10: Management of obesity

Joseph Proietto and Louise A Baur

Improved understanding of the regulation of body weight and activity is leading to new treatment strategies

OBESITY IS THE EXCESS accumulation of body fat. It can be assessed by the body mass index (weight in kilograms divided by the square of height in metres [BMI]). The relationship of BMI to total body and visceral fat, and consequent complications, varies between ethnic groups[1] (Box 1). Asian populations, particularly those from South East Asia and the Indian subcontinent, have more fat and more comorbidities for any given BMI, resulting in different suggested BMI cut-off points.[3] In contrast, Polynesians have higher BMI cut-off points.[4]

The risk of comorbidities rises with increasing BMI, with a mild rise in the overweight range, moderate in class I, severe in class II and very severe in class III obesity. As well as the total fat, distribution is important to the incidence of comorbidities. Central abdominal fat, particularly visceral fat, is a risk factor for the metabolic syndrome. Waist circumference cut-off points in white European populations are shown in Box 2.

Adult BMI cut-off points cannot be used for assessing children and adolescents, as BMI varies throughout childhood, being high in the 2nd year of life, dropping to a nadir at age 4–7 years and then rising again to adult values (Box 3). BMI-for-age charts, such as those developed by the Centers for Disease Control and Prevention in the United States,[6] can be used in clinical practice to assess and monitor BMI over time in children (Box 3). Overweight is defined as BMI between the 85th and 95th percentiles, and obesity as BMI > 95th percentile,[7] but these definitions are arbitrary, as, unlike adult BMI cut-offs, they are not linked to morbidity data. Although a table of age- and sex-specific cut-offs developed for epidemiological research[8] allows international comparison of trends in overweight and obesity in children, it is not intended for routine clinical use.

In children and adolescents, as in adults, waist circumference is strongly correlated with abdominal fat and markers for comorbidities, such as adverse lipid and glucose profiles and hypertension.[9] However, there are no internationally accepted

ABSTRACT

- Obesity has reached epidemic proportions in Australia, with 67.5% of men, 52.1% of women and 19%–23% of children and adolescents being overweight or obese.

- Genetically predisposed individuals are especially vulnerable to developing obesity in the highly obesogenic environment of 21st century Australia.

- Obesity causes or contributes to many comorbidities, including type 2 diabetes, hypertension, dyslipidaemia, sleep apnoea, non-alcoholic steatohepatitis, orthopaedic problems and polycystic ovary syndrome.

- Management in the individual requires their complete co-operation and should be tailored to individual needs and complications.

- Management of obesity in children should consider the family context and involve the parents.

- All treatment strategies must involve lifestyle modification, with a reduction of energy intake and an increase in physical activity.

- Some patients may also require the assistance of drug therapy or bariatric surgery.

criteria for waist circumference in this age group and, as for adults, racial and ethnic variations exist. For example, African American, Mexican American and Mohawk Indian children carry more abdominal fat than white children.[10]

The epidemic

The prevalence of obesity appears to have more than doubled in Australian adults in the decades from 1980 to 2000. A 1980 survey of people aged 25–64 years living in capital cities found that 7.1% were obese.[11] In contrast, the 1999–2000 Australian Diabetes, Obesity and Lifestyle (Aus-Diab) study found in the same urban age group that 18.4% were obese, a 2.5-fold increase.[12] Overall, the AusDiab study found, among 11 000 adults from around Australia, that 48.2% of men and 29.9% of women were overweight, while 19.3% of men and 22.2% of women were obese.[12]

The increase in prevalence of overweight and obesity is even more striking among Australian children.[13] Over the decade 1985–1995, the combined prevalence of the two conditions in children almost doubled (from 10.7% to

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1: Definitions of obesity in adults[2]

