

Anaesthesia and morbid obesity

Sharmeen Lotia MBBS MRCP FRCA
Mark C Bellamy MBBS MA FRCA

The prevalence of morbid obesity is increasing in the UK. Recent UK government statistics suggest that 20% of adults are obese and 1% morbidly obese. Anaesthesia and surgery may entail considerable risk for obese patients. Obesity is a multi-system disorder, particularly involving the respiratory and cardiovascular systems; therefore, a multidisciplinary approach is required. This article presents a broad overview of the pathophysiological and practical considerations for anaesthetizing such patients for major (non-bariatric) surgery.

A body mass index (BMI) of $>35 \text{ kg m}^{-2}$ with associated comorbidity, or $>40 \text{ kg m}^{-2}$ without significant comorbidity, is considered to be morbidly obese; $>55 \text{ kg m}^{-2}$ is considered super-morbidly obese (Table 1). However, morbidity and mortality increase sharply when BMI is $>30 \text{ kg m}^{-2}$, particularly in smokers, and risk is proportional to duration of obesity.¹ For a given BMI, men are at higher risk of cardiovascular complications than women. Obesity is described classically as conforming to an android or gynaecoid fat distribution ('apples and pears'). Both the actual BMI of a particular patient and the distribution of fat are important considerations. The gynaecoid fat distribution characteristically involves more fat distributed in peripheral sites (arms, legs, and buttocks). An android fat distribution involves more central fat (intra-abdominal fat, including involvement of the liver and omentum). Specific definitions have been proposed based on the waist-to-hip ratio. A value >0.8 in women or 1.0 in men is typical of the android distribution. Although the android distribution predominates in males and is associated with a higher risk of morbidity, either distribution can occur in each gender. Weight loss reduces risk for both groups.

Obese people have greater energy expenditure than lean individuals, and this is balanced by increased caloric intake. Basal metabolic rate is 'normal' in obese individuals when corrected for body surface area. However, with increasing weight, body surface area increases and hence

absolute basal metabolic rate values are higher than in lean individuals. Consequently, there is a greater absolute oxygen consumption and carbon dioxide production.

Causes of obesity

The causes of obesity are multifactorial and include genetic and environmental components that are as yet undefined. Resting energy expenditure is increased, but this is countered by dramatically increased calorie consumption. The regulation of appetite and satiety is a complex process under the control of multiple humoral and neurological mechanisms integrated and centrally processed in the hypothalamus. Hormones include leptin, adiponectin, insulin, ghrelin, and peptide YY₃₋₃₆. Leptin and adiponectin are produced by adipocytes, and their levels represent a total adipocyte mass. Leptin signals satiety and is important in reduction of eating and food-seeking behaviours. Obese patients have increased plasma leptin concentrations, but frequently exhibit leptin insensitivity. Moreover, vigorous dieting produces a reduction in adipocyte mass with an associated reduction in leptin levels, which itself may result in an increase in appetite and food-seeking behaviours. Adiponectin has a similar signalling role to leptin, but concentrations are not increased in obesity. Both leptin and adiponectin regulate long-term changes in appetite, whereas short-term effects are signalled by insulin acting on the hypothalamus.

Satiety is also signalled by a further group of peptides, including ghrelin which is released by the wall of the stomach. Eating stretches the wall of the stomach, suppresses ghrelin production, and reduces hunger. Ghrelin is also thought to be involved in the regulation of insulin sensitivity. When food subsequently passes into the small bowel, peptide YY₃₋₃₆ and related peptides are released, signalling satiety. The efferent limbs of the energy balance and appetite reflexes are mediated via the autonomic nervous system.

Key points

Morbid obesity in both adults and children is becoming more prevalent in the UK.

An understanding of the relevant pathophysiology and drug pharmacokinetics aids the anaesthetist in providing safe anaesthesia.

The patient must be nursed in an appropriate facility after operation. Level 2 or 3 care should be available, if required.

Regional anaesthesia may be an attractive option but presents technical challenges.

