

Obesity

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Obesity is the most common nutrition related disorder in Australia. It has been estimated that 34% of males and 24% of females are overweight (but not obese). A further 7% of both males and females between the ages of 25 and 64 years are classified as obese.¹ Obesity *per se* does not feature as a cause of premature deaths in Australia. However, many of the major causes such as ischaemic heart disease, stroke and even cancers have obesity as a significant part of their aetiology.²

Clinical features and diagnostic considerations

Aetiology

- The cause of obesity is multifactorial, the end result being increased body fatness (greater than 30% of total bodyweight in females and greater than 25% in males).
- It is due primarily to an energy imbalance, with energy input at some stage being in excess of energy requirements. Both genetic and environmental factors are involved, although the absolute contribution of each of these is unknown.
- Increases in food intake as well as reduction in physical activity are responsible for the development of obesity in many cases.
- The Prader-Willi syndrome is a rare genetic disorder characterised by obesity, hyperphagia, short stature, mental retardation, noninsulin dependent diabetes and hypogonadism. Deletion of the long arm of chromosome 15 has been found in 60% of patients.

Onset

Onset of obesity can occur at any age.

- Infancy, childhood and late adolescence are times during which excess intake may produce an energy imbalance.
- In early and later adult life, lack of physical exercise is more likely to lead to obesity.
- After pregnancy, obesity may result from a failure to return to prepartum energy requirements.

Grades of obesity

- Obesity can be divided into categories³ which reflect mortality related to excess fat, using the body mass index (BMI), i.e. weight in kilograms divided by the square of height in metres.

Grade 0 = BMI 20 to 25: minimal mortality related to bodyweight.

Grade I = BMI 25 to 29: overweight.

Grade II = BMI 30 to 40: obesity.

Grade III = BMI > 40: morbid obesity.

- The effect on mortality is evidenced by a J-shaped curve which relates mortality to BMI. There is a threefold increase in mortality with a BMI of > 40.
- Some factors such as increased muscle mass in men and in athletes or fluid retention need to be taken into account, however once a BMI of > 30 is reached, the diagnosis of excess body fat is fairly definite.
- Abdominal fatness is independently associated with an increased risk of noninsulin dependent diabetes mellitus, hypertension and hyperlipidaemia. It is defined as a waist to hip (W:H) ratio of > 0.85 in women and > 0.95 in men.

Diagnosis

- Measurement of bodyweight (kg) and height (m) for calculation of BMI. ('Healthy' range is between 20 and 25.)
- Single skinfold thickness (> 25 mm suggests increased body fat).
- Four skinfold thicknesses (sum of suprailiac, subscapular, triceps and biceps skinfolds) for calculation of percentage body fat (see table 1).
- Maximum circumference of the waist (cm) and hip (cm) for calculation of W:H ratio. ('Healthy' range is < 0.9.)
- More specialised techniques such as bioelectrical impedance, heavy water dilution, *in vivo* neutron activation, dual energy X-ray absorptiometry, magnetic resonance imaging (MRI) and CT scanning.

Table 1. Body fat and skinfolds (adapted from Durnin & Womersley⁵)

Sum of four skinfolds (mm)	% Body fat (30 to 39 years)	
	Males	Females
20	12.2	17.0
40	19.2	25.5
80	26.6	34.3
120	31.1	39.6
160	34.3	43.6
200	-	46.5

Note. This table shows the equivalent fat content as a percentage of bodyweight for a range of values for the sum of four skinfolds (biceps, triceps, subscapular and suprailiac) of males and females aged between 30 and 39 years. The 'healthy' range of percentage body fat is between 15 and 25% for men, and between 20 and 30% for women

Differential diagnosis

- Other medical conditions such as cardiac failure, the nephrotic syndrome and liver failure can lead to increased bodyweight. This increase in body water needs to be distinguished from increased body fat.
- Ascites, pregnancy and even severe constipation can lead to abdominal bloating and weight gain which may be initially thought to be fat.

Management principles

- Treatment revolves around four major interventions:
 - a) Reduction in energy intake.
 - b) Change in diet composition.
 - c) Increased physical activity.
 - d) Behavioural therapy.

The choice of intervention depends on the degree of obesity, the associated health problems and the health risk posed.

- Pharmacological agents (see below) are not used for first line therapy, although they may have some place in management.
- Surgery may be an option for the treatment of morbid obesity with associated complications.
- Management must be continued over a lengthy period of time with frequent contact with the treating physician. There is no quick cure.
- Differentiate between rapid weight loss (usually due to fluid shifts) and slow, progressive loss of body fatness.