| | Body mass index (kg/m²) | | |
Classification	White European	Asian	Polynesian
Underweight	< 18.5	< 18.5	
Normal range	18.5–24.9	18.5–22.9	
Overweight	25.0–29.9	23.0–25.9	26.0–31.9
Obese	> 30.0	> 26.0	> 32.0
Class I	30.0–34.9	26.0–29.9	
Class II	35.0–39.9	30.0–35.0	
Class III	> 40.0	> 35.0	
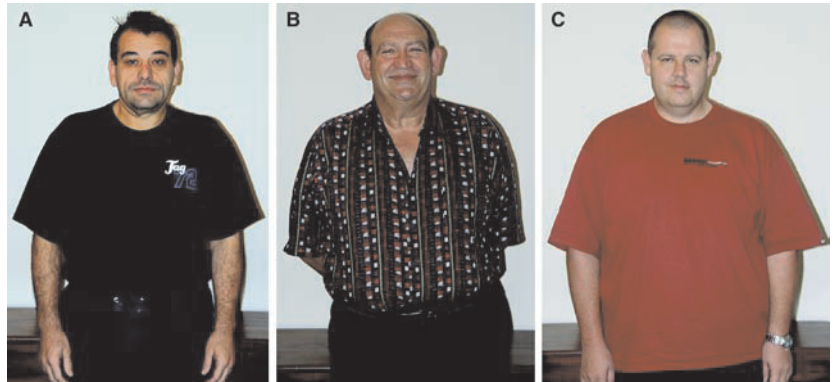
Patient photographs demonstrate different classes of body mass index (BMI). A: 33 kg/m² (class I obesity). B: 38 kg/m² (class II obesity). C: 45 kg/m² (class III obesity). (Photos courtesy of Dr J Dixon and patients, Monash University, Centre for Obesity Research and Education, Melbourne, VIC.)

19.5% in boys and from 11.8% to 21.1% in girls), while that of obesity on its own more than tripled (from 1.4% to 4.5% in boys and from 1.2% to 5.3% in girls).[13]

Aetiology and effects of obesity

The aetiology of obesity is complex, with both environmental and genetic influences. The recent increase in prevalence is clearly due to the continuous availability of high-energy foods, together with a major reduction in the obligatory need for physical activity that has characterised human existence until very recently.

However, not everyone in an obesogenic environment becomes obese, indicating that a genetic predisposition is required. Studies of twin pairs reared together or apart suggest that about 70% of the influence on body weight is genetic, while about 30% is environmental.[14] The dominance of genetic influences has been confirmed by adoption studies, which found that adoptees resemble their biological parents in body size, with very little resemblance to their adoptive parents.[15] Some genes that could predispose to obesity have been identified (including genes encoding leptin, the leptin receptor, pro-opiomelanocortin and the melanocortin-4 receptor), but many more are probably as yet undiscovered.[16]

In children, environmental contributors to obesity include prolonged television viewing, playing of computer and video games,[17] decreased physical activity (especially incidental activity) and increased consumption of energy-dense foods and sugar-containing drinks.[18]

Obesity has been linked to complications in many body systems (Box 4).

Preventing obesity

The magnitude of the problem of obesity in most westernised countries mandates population-level strategies. Interventions focusing on simply educating individuals and communities about behaviour change have had limited or no success.[19] Instead, there is a need to produce an environment that supports healthy eating and physical activity throughout the community.

A recent systematic review of interventions for preventing obesity in children concluded that there are "limited high quality data on the effectiveness of prevention programs".[20] This may well reflect the methodological and ethical challenges of conducting such studies, often in a sociopolitical environment not conducive to change. However, the review did highlight the potential effects of reducing sedentary behaviours and increasing physical activity.

Possible strategies include:

- regulating the nature and amount of food advertising directed at children;
- providing high-quality recreation areas, safe cycle paths and safe street lighting in local neighbourhoods;
- improving public transport;
- providing economic incentives for production and distribution of vegetables and fruit; and
- developing town planning policies that promote active or public transport over private motor cars.

Such multifaceted large-scale interventions require cooperation and support from many sectors of society and government, with adequate resourcing and significant community ownership.

Lessons may be learnt from previous successful campaigns for long-term social change, such as tobacco control and the

2: Waist circumference associated with increased risk of metabolic complications in white European adults*

	Increased risk	Substantially increased risk
Men	≥ 94 cm	≥ 102 cm
Women	≥ 80 cm	≥ 88 cm

* Reported by the World Health Organization,[2] based on a study of 4881 adults in the Netherlands.[5] Note that waist circumference cut-offs are population-specific.

promotion of breastfeeding.[21] However, as few previously studied behaviours are driven by biological drives as powerful as the drive to eat, it is imperative that the efficacy of any public health measure be tested before it is widely applied.

Individual therapy for obesity

A weight loss and maintenance strategy for individual patients is outlined in Box 5. The weight-loss phase involves establishing a negative energy balance by reducing energy intake and increasing physical activity. Most motivated patients can succeed in this phase, but different strategies suit different individuals.

The next phase, weight maintenance, is more difficult and requires more emphasis. Lifestyle modification plus pharmacotherapy, if required, offer the best chance of success. For those who have failed in weight maintenance, bariatric surgery currently produces the best results and should be considered.

Clearly, it is important to set realistic goals from the start. Weight loss that is useful from a medical point of view (5%–10%) often does not satisfy patients. It may help to point out that more modest losses may be easier to maintain in the long term.