Sharmeen Lotia MBBS MRCP FRCA

Specialist Registrar in Anaesthesia
St James's University Hospital
Beckett Street
Leeds LS9 7TF
UK

Mark C Bellamy MBBS MA FRCA

Professor of Critical Care
St James's University Hospital
Beckett Street
Leeds LS9 7TF
UK
Tel: +44 113 206 6813
Fax: +44 113 206 4141
E-mail: m.c.bellamy@leeds.ac.uk
(for correspondence)

Table 1 Definitions of BMI, calculated as weight (kg) divided by height² (m)²

BMI (kg m ⁻²)	
<25	Normal
25–30	Overweight
>30	Obese
>35	Morbidly obese
>55	Super-morbidly obese

Comorbidity

Effect of fat distribution

Obesity is associated with hypertension, dyslipidaemia, ischaemic heart disease, diabetes mellitus, osteoarthritis, liver disease, and asthma. Obstructive sleep apnoea (OSA) is a common problem in the morbidly obese. Less well known is the obesity-hypoventilation syndrome. This and OSA are discreet but often coexisting entities (discussed later). BMI alone is a poor predictor of comorbidity, surgical, or anaesthetic difficulty. Fat distribution is often more useful; waist or collar circumference are more predictive of cardio-respiratory comorbidity than BMI. An android distribution makes intra-abdominal surgery more difficult and is associated with increased fat deposition around the neck and airway (hence greater difficulty in airway management and ventilation of the lungs). Furthermore, the android fat distribution is associated with greater risk of metabolic and cardiovascular complications.

The risk of cardiorespiratory and other comorbidity increases with the duration of obesity ('fat years'). However, the presence and severity of comorbidity may be masked by a sedentary lifestyle. The true significance of much obesity-related illness may only emerge during preoperative investigation or in the perioperative period.

Respiratory system

OSA is defined as apnoeic episodes secondary to pharyngeal collapse that occur during sleep; it may be obstructive, central, or mixed. The incidence of OSA increases with obesity and increasing age. More than 95% of cases go unrecognized. The diagnosis is confirmed by sleep studies. The characteristic features are:

- (i) frequent episodes of apnoea or hypopnoea during sleep, where five or more per hour or >30 per night are often quoted as clinically significant. An apnoeic episode is defined as 10 s or more of total cessation of airflow, despite continuous respiratory effort against a closed airway;
- (ii) snoring;
- (iii) day-time somnolence, associated with impaired concentration and morning headaches;
- (iv) pathophysiological changes: hypoxaemia (leading to secondary polycythaemia); hypercapnia; systemic vasoconstriction, or pulmonary vasoconstriction (leading to right ventricular failure).²

Patients with OSA frequently have increased adipose tissue in the pharyngeal wall, particularly between medial and lateral pterygoids. This results in increased pharyngeal wall compliance, with a tendency to airway collapse when exposed to negative pressure. There is also a change in airway geometry, so the axis at the open part of the airway is predominantly antero-posterior rather than lateral. Consequently, the increase in genioglossus tone seen during inspiration is far less effective at maintaining airway patency. In the long term, OSA affects control of breathing by desensitization of the respiratory centres, increasing reliance on hypoxic drive and eventually causing type 2 respiratory failure.

The obesity hypoventilation syndrome affects control of breathing, and includes obese patients who have a diurnal variation in ventilation and a $P_{a_{CO_2}} > 5.9$ kPa. Carbon dioxide sensitivity and respiratory drive are partly under the control of leptin. Relative leptin insensitivity in obesity is associated with a reduced ventilatory response to carbon dioxide. Depressant drugs, including many anaesthetic agents and analgesics, accentuate this. The obesity hypoventilation syndrome, although discreet from OSA, is often found in the same individuals.

The combined effect of these changes is a tendency to hypoxaemia at rest, further accentuated in the supine position and under anaesthesia. Combined with this is a tendency to rapid desaturation under conditions of apnoea. Lung compliance is decreased due to increased pulmonary blood volume. Reduced chest wall compliance results in part from the weight of adipose tissue around the thoracic cage, affecting the inspiratory threshold. Small airways collapse, cephalad displacement of abdominal contents, and increased thoracic blood volume contribute to reduced functional residual capacity (FRC). FRC declines steeply with increasing BMI and reaches values of around 1 litre or less in subjects whose BMI exceeds 40 kg m⁻². Similarly, there is a linear increase in alveolar–arterial (A–a) oxygen tension gradient with increasing BMI. Therefore, closing volume can encroach on FRC during normal tidal ventilation, leading to airway closure and ventilation/perfusion (V/Q) mismatching. Intrapulmonary shunt is increased.

Loss of compliance may be accentuated during laparoscopic surgery, particularly if the excessive pneumoperitoneal insufflation pressures are used. Although some authors have reported an improvement in respiratory mechanics when the reverse Trendelenburg position is used, this has not been a universal finding, and may be a further consequence of diaphragmatic splinting.³ Airway resistance is increased and correlates with BMI; it is further increased when transferring from a sitting to supine position. These factors contribute to an increased work of breathing in morbid obesity.

