Obesity is a global health problem whose prevalence is increasing. The World Health Organization (WHO) characterised obesity as a pandemic issue whose prevalence is higher in women than in men [1]. Consequently, the anaesthetist is increasingly confronted with the problems of anaesthetising obese patients, and even more so the obstetric anaesthetist. Adding to the spectrum of medical and surgical pathologies, obesity is also associated with an increased incidence of antenatal disorders. A thorough understanding of physiology, pathophysiology, associated conditions, their complications and the implications for analgesia and anaesthesia should place the anaesthetist in a better position to care for these patients.

**Definition**

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue to the extent that health may be impaired [2]. The underlying process is positive energy balance and weight gain. Obesity is often expressed with reference to body mass index (BMI).

\[
\text{Body mass index} = \frac{\text{weight (in kg)}}{\text{height}^2 \ (\text{in m})}
\]

Though BMI is a useful measure of prevalence and associated health risks of obesity, it does not account for the wide variation in the distribution of fat and may not correspond to the same degree of fitness in different individuals. WHO classifies obesity primarily based on the association between BMI and mortality (Table 1) [1].

**Epidemiology**

The prevalence of obesity is increasing at an alarming rate in both developed and developing countries. In pregnant women in the United States at the end of the last century, the prevalence ranged from 18.5% to 38.3% according to the cohort studied and the cut-off point used to define overweight [3]. A Brazilian study at a similar time reported the prevalence of obesity in pregnancy to be 5.5% [4]. The Health Survey of England published in

---

**Correspondence to:** K. Saravanakumar

E-mail: saravankumark@yahoo.com

Accepted: 18 September 2005
2002 gives us data about the prevalence of obesity in England. Females in the reproductive age group (16–44 years) have shown a dramatic increase in BMI (Fig. 1). The percentage of women with BMI above 30 increased from 12% in 1993 to 18.3% in 2002. Even more alarming is that the percentage of morbidly obese women has doubled in the last decade [5]. This illustrates the problem we are facing.

Physiological changes in obesity and pregnancy

Physiological changes associated with pregnancy are significant enough to have serious anaesthetic implications. When these are compounded by obesity, the anaesthetist may have to deal with a patient with seriously limited physiological reserve. Often, obesity has associated co-morbidities and pathological changes in different organ systems.

Respiratory system

All aspects of oxygenation and ventilation are affected in pregnancy. The physical, mechanical and hormonal changes influence the respiratory system. Table 2 summarises these changes in pregnancy, obesity and the combined effect of pregnancy with obesity [6–10]. Fortunately, not all changes associated with pregnancy in obese women are detrimental. In fact, there may be some improvement in respiratory function in an obese patient who becomes pregnant; especially the functional residual capacity, which usually improves [6]. Hormonal changes, through the relaxing effect of progesterone on smooth muscle, decreases airway resistance, thus reducing some of the negative effects of obesity on the respiratory system [8, 9]. Studies that have shown the respiratory effects of obesity in pregnancy to be minimal were carried out in the sitting position, whereas the supine position may alter the volume, flow and mechanical properties of breathing.

Obstructive sleep apnoea (OSA) is not uncommon in obese women who become pregnant. Pregnancy has some protective effects on sleep apnoea, despite the hyperaemia of nasal passages. Early in pregnancy, the increased sensitivity of the respiratory centre decreases apnoeic episodes and, in the later part of pregnancy, women tend to sleep on their side, thereby decreasing the likelihood of airway obstruction. Chronic hypoxia, hypercapnia and pulmonary hypertension increase

<p>| Table 1 WHO classification of obesity. |</p>
<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI; kg.m⁻²</th>
<th>Risk of co-morbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>18.5–24.9</td>
<td>Average</td>
</tr>
<tr>
<td>Overweight</td>
<td>≥25</td>
<td>Increased</td>
</tr>
<tr>
<td>Pre-obese</td>
<td>25–29.9</td>
<td>Increased</td>
</tr>
<tr>
<td>Obese class 1</td>
<td>30–34.9</td>
<td>Moderate</td>
</tr>
<tr>
<td>Obese class 2</td>
<td>35–39.9</td>
<td>Severe</td>
</tr>
<tr>
<td>Obese class 3</td>
<td>≥40</td>
<td>Very severe</td>
</tr>
</tbody>
</table>

WHO, World Health Organization; BMI, Body Mass Index.