3: Example of chart of body mass index (BMI) for age[6]
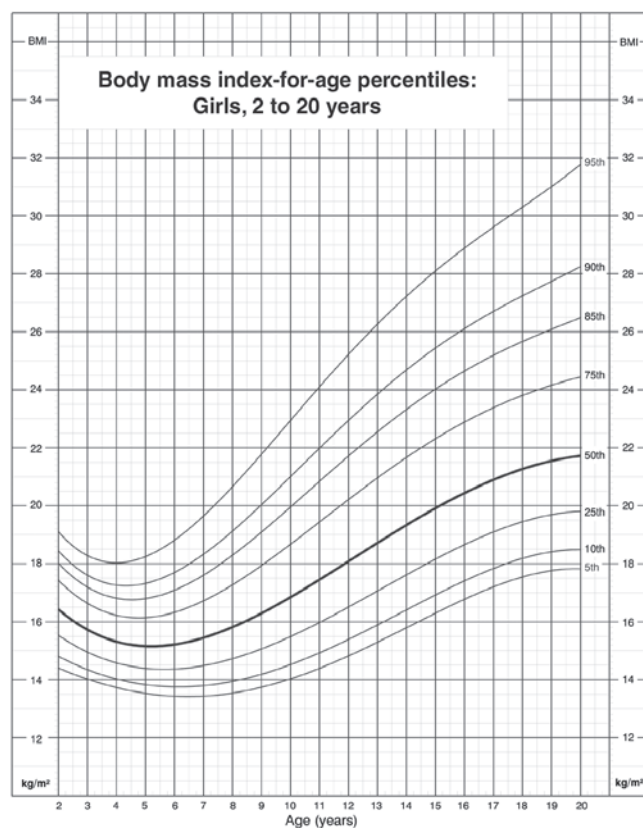
Charts available from Centers for Disease Control and Prevention, National Center for Health Statistics www.cdc.gov/growthcharts

Lifestyle change in adults

It is important to ensure that patients are ready and enthusiastic about attempting weight loss, as their cooperation is essential. A negative energy balance must be established, by reducing energy intake and increasing energy expenditure. A practical way to reduce energy intake is to limit fat intake. Patients may need information about foods that contain fat. It may help to also limit carbohydrates, especially those with a high glycaemic index (rapidly digested and absorbed carbohydrates), as high insulin levels can encourage weight gain[22,23] (see also National Health and Medical Research guidelines on managing obesity,[7] section 5, available at www.obesityguidelines.gov.au/pdf/adults_part5.pdf). A reduction of 2500 kJ (600 calories) from the stable prior intake is generally advised, which should lead to weight loss of 0.6 kg per week. This can be calculated from a diet diary kept over a week before starting treatment. The advice of a trained dietitian will be of great help.

4: Complications of obesity

Cardiovascular: Hypertension, dyslipidaemia, increased risk of coronary heart disease and stroke

Respiratory: Obstructive sleep apnoea, asthma

Endocrine: Glucose intolerance, insulin resistance, type 2 diabetes, polycystic ovary syndrome

Orthopaedic: Back pain, osteoarthritis, flat feet

Dermatological: Acanthosis nigricans, skin tags, intertrigo

Gastrointestinal: Non-alcoholic steatohepatitis, reflux oesophagitis, gallstones

Psychosocial: Social isolation and discrimination, decreased self-esteem, binge-eating disorder, bulimia, and depression

Other: Increased risk of breast and other cancers, increased intracranial pressure, proteinuria

5: Management of obesity in motivated adult patients

Step 1. History, examination and investigation
(plus management of comorbidities if not resolved by weight loss)

Step 2. Weight loss (3–12 months)
- Lifestyle modification (decrease energy intake and increase physical activity)
- Diet
  - ➤ Low-fat, reduced-carbohydrate diet
  - ➤ Very low energy diet (VLED) (commercial, over-the-counter mixture of essential nutrients with defined energy content, usually supplied as a powder to be mixed with water and drunk three times daily)
  - ➤ Meal replacement program (eg, commercial program that provides preprepared meals)
  - ➤ Commercial weight loss centre

Step 3: Weight maintenance (lifelong)
- Lifestyle modification
- Pharmacotherapy (if required)

Step 4: Bariatric surgery
(for obese patients who have failed medical therapy)

6: Case report — an overweight adolescent

Presentation: A 13-year-old girl presented to her general practitioner with a respiratory tract infection. During the consultation, her mother commented that she was concerned about her weight and was being teased about this at school. She had left her previous school because of bullying. Aware of the time needed to discuss this issue, the GP made a separate appointment for the girl and her mother to return.