A modest preoperative $P_{(A-a)O_2}$ gradient and shunt fraction can deteriorate markedly on induction of anaesthesia requiring high $F_{I_{O_2}}$ to maintain an adequate arterial P_{O_2} , and PEEP may also be required. The combination of reduced chest wall and diaphragmatic tone during general anaesthesia, the increased incidence of atelectasis, and secretion retention resulting from reduced expiratory reserve volume and FRC render the morbidly obese patient at risk

of rapid desaturation during hypoventilation or apnoea. These problems persist into the postoperative period. Supplemental oxygen alone may be insufficient and may predispose to further atelectasis. A multimodal approach, involving posture, breathing exercises, physiotherapy, and in some cases continuous positive airways pressure (CPAP) or bilevel positive airways pressure, may be necessary in the immediate postoperative period. The duration of these interventions is assessed on an individual basis.

Cardiovascular system

Blood volume, cardiac output, ventricular workload, oxygen consumption, and CO₂ production are all increased. These may lead to systemic and pulmonary hypertension and later cor pulmonale and right ventricular failure. Absolute blood volume is increased, although this is low relative to body mass, reaching values as small as 45 ml kg⁻¹. Increased activity in the renin–angiotensin system and secondary polycythaemia play a role in this volume expansion. Blood is distributed mainly to tissue beds with increased fat deposition; cerebral and renal blood flows are relatively unchanged. Initially, there is an increase in left ventricular (LV) filling, and hence stroke volume.

Systemic hypertension is 10 times more prevalent in obesity. LV dilatation results in increased LV wall stress and hypertrophy, progressing to reduced ventricular compliance. Diastolic dysfunction is characterized by impaired ventricular filling, and ultimately by an elevated LV end-diastolic pressure. In combination with an increased blood volume, this leads to an increased risk of heart failure. ‘Obesity cardiomyopathy’ (systolic dysfunction) occurs when wall hypertrophy fails to keep pace with dilatation.¹ Furthermore, LV failure and pulmonary vasoconstriction result in pulmonary hypertension and dilatation of the right heart.

The increase in total blood volume, cardiac output, oxygen consumption, and arterial pressure is a result of the metabolic demands of the excess adipose tissue.

The obese patient is more at risk from arrhythmias because of: myocardial hypertrophy and hypoxaemia; hypokalaemia from diuretic therapy; coronary artery disease; increased circulating catecholamines; OSA (sinus tachycardia and bradycardia); and fatty infiltration of the conducting and pacing systems.⁴

Ischaemic heart disease is more prevalent in obese patients. Underlying causes include hypercholesterolaemia, hypertension, diabetes, lower HDL concentrations, and physical inactivity.

Other systems

Obesity is associated with macrovesicular fatty liver, which is reversible with weight loss but progresses to steato-hepatitis and cirrhosis if left untreated. There is a high incidence of gastro-oesophageal reflux and hiatus hernia. When coupled with increased gastric juice volumes, low gastric pH, and increased intra-abdominal pressure, the risk of gastric aspiration is high. There is an increased incidence of insulin resistance and diabetes. Hence, tight glycaemic

control in the perioperative period is both important and potentially difficult. Although preoperative weight loss dramatically reduces perioperative risk, even patients presenting for well-planned elective surgery generally fail to achieve significant weight reduction.

Preoperative assessment

The perioperative management of obese and morbidly obese patients presents significant organizational and practical issues. The Association of Anaesthetists has recently produced a helpful guideline which can be used as the basis of a rational approach to provision of safe anaesthetic services.⁵ However, individual patients require a ‘tailored’ plan. A detailed anaesthetic assessment must be performed. Many morbidly obese patients have limited mobility and may therefore appear relatively asymptomatic, despite having significant cardio-respiratory dysfunction. The drug history should note any amphetamine-based appetite suppressants as these contribute to increased perioperative cardiac risk. Symptoms and signs of cardiac failure and OSA should be sought actively. Many patients have been unable to lie flat for several years, and may routinely sleep sitting up in an armchair. An assessment of the ability to tolerate the supine position may reveal unexpected profound oxygen desaturation, airway obstruction, or respiratory embarrassment. Awake intubation in a sitting or semi-recumbent position is often better tolerated than supine induction of anaesthesia and asleep endotracheal intubation.