<p>| Table 2 Changes in the respiratory system (changes represent a general trend rather than absolute values). |</p>
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pregnancy</th>
<th>Obesity</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progesterone level</td>
<td>↑</td>
<td>↔</td>
<td>↑</td>
</tr>
<tr>
<td>Sensitivity to CO₂</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>↑</td>
<td>↔ or ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Minute volume</td>
<td>↑</td>
<td>↓ or ↔</td>
<td>↑</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Inspiratory reserve volume</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>↓</td>
<td>↓↓</td>
<td>↓</td>
</tr>
<tr>
<td>Residual volume</td>
<td>↓</td>
<td>↓ or ↔</td>
<td>↑</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↓</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>FEV₁/VC</td>
<td>↔</td>
<td>↓ or ↔</td>
<td>↔</td>
</tr>
<tr>
<td>FEV₁/VC</td>
<td>↔</td>
<td>↔</td>
<td>↔</td>
</tr>
<tr>
<td>Total lung capacity</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Compliance</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>V/Q mismatch</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Resistance</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>DL₅₀</td>
<td>↑</td>
<td>or ↔</td>
<td>↑</td>
</tr>
<tr>
<td>PₐO₂</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>PₐCO₂</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

↑ = increase; ↓ = decrease; ↔ = No change (multiple arrows represent the degree of intensity).

CO₂ = carbon dioxide; FEV₁ = forced expiratory volume in 1 s; VC = vital capacity; V/Q = ratio of ventilation to perfusion; DL₅₀ = diffusion capacity of lung for carbon monoxide; PₐO₂ = Partial pressure of oxygen; PₐCO₂ = partial pressure of carbon dioxide.

Figure 1 Prevalence of obesity among females aged 16–44 in England as measured by BMI (source: Health survey for England 2002).
maternal morbidity and mortality significantly [11, 12]. Caesarean section, despite being lower abdominal surgery, potentially leads to reduced lung volume and capacities compared to non-obese patients [13].

**Cardiovascular system**

Pregnancy is associated with wide-ranging cardiovascular changes in line with increased oxygen demand. Obesity induced pathological changes have profound effects on cardiac, endothelial and vascular function. Unlike the respiratory system, where pregnancy offers some favourable effects in obese patients, the cardiovascular system is further stressed. The endocrinological, inflammatory and microvascular changes associated with obesity remain and are further augmented in pregnancy [14–16]. Table 3 summarises the changes in normal pregnancy, obese patients and obese pregnant women [14–20].

The extent of cardiovascular pathological changes secondary to obesity is dependent on the duration of obesity and its severity [18]. Any extra amount of fat deposited in the body demands its share of cardiac output. Every 100 g of fat deposited increases the cardiac output by 30–50 ml.min⁻¹. This is also accompanied by an increase in blood volume. Volume load initially brings about left ventricular hypertrophy and then subsequently the myocardium starts to dilate against the increased pressure overload. The pressure overload is secondary to increased sympathetic activity due to the potentiating effects of hormones such as leptin, insulin and some inflammatory mediators. The heart rate increases in line with elevated cardiac output, thereby decreasing the diastolic interval and thus the time for myocardial perfusion. Impaired myocardial diastolic relaxation leads to diastolic dysfunction. If fat deposition occurs in myocardial tissue, then conduction and contractility can be seriously affected [18, 19]. Hence, it is not uncommon to see systolic, diastolic or both systolic and diastolic dysfunction of the left ventricle. Right ventricular failure can be present in patients with pulmonary hypertension and OSA. Congestive heart failure is a consequence in the presence of any additional stress [9, 10, 16, 18, 19].