History: The girl was an only child and appeared to have good relationships with her parents and peers. Her general health was good. Several family members were obese (mother, maternal aunts, three grandparents), and the mother described the family as "heavy-boned". However, there was no significant family history of other disorders associated with insulin resistance (eg, type 2 diabetes, premature heart disease). The girl's lifestyle was sedentary, with her main interests being playing music, sewing, reading and talking on the telephone. She was driven to and from school and watched about 3 hours of television per day. Her diet included at-risk features, such as occasionally skipping breakfast, consuming full cream milk, "something nice" for morning and afternoon tea and about 500 mL of soft drink per day, regularly buying food from a milk-bar in the afternoon, and "grazing" at home (eg, on biscuits from the cupboard).

Examination: Weight was 72.6 kg (> 97th percentile) and height 161.5 cm (< 75th percentile), giving a body mass index (BMI) of 28.0 kg/m^2 (> 95th percentile for age; adult overweight range). Her waist circumference was 85 cm (adult "at risk of metabolic

complications" range). She was in mid-puberty, with no striae on the abdomen or upper thighs. Blood pressure was 120/80 mmHg.

Investigations: A fasting blood test showed normal lipid profile, liver function, glucose and insulin levels. Urinalysis gave normal results.

Management: The GP arranged to see the girl and her mother together and separately, initially every 3 weeks and then less often. Two visits to a local dietitian were arranged; a long waiting list made further follow-up impossible. The girl was encouraged to set her own goals for food and activity changes, which were discussed with the GP at each consultation. The family was supported in changing their eating patterns and television use. The dietitian looked with the girl at ways to recognise and deal with eating cues.

Course: Over time, the mother herself started to lose weight because of altered cooking practices and being more active. Water rather than soft drink was offered at the evening meal, biscuits and less-healthy snacks were no longer stored in the cupboards, and the whole family moved to eating more vegetables and having smaller meat portions at the evening meal. The girl ate something for breakfast each morning and started walking to and from school each day. She began tennis lessons and found an interest in tap dancing.

Ten months later, the girl's weight was 69.3 kg, height 163.0 cm, BMI 26.1 kg/m^2, and waist circumference 80 cm. She reported being fitter and said that she was greatly enjoying school and was no longer being bullied.

Increased physical activity is an important component of lifestyle modification. The increase must be substantial (80 minutes of moderate-intensity activity per day), but cannot usually be achieved immediately. A more modest initial target can be set (eg, 30 minutes of walking 3–5 days per week) and increased gradually. While reduced energy intake is the major method of losing weight, it has been shown that a high level of physical activity is essential to assist with maintaining weight loss. Irrespective of its impact on weight, physical activity has wider benefits on well-being, including improved cardiovascular fitness.

A Cochrane review on the effectiveness of intervention by health professionals in weight management concluded that "there are few solid leads about improving obesity management, although reminder systems, brief training interventions, shared care, in-patient care and dietitian-led treatments may all be worth further investigation".[24]

Lifestyle change in children and adolescents

For children and adolescents, the broad principles of management are well recognised and comprise behaviour modification (eg, patterns of television watching, computer use, socialising and leisure activity), family support, dietary change, increased physical activity, decreased sedentary behaviour, and a developmentally appropriate approach.

To be effective, therapy must take account of family influences on food and activity habits.[24] Parental involvement is necessary with both young children and adolescents. Several studies have now shown that long-term (2–10-year) maintenance of weight loss can be achieved with family-based intervention.[25] There is no direct evidence on which dietary modification is most effective in this age group. Interventions should follow national nutrition guidelines and emphasise lower-fat options, increasing vegetable and

fruit intake, healthier snack-food choices and probably decreased portion sizes. Reducing soft drink and fruit juice intake is also important.[18]

Participation in an exercise program is a long-term predictor of successful weight control in children.[26] The type of exercise appears important for sustained weight loss. While both "lifestyle" (eg, walking and cycling) and programmed aerobic exercise promote weight loss in the initial phase, "lifestyle" exercise is more likely to be continued long term.

In pre-adolescents, an approach using parents as the exclusive agents of lifestyle change appears superior to a child-centred approach, where the child is expected to take major responsibility for making changes.[27,28] Thus, sessions involving one or both parents without the child are likely to be the most effective.