Mouth opening, Mallampati score, neck extension, and circumference (collar size >17.5 in) should be noted; in combination, these help to predict a potentially difficult airway. Airway obstruction may be clinically obvious at the bedside, as patients with severe sleep apnoea may even ‘snore’ while awake, for example, when drawing breath while speaking.

Investigations should be tailored to the individual patient, depending on comorbidity and the type and urgency of surgery. A full blood count, electrolytes, renal and liver function tests, and blood glucose form a basic set of investigations. Arterial blood gas analysis may be useful in those suspected of respiratory comorbidity (OSA, obesity hypoventilation syndrome, large collar size, and other pulmonary disease) as the patient’s habitual values provide a useful guide to weaning from ventilation and the potential need for extended perioperative respiratory support.

A preoperative ECG is essential (Table 2) to exclude factors such as significant rhythm disturbances and cor pulmonale, and as a guide to the need for more extensive cardiac investigation. Patients with evidence of right ventricular hypertrophy or cor

Table 2 Common ECG abnormalities associated with morbid obesity

Low voltage complexes
LV hypertrophy or strain
Prolonged QT/QTc
Inferolateral T wave abnormalities
Right axis deviation or RBBB
P pulmonale

pulmonale may benefit from a period of elective nocturnal non-invasive ventilation before elective surgery. This can be spectacularly effective in relieving right heart failure, day-time somnolence, and pulmonary hypertension.

Echocardiography may estimate systolic and diastolic function and chamber dimensions, although good images may be difficult to obtain by the transthoracic technique. Chest X-ray may be used to assess cardiothoracic ratio and evidence of cardiac failure. Pulmonary function tests may reveal a restrictive defect, but are not performed on all patients. Younger patients, those at the lower end of the BMI range, those with a good exercise tolerance, and those with a benign fat distribution need not be tested unless there is a specific indication. Unfortunately, most cardiorespiratory investigations are technically difficult owing to patient body habitus. Exercise ECG testing may be impracticable, but even a short walk along the ward or an attempt at climbing a flight of stairs can give useful functional information.

A significant residual gastric volume and acid pH is common. Antacids, proton-pump inhibitors, histamine H₂ receptor antagonists, and prokinetic agents are all likely to be of value in the perioperative period. Routine prophylaxis with ranitidine or a proton-pump inhibitor is advisable and can be administered orally at the time of premedication. Sodium citrate (0.3 M) may be given to patients with significant reflux symptoms. Obese patients are at increased risk of venous thromboembolism; appropriately sized compression stockings, low molecular weight heparin, and dynamic flow boots should be used from arrival in theatre until full postoperative mobilization.

Regional anaesthesia

Lung volumes in obese patients are reduced significantly in the postoperative period. There is a linear inverse correlation after premedication between vital capacity and BMI and a curvilinear inverse correlation between FRC and BMI. Although the subject of ongoing debate, many anaesthetists consider perioperative epidural anaesthesia (EDA) an important part of a multimodal approach to improving patient outcome and analgesia rather than relying solely on systemic opioid administration. EDA seems particularly attractive in obese patients undergoing major abdominal surgery, although the superiority of EDA in obese patients is not yet proven. In a recent study, there was less postoperative reduction in vital capacity and other spirometric values, and lung volumes recovered occurred more quickly in patients receiving EDA compared with those treated with opioids.⁶

Abdominal wall muscles play the major role in forced expiration. These may become less effective in the presence of a good epidural block.⁷ Moreover, there are significant practical difficulties in siting epidural catheters in the morbidly obese. These include the lack of palpable bony landmarks, the depth of the space (extra long needles may be required), and 'false' loss-of-resistance in fatty tissues. A multimodal analgesic approach is often required. This encompasses opioids, non-steroidal

anti-inflammatory drugs (NSAIDs), acetaminophen, and other local anaesthetic blocks (e.g. rectus sheath block and wound infiltration).

Pharmacokinetics of anaesthetic agents

Calculation of appropriate dosages may be difficult. Should the drug doses be calculated according to total body weight, BMI, lean body mass, or ideal body weight? The answer to this question is not simple.

