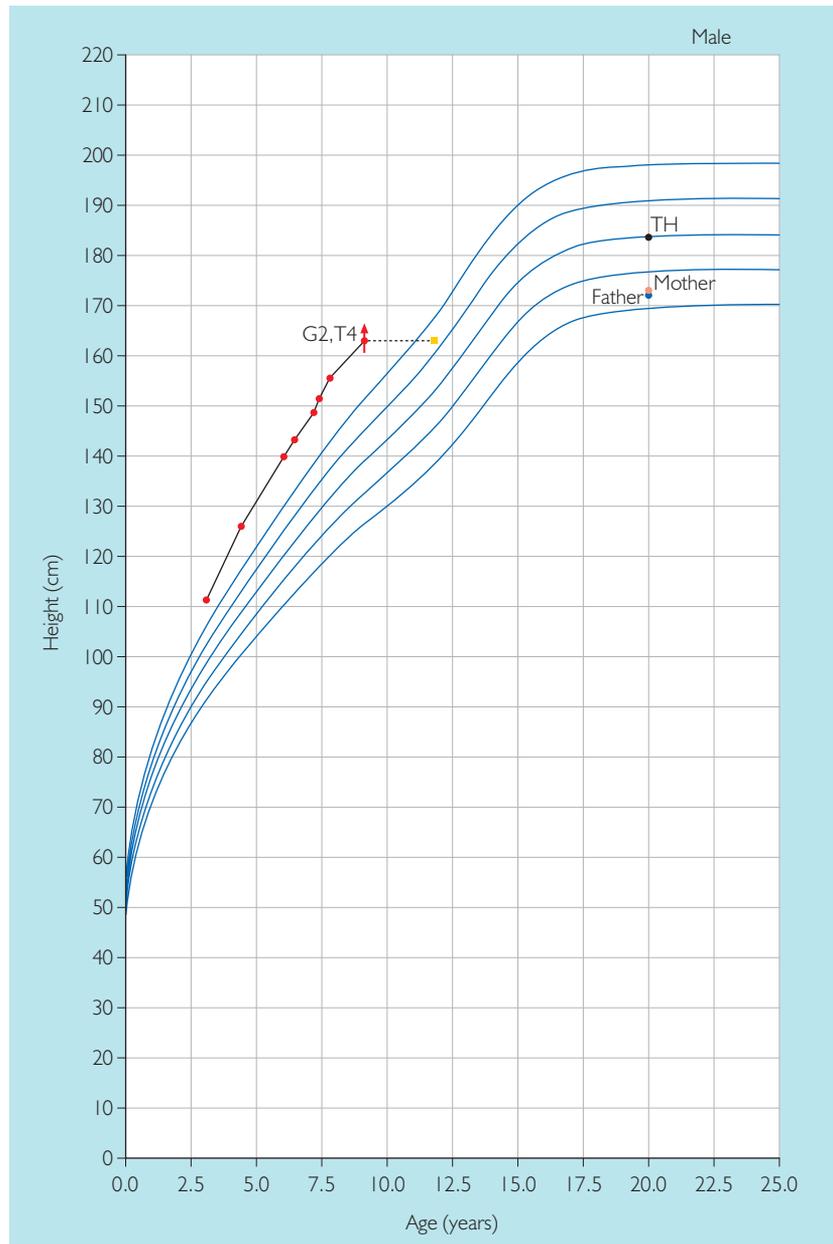
$5 \times 9000 = 45\ 000/365 = 123$ kcal per day excess to requirements, or about half a bag of potato chips.

- Polydipsia and polyuria = diabetes mellitus (type 2); rarely, diabetes insipidus.
- Headache, neurologic symptoms and signs = suspicion of CNS cause.
- Hypertension, plethora = Cushing syndrome.
- In female adolescents menstrual irregularities, hirsutism, acne, acanthosis nigricans = PCOS.
- Hirsutism = PCOS or Cushing syndrome.
- Fat distribution – a central (masculine, android) distribution is associated with an increased risk for cardiovascular morbidity and can be found in Cushing syndrome.
- A peripheral (feminine, gynoid) pattern is predominantly located in the lower body.
- Acanthosis nigricans = insulin resistance.
- Early hypotonia, small hands and feet, intellectual impairment, marked increase in energy intake after age 2 years = Prader–Willi syndrome.
- Polydactyly, retinitis pigmentosa = Laurence–Moon–Biedl syndrome.
- Hypogonadism = Prader–Willi syndrome, Laurence–Moon–Biedl syndrome, GH deficiency, leptin deficiency or CNS cause.