In adolescents, features of successful interventions include separate sessions for the adolescent and parent, and a structured but flexible program that encourages sustainable modifications in lifestyle, relationships and attitudes[29] (see case report, Box 6). There is also a report of success, at least in the short term (3 months), with a 4-month behavioural weight control program for overweight adolescents initiated in a primary care setting and extended through telephone and mail contact.[30]

The National Health and Medical Research Council recently published clinical practice guidelines for management of overweight and obesity in children and adolescents, which review in detail the evidence on interventions.[31]

Drug therapy

Role of leptin

The use of drugs to assist in management of obesity has always been controversial. This is partly due to the widespread belief that losing weight and maintaining weight loss

7: Case report — relapsing obesity in an adult

Presentation: A 42-year-old woman was referred for management of severe obesity and type 2 diabetes.

History: She had been thin as a child but started to gain weight after the birth of her first child, 20 years before. She had attempted weight loss several times previously, at one time losing 30 kg using a commercial weight-loss program, but always regained the weight. Two years prior she was diagnosed with diabetes after presenting with polyuria and thrush. This was treated with metformin (1000 mg twice daily) and glibenclamide (5 mg once daily). She had a past history of hypercholesterolaemia and hypertension, treated with atorvastatin (40 mg in the morning) and ramipril (2.5 mg in the morning), and pre-eclampsia during one pregnancy. She had a maternal history of obesity and diabetes. She was a non-smoker. She was married with two daughters and was a full-time mature-age university student.

Examination: She appeared well. Weight was 114.5 kg, height 1.65 m (body mass index, 42.1 kg/m^2, corresponding to obesity class III), waist circumference 125 cm, and hip circumference 131 cm. Pulse was regular at 98 beats/min, blood pressure was 130/90 mmHg. She had mild acanthosis nigricans around the neck and a few skin tags. Cardiorespiratory examination revealed no abnormalities. Pulses were difficult to feel. There was no evidence of microangiopathy.

Investigations: Serum levels were: glycosylated haemoglobin, 10.2% (reference range [RR], < 6.1%); total cholesterol, 4.8 mmol/L (RR, < 5.6 mmol/L); triglyceride, 3.3 mmol/L (RR, < 2.6 mmol/L); high-density lipoprotein (HDL) cholesterol, 0.86 mmol/L (RR, > 1.0 mmol/L); low-density lipoprotein (LDL) cholesterol, 3.34 mmol/L (RR, < 3.51 mmol/L); LDL/HDL cholesterol ratio, 3.9 (RR, < 3.6). Serum levels of electrolytes, urea and creatinine, and liver function results, were in the reference range.

Management: The patient said that she was ready to attempt weight loss as she understood that obesity was contributing to her diabetes, hypertension and dyslipidaemia. A diet and exercise regimen was discussed, and she was referred to a dietitian for help in calculating a 2 500 kJ (600 calorie) deficit diet. She undertook to try to increase her physical activity.

1-month review: The patient said she was struggling, as combining full-time study, part-time work and maintaining a household left little time for exercise and, although she tried to change her diet, she found it difficult not to snack. She said she had found it easier to diet when she was younger. Her diabetes had not improved.

Management: Given the patient's medical comorbidities, it was worth considering more aggressive management of obesity.

Gastric banding was discussed. She had considered this after seeing it on television. However, she had no private insurance, and waiting lists at the few public hospitals that perform the operation are up to 5 years.

A medical strategy was adopted. She began a 3-month course of:

- A very low energy diet (VLED), comprising a sachet of essential nutrients mixed with water three times daily (providing vitamins, minerals, amino acids, essential fatty acids and 1914 kJ [456 calories] per day). This was purchased over-the-counter from a pharmacist.

- A bowl of vegetables or salad (to supply roughage) sprinkled with a teaspoon of olive oil (to contract the gall bladder) in the evenings.

She stopped taking glibenclamide, both to remove the risk of hypoglycaemia and to allow insulin levels to fall, producing mild ketosis to help control hunger. She was asked to have serum electrolytes, urea, creatinine and uric acid levels measured 6 weeks into the diet. She saw the dietitian fortnightly.

3-month review: After 3 months of the diet she had lost 17 kg, and blood glucose levels had improved dramatically, with glycosylated haemoglobin dropping to 7.9%.

Management: She was advised to phase out the VLED over the next 2 months and to adopt a low-fat, low-carbohydrate diet. However, as previous lost weight had always been regained, sibutramine (10 mg, mornings) was prescribed to assist weight maintenance. She reported it had a noticeable effect on hunger and satiety, but she was struggling financially because of its cost ($120 per month).