The pharmacokinetics of most general anaesthetic drugs are affected by the mass of adipose tissue, producing a prolonged, less predictable effect. The volume of the central compartment is largely unchanged, but dosages of lipophilic and polar drugs need to be adjusted due to changes in volume of distribution (Vd). An increase in Vd prolongs the elimination half-life, despite increased clearance (Table 3). For example, there is a significant increase in the volume of distribution for a number of highly fat-soluble drugs, for example, benzodiazepines and barbiturates. For these types of drug, the ideal body weight should be used when calculating the dose. However, less fat-soluble drugs show little or no change in volume of distribution (e.g. some neuromuscular blocking agents). For these drugs, the lean body mass (or the ideal body weight plus 20%) should be used. An exception to this is succinylcholine, which should be dosed to total body weight. Propofol is highly lipid-soluble, but also has a very high clearance. Its volume of distribution at steady state and clearance are proportional to total body weight. Therefore, when using total i.v. anaesthesia, the infusion rate should be calculated on total body weight, not ideal body weight.⁸ Estimations of maximum recommended local anaesthetic doses for infiltration should be based upon ideal body weight. Local anaesthetic doses should be reduced by 25% for subarachnoid and epidural blocks as engorged epidural veins and fat impinge on the volume of the epidural space. Some drugs are cleared more rapidly in obese patients than in normal controls. This is in part due to higher cardiac output and splanchnic blood flow.

Table 3 Factors affecting drug pharmacokinetics in obesity⁴

<i>Volume of distribution</i>	
Decreased fraction of total body water	
Increased adipose tissue	
Increased lean body mass	
Altered tissue protein binding	
Increased blood volume and cardiac output	
Increased concentration free fatty acids, cholesterol, α_1 acid glycoprotein	
Organomegaly	
<i>Plasma protein binding</i>	
Adsorption of lipophilic drugs to lipoproteins so increased free drug available	
Plasma albumin unchanged	
Increased α_1 -acid glycoprotein	
<i>Drug clearance</i>	
Increased renal blood flow	
Increased GFR	
Increased tubular secretion	
Decreased hepatic blood flow in congestive cardiac failure	

Perioperative conduct of anaesthesia

Many anaesthetists choose to induce anaesthesia on the operating table. A theatre table with an appropriate maximum weight allowance must be used. There must be enough trained and experienced staff in theatre to assist with moving the patient quickly, should it become necessary during induction. Standard monitoring should include a correct-sized blood pressure cuff. Venous cannulation can sometimes be difficult and central venous cannulation may be necessary. Direct intra-arterial monitoring should be considered for situations where rapid haemodynamic changes are possible, surgery is prolonged, in patients with cardiorespiratory disease or if non-invasive arterial pressure monitoring is impractical.

Patient positioning is of paramount importance before induction, particularly head position. A 'sniffing the morning air' position may be difficult to achieve due to the large soft tissue mass of the neck and chest wall, and a wedge or blanket beneath the shoulders is of benefit ('ramped' technique). A degree of head-up tilt may slow the rapid desaturation that can occur on lying supine. Because of the reduced FRC, preoxygenation is less effective than in lean subjects. For this reason, in many bariatric surgery programmes, awake fiberoptic intubation is a routine. This should be considered in any patient who is hypoxaemic at rest or who has a history or clinical signs suggestive of airway problems.

However, for the majority of obese patients, a standard asleep intubation is both practicable and safe. Difficulties encountered in bag and mask ventilation can be overcome either by a four-handed technique or by the use of the mechanical ventilator with the mask. Laryngoscopy and intubation are often relatively straightforward with normal laryngeal anatomy. A polio handle, a long blade, or both are of value in overcoming the problems of the geometry of the head, neck, and chest wall.

Postoperative shivering, which increases oxygen consumption, prolongs the effects of some anaesthetic agents, and increases cardiovascular stress. Effective temperature maintenance is important; it also reduces postoperative wound infection. Forced warm air over-blankets are extremely effective, particularly when used in combination with fluid warmers.

Calf compression devices should be used and particular care given to pressure areas to prevent sores and nerve injury. If arm boards are used, over-abduction must be avoided as this risks brachial plexus injury. If pneumoperitoneum is used, remember that it causes a significant decrease in static respiratory system compliance and an increase in inspiratory resistance (though little increase in Δa -a gradient); ventilatory variables must be adjusted accordingly, and PEEP is desirable to maintain oxygenation during controlled ventilation. The use of short-acting anaesthetic agents such as remifentanyl, sevoflurane, or desflurane helps to aid rapid recovery from anaesthesia and minimize postoperative hypoventilation and hypoxaemia. Monitoring of neuromuscular block is essential, as incomplete reversal of neuromuscular blocking agents is poorly tolerated in morbid obesity and can have disastrous consequences.