- Neonatal hyperinsulinemic hypoglycemia and macrosomia in infancy, visceromegaly, macroglossia = Beckwith–Wiedemann syndrome.
- Early onset of voracious appetite, parental consanguinity, a family history of hypogonadism and extreme obesity = leptin deficiency or receptor abnormality.
- Red hair with early onset of voracious appetite, parental consanguinity and extreme obesity = POMC abnormality.

INVESTIGATIONS

If height is within the population and target ranges (particularly if it is in the upper range of the target range), and height velocity over the foregoing years is normal, an endocrine cause is virtually excluded and the likelihood of the diagnosis of simple obesity is very high. In the absence of other clues from the history and examination, except for clear indications that calorie and fat intake is too high in relation to energy expenditure, *further laboratory investigations are not strictly necessary.* Documentation of metabolic abnormalities associated with overweight may be helpful, as abnormal values may reinforce the wish of the child or adolescent and parents to work hard to lose weight. This can include liver function, fasting triglycerides, low and high density lipoproteins and total cholesterol, glucose, insulin and glycosylated hemoglobin levels. A bone age assessment does not provide much additional information, although it may be useful in predicting the age of pubertal onset.

Subclinical hypothyroidism can be excluded by measuring the level of thyroid stimulating hormone (TSH).

In the well growing, obese, adolescent female, if menstrual irregularities are reported, possibly combined with signs of hirsutism and/or acanthosis nigricans, investigation for PCOS may be required:

- serum estrogens (E_1, E_2), androgens (testosterone, androstenedione, dehydroepiandrosterone sulfate (DHEAS)), SHBG and gonadotropins (LH, follicle stimulating hormone (FSH), either basal or stimulated). Classically, raised serum E_1, normal E_2, low SHBG, raised testosterone and free testosterone and/or androstenedione concentrations are found, as well as an LH level that is considerably higher than that of FSH. Abdominal sonography may show ovarian cysts but is not necessary for diagnosis in the presence of other features.

If the child is short, or the height velocity is low, then hypothyroidism, Cushing syndrome, GH deficiency and pseudohypoparathyroidism need exclusion (see Chs 2, 9 & 11, and Appendix).

If there are signs of Prader–Willi syndrome, specific genetic analysis is performed for deletion of the paternal allele at 15q11-13 or for a duplication of the maternal allele at this locus.

Serum leptin can be assayed in research protocols searching for rare genetic causes of obesity.

TREATMENT

The large increase of the prevalence of overweight and obesity in past decades would suggest that lifestyle changes, in particular diet and physical activity, should be the targets for preventive intervention at a population level. However, there are currently few societies where this action is being taken in a concerted manner.

At the same time, this suggests that treatment for simple obesity should consist of limiting calorie and fat intake, and stimulating physical activity. In practice, however, it appears very difficult for children and adolescents to lose weight successfully.

If the child or adolescent has hypertension, hepatitis, hyperlipidemia or glucose intolerance, an active approach toward weight reduction should be taken. This should ideally consist of a closely supervised diet and exercise plan in a family-based behavior modification program.

However, even in the absence of such abnormalities, treatment is indicated, as childhood obesity leads to approximately 30% of adult obesity and the obese child who becomes an obese adult will have more severe adult obesity than those whose obesity begins in adulthood. Long-term follow-up of obese adolescents has demonstrated that cardiovascular mortality and morbidity rates are increased compared with those in lean adolescents. The most effective treatment is a multidisciplinary, comprehensive and family-oriented approach. This includes a combination of behavioral modification procedures, education, dietary intervention with calorie restriction, and an activity and exercise program. However, such treatment programs are time consuming for patient, parent and health personnel, and unfortunately there is a considerable drop-out rate and a high rate of recidivism to obesity in the following years. For a more successful long-term outcome the child or adolescent, and if possible the parents, should change or modify attitudes, beliefs and behavior with respect to eating and physical activity.

Dietary therapy should focus on a calorie-restricted diet of normal foods that is balanced to provide the

conventional distribution of carbohydrate (approximately 55% of total calories), fat (approximately 30%) and protein (approximately 15%). Intensive 'fad diets', as widely advertised for adults, should not be used in children and adolescents, because of health hazards. Moreover, such diets do nothing to modify the patient's eating habits so do not lead to sustained weight loss. While a decrease in physical activity is an important factor in the current increase of obesity, exercise is not an effective means of inducing weight loss unless combined with the reduction of calorie intake. Jogging for 2 km expends only as much energy as is contained in half a bar of chocolate. Exercise may have a greater impact on the maintenance of weight loss and fitness level.

Perversely, in societies where overweight is becoming more common the fear of obesity and the desire for a largely unattainable ideal body image is leading to unnecessary and potentially harmful dietary restriction in small growing children by well-meaning parents, and to a substantial increase in eating disorders amongst adolescents.