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is simply a matter of exercising free will, which has even led to a suggestion that it is unethical to be obese.[32] However, this view cannot explain why the overwhelming majority of obese individuals regain weight after successful weight loss,[33] despite an often desperate desire to remain lean. The discovery of the hormone leptin in 1994 shed light on this paradox.

Leptin is a cytokine hormone produced in fat cells in proportion to their size. It is secreted into the bloodstream and crosses the blood–brain barrier via an active, saturable process. It acts in the hypothalamus to alter the expression of neurotransmitters (eg, it inhibits production of neuropeptide Y and stimulates production of melanocyte-stimulating hormone), resulting in suppression of hunger and an increase in spontaneous activity. Hence, as weight increases, fat cells increase in number and size, increasing the production of leptin, which feeds back to inhibit food intake and increase energy expenditure, limiting weight gain. It appears that a genetically lean individual will gain an extra 7–8 kg before leptin increases sufficiently to stop the weight gain. Individuals who gain more than this amount must be unresponsive to the hormone, either because it cannot enter the brain efficiently or because there is a mutation in one of the many steps required for leptin action, such as at the

melanocortin receptor. When obese individuals with a high leptin level lose weight, the leptin level falls dramatically,[34] resulting in relative leptin deficiency.

It is known from a study of two children with a homozygous mutation in the leptin gene that leptin deficiency leads to insatiable hunger.[35] Thus, it is likely that weight-reduced obese individuals regain weight despite a great desire to remain lean because they cannot tolerate hunger long term in the face of abundant food. This has led to reappraisal of the role of drugs and surgery in management of obesity (see case report, Box 7).

Available drugs

Drugs available for treating obesity are shown in Box 8. Both the intestinal lipase inhibitor orlistat and the serotonin- and noradrenaline-reuptake inhibitor sibutramine have been shown to limit weight regain in large randomised placebo-controlled trials.[37,38] The lack of long-term studies of the noradrenergic agonists phentermine and diethylpropion limits their usefulness in long-term management.

Some antidepressants affect body weight, including the selective serotonin-reuptake inhibitors (fluoxetine, paroxetine, fluvoxamine, citalopram and sertraline) and the serotonin- and noradrenaline-reuptake inhibitor venlafaxine. The

8: Drugs currently available to treat obesity

Drug	Function	Side effects	Weight loss v placebo*	Comments
Phentermine, diethylpropion	Noradrenergic agonists that stimulate adrenergic pathways, suppressing hunger	Insomnia, dry mouth, nervousness, irritability, increase in blood pressure, angina	6.3 kg v 2.8 kg	No studies supporting long-term use.
Orlistat	Inhibits intestinal lipase, reducing fat absorption by 30%	Steatorrhoea, bloating, faecal leakage, especially if excess fat is eaten	7.1 kg v 5.0 kg	Unpleasant side effects may encourage adherence to low-fat diet.
Sibutramine	Metabolites inhibit serotonin- and noradrenaline-reuptake, enhancing satiety and suppressing hunger	Dry mouth, constipation, insomnia, increase in blood pressure (usually small) and heart rate	5.3 kg v 1.8 kg	Should be avoided in patients with uncontrolled hypertension or angina. Effects on cardiovascular endpoints (stroke, myocardial infarction) unknown.
Fluoxetine	Selective serotonin-reuptake inhibitor that inhibits hunger	Seizures, nausea, insomnia, sexual dysfunction	4.1 kg v 0.8 kg	Not approved for treating obesity but drug of choice for treating depression in overweight patients.

*From Haddock et al, meta-analysis of weight loss studies.[36]

effects of fluoxetine are best studied, with several studies showing a modest but reproducible effect on weight loss. These drugs are not approved for treating obesity, but should be the drugs of choice when treating depression in overweight patients.

Many compounds are currently undergoing clinical trials including:

- leptin and leptin analogues;
- topiramate, an antiepileptic drug with appetite-suppressant action;
- rimonabant, an inhibitor of the cannabinoid-1 receptor;
- amylin, a protein secreted by pancreatic beta cells; and
- AOD 9604, a fragment of the growth hormone molecule.

Many other compounds are in earlier stages of development.