Pharmacological agents available for treatment

- *Drugs that reduce hunger* (19e), e.g. phentermine, diethylpropion, mazindol: although structurally related to amphetamine, these agents have modified side chains and a much lower potential for abuse. Nevertheless, they retain many of the common side effects of the amphetamines such as sleeplessness, palpitations, dry mouth, nervousness and irritability.
- *Agents that enhance satiety* (19e) (fenfluramine and dexfenfluramine): unlike the above agents, *fenfluramine* has no CNS stimulant effect. Although side effects of drowsiness, diarrhoea or depression (on abrupt withdrawal) may occur, it has until recently been the anorectic drug of choice. By interacting with brain serotonin (as opposed to the catecholamine neurotransmitters affected by the amphetamines) it enhances satiety rather than suppressing hunger.

Dexfenfluramine, the dextroisomer of fenfluramine, has the potential for long-term use as it has far fewer side effects and no addictive properties. It has been shown to be of use particularly in subjects who are 'carbohydrate cravers' due to its effects on serotonin neurotransmitters.

Note. Drugs that either reduce appetite or increase satiety do not modify basic eating behaviour. Short-term weight loss has been documented but the long-term efficacy of such treatment is yet to be proven.

- *Drugs that increase energy expenditure: dexfenfluramine*, in addition to induction of satiety, is one of the few drugs shown to safely increase the thermic effect of feeding. It has no effect on resting metabolic rate and has not been shown to reduce lean body mass.
- *Bulking agents* (1c, 19e): methylcellulose and other nonenergy bulking agents have been used to reduce food intake, but there is little associated reduction in hunger or increase in satiety and they have not been demonstrated to be of long-term benefit.

Contraindicated drugs

- *Diuretics*: although these drugs may cause rapid weight loss due to fluid shifts, they do not alter body fatness and are of no benefit in the treatment of uncomplicated obesity.
- *Thyroid hormones*: the side effects of precipitation of ischaemic heart disease as well as loss of lean body mass make these drugs contraindicated unless hypothyroidism is diagnosed.

Surgical procedures

- *Gastric reduction surgery*: vertical banded gastroplasty which leaves the stomach with an average capacity of 17 mL (range 10 to 25 mL) is one method of reducing food intake. However, as with other therapies for obesity, it needs to be used in conjunction with behavioural therapy. Gastric stapling and gastric by-pass are other techniques considered to be of value in the morbid obese patient (BMI > 40 and, possibly, > 35) where the risk of complications associated with obesity outweigh the risk of surgical complication.
- *Jaw wiring* has been used in an attempt to reduce food intake and has had some limited success.

Optimum treatment**Grade I obesity (BMI 25 to 29)**

- An increase in exercise may be most beneficial, especially at times of growth spurts such as childhood and puberty when energy restriction may not be appropriate.
- It is important to make an estimate of percentage body fat as in this 'overweight' range, excess muscle or body water may elevate the BMI.
- If abdominal obesity is present, a reduction in both total fat and alcohol intake is recommended.

Grade II obesity (BMI 30 to 40)

- This requires more intensive therapy; a reduced energy intake (1,000 to 1,500 kcal/day) combined with a reduction in energy intake from fat to 30% with a subsequent increased proportion of total energy intake from unrefined carbohydrate is usually recommended.
- The reduced energy intake should be combined with exercise (often walking or swimming offer the best low impact options).

- Behavioural therapy, such as documenting seven day food intake, also plays a large part in therapy.
- In some instances, the addition of an appetite suppressant (see above) may give added benefit.

Grade III obesity (BMI > 40)

- If energy reduction or an increase in energy expenditure is not successful, the use of more drastic measures such as very low energy diets (300 to 500 kcal/day of a complete formula diet) or even surgery such as gastric stapling or gastric bypass procedures is justified.
- Once weight reduction has been achieved, it is necessary to undertake long-term, even life-long maintenance programs coupled with behaviour therapy, as these patients tend to return to their previous weight very quickly.
- Dexfenfluramine may be of use as a long-term satiety inducing agent.

Avoiding treatment errors

- Adequate assessment of body composition and degree of obesity is necessary.
- Treatment must be tailored to individual needs and depends on the associated complications of obesity.
- Drugs are only useful if given in conjunction with an energy modified diet and exercise program, and as the beginning of a long-term management program. They are of no benefit as a quick cure.

When to refer

Referral to a specialist is advisable:

- For patients with Grade II or III obesity (BMI > 30) who are resistant to simple weight control measures.
- For patients with associated medical problems such as angina or severe osteoarthritis who require rapid weight reduction.
- If there is a possibility of an endocrine cause of obesity on clinical assessment.

References

1. Bray GA. Obesity definition, diagnosis and disadvantage. *Medical Journal of Australia Special Supplement* 1985; 142: S2-8
2. Wahlqvist ML (ed). *Food and nutrition in Australia*, 3rd ed. Melbourne: Thomas Nelson, 1988
3. Garrow J. *Treat obesity seriously*. Edinburgh: Churchill Livingstone, 1981
4. Bray G. Complications of obesity. *Annals of Internal Medicine* 1985; 103 (No. 6, pt 2): 1052-1062
5. Durnin JVGA, Womersley J. Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition* 1974; 32: 77

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