Postoperative considerations

Where possible, those patients fit enough for extubation should be extubated wide-awake in the sitting position and transferred to an appropriate postoperative environment. Where the patient is nursed after operation depends on the nature and extent of the surgery and on the individual patient. In patients undergoing minor surgery whose only risk factor is obesity, there is little evidence that perioperative risk is increased and these patients may be nursed on the surgical wards. However, patients who have obesity-related comorbidities carry a dramatically greater risk of perioperative complications. Therefore, any obese patient undergoing major surgery, or those with a history of comorbidities, should be nursed in an appropriate level 2 or level 3 facility.

Many morbidly obese patients use a CPAP machine at home. Additionally, there are other individuals who suffer significant sleep apnoea or arterial desaturation who would also benefit from postoperative CPAP. There may be a considerable advantage in progressing directly from extubation onto a CPAP system. This may be needed for several nights after operation, as OSA occurs during deep sleep and rapid eye movement sleep, both of which may be suppressed in the immediate postoperative period and show rebound several nights later.

NSAIDs are extremely effective as part of a multimodal postoperative analgesic regimen, but they should be used judiciously as they may increase the incidence of postoperative renal dysfunction. NSAIDs are best omitted in obese patients with additional risk factors for postoperative renal dysfunction, for example, raised intra-abdominal pressure (particularly in those undergoing laparoscopic surgery) or diabetic nephropathy (sometimes subclinical). Acetaminophen, patient-controlled opioid analgesia, or regional anaesthesia are also useful. In morbid obesity, acetaminophen should be used in standard doses, as its volume of distribution is largely confined to the central compartment. However, as its clearance is increased in obesity, clinicians should give consideration to increasing the frequency of dosing where analgesia is problematic. I.M. injections should be avoided because of unpredictable absorption. Early mobilization is encouraged where possible, as it reduces postoperative atelectasis and the risk of venous thromboembolism. A bed with an overhead trapeze is useful. The catabolic response to surgery may necessitate the use of insulin after operation to maintain normoglycaemia. This reduces susceptibility to wound infections, and protects against myocardial infarction during periods of myocardial ischaemia.

Anaesthesia and the obese child

The prevalence of obesity in children is increasing in the developed world. Although much has been written about the anaesthetic management of obese adults, there is relatively little in the literature relating to anaesthesia in obese children. Generally, obese children experience fewer medical complications than obese adults, although derangements of respiratory physiology

are common across all age groups. Despite the relatively low prevalence of obesity-related comorbidity in children, they carry an increased likelihood of an anaesthetic critical incident, the risk rising with increasing BMI.⁹

References

1. Murphy PG. Obesity. In: Hemmings HC, Jr, Hopkins PM, eds. *Foundations of Anaesthesia, Basic and Clinical Sciences*. London: Mosby, 2000; 703–11
2. Adams JP, Murphy PG. Obesity in anaesthesia and intensive care. *Br J Anaesth* 2000; **85**: 91–108
3. Sprung J, Whalley DG, Falcone T, Wilks W, Navratil JE, Bourke DL. The effects of tidal volume and respiratory rate on oxygenation and respiratory mechanics during laparoscopy in morbidly obese patients. *Anesth Analg* 2003; **97**: 268–74
4. Shenkman Z, Shir Y, Brodsky JB. Perioperative management of the obese patient. *Br J Anaesth* 1993; **70**: 349–59
5. *Peri-operative Management of the Morbidly Obese Patient*. London: Association of Anaesthetists of Great Britain and Ireland, 2007. Available from <http://www.aagbi.org/publications/guidelines/docs/Obesity07.pdf>
6. von Ungern-Sternberg BS, Regli A, Reber A, Schneider MC. Effect of obesity and thoracic epidural analgesia on perioperative spirometry. *Br J Anaesth* 2005; **94**: 121–7
7. Sjögren S, Wright B. Respiratory changes during continuous epidural blockade. *Acta Anaesthesiol Scand* 1972; **16**: 27–46
8. Servin F, Farinotti R, Haberer JP, Desmonts JM. Propofol infusion for maintenance of anesthesia in morbidly obese patients receiving nitrous oxide. A clinical and pharmacokinetic study. *Anesthesiology* 1993; **78**: 657–65
9. Smith HL, Meldrum DJ, Brennan LJ. Childhood obesity: a challenge for the anaesthetist? *Paediatr Anaesth* 2002; **12**: 750–61

Please see multiple choice questions 1–5