Surgery

Surgery should be considered for patients with significant comorbidities associated with obesity, especially if medical therapy has failed repeatedly. Studies in Australia[39] and overseas[40] have shown major benefits of gastric surgery on weight loss, diabetes, hypertension, dyslipidaemia, insulin sensitivity, sleep apnoea, asthma, infertility and quality of life. For example, a study of 50 patients with type 2 diabetes and an initial mean BMI of 48 kg/m^2 found that, a year after laparoscopic gastric banding, fasting serum glucose level had decreased from 9.4 to 6.2 mmol/L, glycosylated haemoglobin level from 7.8% to 6.2%, serum triglycerides from 2.4 to 1.4 mmol/L, and blood pressure from 154/96 to 130/79 mmHg, while high-density lipoprotein cholesterol level had increased from 1.03 to 1.22 mmol/L.[41] Of the 29 patients who had been taking oral hypoglycaemic agents, only eight still required these, after an average 27 kg weight loss. While laparoscopic gastric banding (Box 9) is becoming the favoured approach because of its reversibility and low morbidity, some surgeons still perform gastric stapling or biliopancreatic diversion with good outcomes.[42] Biliopancreatic diversion is indicated when previous restrictive surgery has failed and could be considered in superobesity.

9: Laparoscopic gastric banding
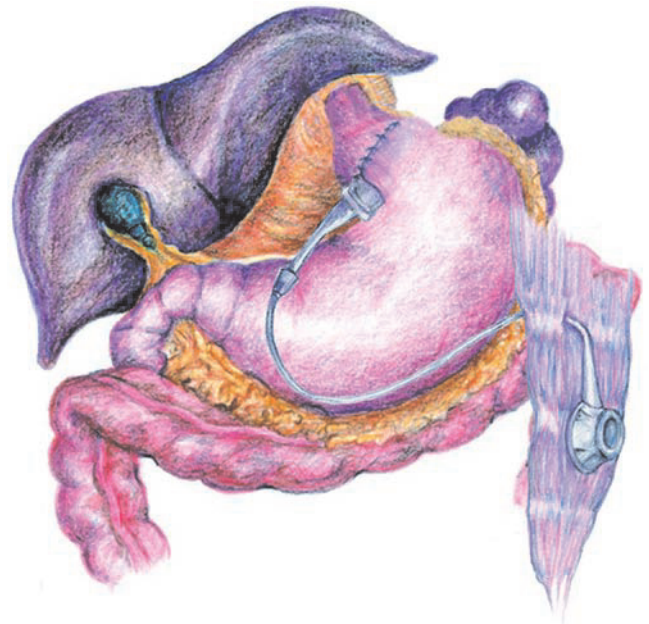
An adjustable band is sutured around the very upper stomach. The restriction on the stomach can be modified by adding or removing saline from a reservoir on the rectus sheath. This procedure can be performed periodically in the office. (Illustration courtesy of Dr J Dixon, Monash University, Centre for Obesity Research and Education, Melbourne, VIC, and InaMed Health, Santa Barbara, USA)

References

1. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. *Int J Obes Relat Metab Disord* 1998; 22: 1164-1171.

2. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation on Obesity. Geneva, 3-5 June 1997. Geneva: WHO, 1998.

3. Ko GT, Tang J, Chan JC, et al. Lower BMI cut-off value to define obesity in Hong Kong Chinese: an analysis based on body fat assessment by bioelectrical impedance. *Br J Nutr* 2000; 85: 239-242.

4. Swinburn BA, Ley SJ, Carmichael HE, et al. Body size and composition in Polynesians. *Int J Obes Relat Metab Disord* 1999; 23: 1178-1183.

5. Han TS, Van Leer EM, Seidell JC, et al. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *BMJ* 1995; 311: 1401-1405.

6. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. *Adv Data* 2000; 8: 1-27.

7. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in adults. Canberra: NHMRC, 2003.

8. Cole TJ, Bellizzi MC, Flegal KM, et al. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; 320: 1240-1243.

9. Maffeis C, Pietrobelli A, Grezzani A, et al. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res* 2001; 9: 179-187.

10. Goran MI, Gower BA. Relation between visceral fat and disease risk in children and adolescents. *Am J Clin Nutr* 1999; 70: S149-S156.

11. National Heart Foundation of Australia. Risk factor prevalence study No 1. 1980 Canberra: NHF, 1980.

12. Cameron AJ, Welborn TA, Zimmet PZ, et al. Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust* 2003; 178: 427-432.

13. Magarey AM, Daniels LA, Boulton TJ. Prevalence of overweight and obesity in Australian children and adolescents: reassessment of 1985 and 1995 data against new standard international definitions. *Med J Aust* 2001; 174: 561-564.

14. Stunkard AJ, Harris JR, Pedersen NL, et al. The body-mass index of twins who have been reared apart. *N Engl J Med* 1990; 322: 1483-1487.

15. Sorensen TI, Holst C, Stunkard AJ, et al. Correlations of body mass index of adult adoptees and their biological and adoptive relatives. *Int J Obes Relat Metab Disord* 1992; 16: 227-236.