Fig. 5.22 Weight changes after resection of craniopharyngioma at age 7 years. Hypopituitarism treated with growth hormone, hydrocortisone and thyroxine, with induction of puberty at 13.5 years. Adult height –0.6 SDS. Relentless weight gain despite dietary intervention and introduction of orlistat at age 15.2 years for a 6-month trial. Patient is now being considered for gastric bypass surgery.
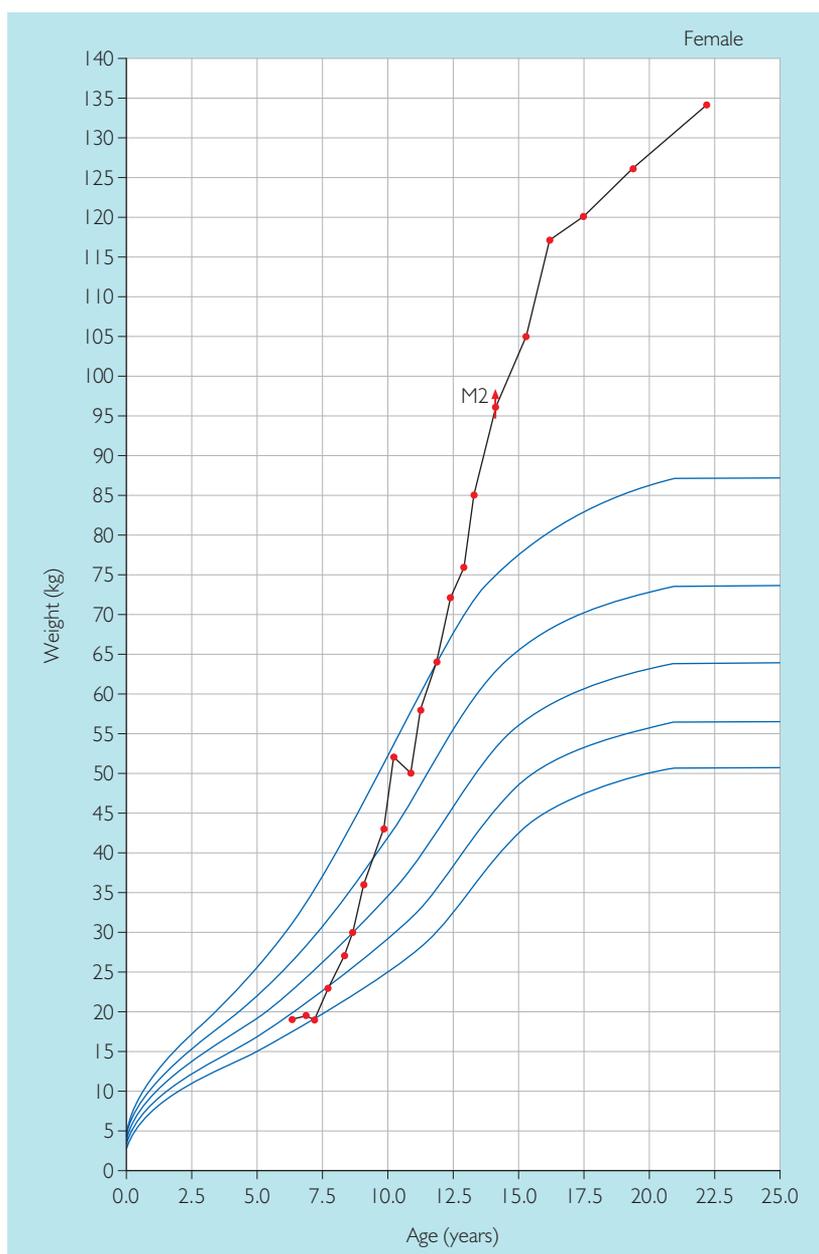
There is little experience of the use of pharmaceutical means of weight reduction in children. Orlistat is an intestinal lipase inhibitor that provokes malabsorption of ingested fat. It has been shown capable of helping adults to sustain dietary weight loss of as much as 10% in some studies, but provokes gastrointestinal symptoms unless fat intake is severely restricted, which may be unsafe in a growing child. Sibutramine is a centrally active appetite suppressant that is used as an adjunct to diet in adult obesity with some success. However, long-term safety is an issue and these agents should not currently be prescribed in childhood outside established research programs. Surgical management of obesity is common in adulthood in some centers and may be the best option for potentially life-saving treatment in extreme morbid obesity (i.e. adult BMI >40), despite the anesthetic risks; however, experience in children is very limited. The massive obesity seen after surgery for craniopharyngioma makes this and Prader–Willi syndrome the most likely pathology to require drug or surgical intervention in pediatric practice (**Fig. 5.22**).