16. Clement K, Boutin P, Froguel P. Genetics of obesity. *Am J Pharmacogenomics* 2002; 2: 177-187.

17. Robinson TN. Television viewing and childhood obesity. *Pediatr Clin North Am* 2001; 48: 1017-1025.

18. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001; 357: 505-508.

19. Jeffery RW. Minnesota studies on community-based approaches to weight loss and control. *Ann Intern Med* 1993; 119: 719-721.

20. Campbell K, Waters E, O'Meara S, et al. Interventions for preventing obesity in children. *Cochrane Database Syst Rev* 2002, CD001871.

21. Economos CD, Brownson RC, DeAngelis MA, et al. What lessons have been learned from other attempts to guide social change? *Nutrition Reviews* 2001; 59: S40-S56.

22. Ludwig DS, Pereira MA, Kroenke CH, et al. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. *JAMA* 1999; 282: 1539-1546.

23. Brand-Miller J, Foster-Powell K, Colagiuri S. The new glucose revolution. 2nd ed. Sydney: Hodder Headline Australia, 2003.

24. Harvey EL, Glenny A-M, Kirk SFL, Summerbell CD. Improving health professionals' management and the organisation of care for overweight and obese people (Cochrane Review). In: *The Cochrane Library*, Issue 1, 2004. Chichester, UK: John Wiley and Sons, Ltd.

25. Epstein LH, Valoski A, Wing RR, et al. Ten-year follow-up of behavioral, family-based treatment for obese children. *JAMA* 1990; 264: 2519-2523.

26. Epstein LH, Cluss PA. A behavioral medicine perspective on adherence to long-term medical regimens. *J Consult Clin Psychol* 1982; 50: 950-971.

27. Golan M, Weizman A, Apter A, et al. Parents as the exclusive agents of change in the treatment of childhood obesity. *Am J Clin Nutr* 1998; 67: 1130-1135.

28. Summerbell CD, Waters E, Edmunds L, et al. Interventions for treating obesity in children (protocol for a Cochrane review). *The Cochrane Library*, 2003. Oxford: Update Software.

29. Brownell KD, Kelman JH, Stunkard AJ. Treatment of obese children with and without their mothers: changes in weight and blood pressure. *Pediatrics* 1983; 71: 515-523.

30. Saelens BE, Sallis JF, Wilfley DE, et al. Behavioral weight control for overweight adolescents initiated in primary care. *Obes Res* 2002; 10: 22-32.

31. National Health and Medical Research Council. Clinical practice guidelines for the management of overweight and obesity in children. Canberra: NHMRC, 2003. Available at: www.obesityguidelines.gov.au (accessed Mar 2004).

32. Burry JN. Obesity and virtue. Is staying lean a matter of ethics? *Med J Aust* 1999; 171: 609-610.

33. Scheen AJ. Results of obesity treatment. *Ann Endocrinol (Paris)* 2002; 63: 163-170.

34. Geldszus R, Mayr B, Horn R, et al. Serum leptin and weight reduction in female obesity. *Eur J Endocrinol* 1996; 135: 659-662.

35. Montague CT, Farooqi IS, Whitehead JP, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. *Nature* 1997; 387: 903-908.

36. Haddock CK, Poston WS, Dill PL, et al. Pharmacotherapy for obesity: a quantitative analysis of four decades of published randomized clinical trials. *Int J Obes* 2002; 26: 262-273.

37. Torgerson JS, Hauptman J, Boldrin MN, Sjostrom L. XENical in the prevention of diabetes in obese subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. *Diabetes Care* 2004; 27: 155-161.

38. James WP, Astrup A, Finer N, et al. Effect of sibutramine on weight maintenance after weight loss: a randomised trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. *Lancet* 2000; 356: 2119-2125.

39. Dixon JB, O'Brien PE. Changes in comorbidities and improvements in quality of life after LAP-BAND placement. *Am J Surg* 2002; 184: 51S-54S.

40. Sjostrom L. Obesity, diabetes and other cardiovascular risk factors: lessons from SOS. *Int J Obes* 2000; 24: S7.

41. Dixon JB, O'Brien PE. Health outcomes of severely obese type 2 diabetic subjects 1 year after laparoscopic adjustable gastric banding. *Diabetes Care* 2002; 25: 358-363.

42. Allen JW, Coleman MG, Fielding GA. Lessons learned from laparoscopic gastric banding for morbid obesity. *Am J Surg* 2001; 182: 10-14.