Insulin resistance and dyslipidaemias affect the vascular tree and increased inflammatory mediators such as C-reactive protein, IL-6, and TNF-α affect endothelial function. This endothelial dysfunction in pregnant women may predispose to the development of pregnancy-induced hypertension [15, 16, 19, 21]. The well-known effect of an enlarged uterus compressing abdominal major vessels and causing supine hypotension syndrome (SHS) can also be seen in obese patients. This can be greatly exacerbated in obese parturients where a large panniculus adds to the uterine compression. The problem may extend postoperatively if the panniculus is large enough to cause compression of the vessels by itself. Tsuuda et al. reported two cases of sudden death on assuming the supine position in morbidly obese patients, which they attributed to circulatory changes brought about by changes in their position [22]. Drenick & Fisler also reported cases of postoperative cardiac arrest in obese surgical patients. There was no pathology found at autopsy to explain the cardiac arrest [23].

### Gastrointestinal changes

Both anatomical and hormonal changes increase the incidence and severity of gastric reflex in pregnant women. Hiatus hernia is more common in obese patients and obesity itself increases the risk of aspiration under anaesthesia. When pregnancy is associated with obesity, the likelihood of regurgitation and aspiration substantially increases. Roberts and Shirley studied obese and non-obese pregnant women in labour; the gastric volume in obese parturients was five times greater than in controls [24].

### Endocrine and reproduction

Because obese girls reach critical mass earlier than normal weight girls, they tend to reach menarche earlier. An ob protein called leptin is implicated in the mechanism. Obesity-induced changes in the reproductive system mean there is a reduced likelihood that these women will become pregnant [25]. Despite the potential

---

**Table 3** Changes in the cardiovascular system. Table shows the general trend. Extent of variability in each parameter depends on duration, degree of obesity and associated co-morbid states.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pregnancy</th>
<th>Obesity</th>
<th>Combined</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>↑</td>
<td>↑↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>↑↑↑</td>
<td>↑↑□</td>
<td>↑↑</td>
</tr>
<tr>
<td>Cardiac Output</td>
<td>↑ or ↔</td>
<td>↑↓</td>
<td>↔ or ↓</td>
</tr>
<tr>
<td>Cardiac Index</td>
<td>↑</td>
<td>↑□</td>
<td>↑□</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>□□□</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Blood volume</td>
<td>□□□□</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Systemic vascular resistance</td>
<td>□□□□</td>
<td>□□□□□</td>
<td>□□□□□</td>
</tr>
<tr>
<td>Mean arterial pressure</td>
<td>↑</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Supine hypotension</td>
<td>Present</td>
<td>Hypertrophy</td>
<td>Hypertrophy and dilation</td>
</tr>
<tr>
<td>Left ventricular</td>
<td>Hypertrophy</td>
<td>Hypertrophy and dilation</td>
<td>Hypertrophy and dilation</td>
</tr>
<tr>
<td>morphology</td>
<td>↑</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Sympathetic activity</td>
<td>↑</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Systolic function</td>
<td>↔</td>
<td>↔ or ↓</td>
<td>↔ or ↓</td>
</tr>
<tr>
<td>Diastolic function</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>↔</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Pulmonary wedge pressure</td>
<td>↔</td>
<td>↑□□□□</td>
<td>↑□□□□</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>Absent</td>
<td>May be present</td>
<td>May be present</td>
</tr>
<tr>
<td>Pre-eclampsia</td>
<td>↔</td>
<td>n/a</td>
<td>↑□□□□</td>
</tr>
</tbody>
</table>

↑ = increase, ↓ = decrease, ↔ = no change (multiple arrows represent the degree of intensity). n/a = not applicable.
anatomical difficulties of intercourse, pregnancy is by no means uncommon. Onset of menopause is earlier by an average of 4 years [25]. Obese parturients exhibit insulin resistance [21] and are more prone to develop gestational diabetes, which may persist even after pregnancy [26].

**Perinatal outcomes of obesity**

**Maternal morbidity**

The major maternal complications reported to be associated with obesity during pregnancy include hypertensive disease (chronic hypertension and pre-eclampsia), diabetes mellitus (pregestational and gestational), respiratory disorders (asthma and sleep apnoea), thromboembolic disease, Caesarean section and infections (primarily urinary tract infections, wound infections and endometritis) [27]. Association between obesity and hypertensive disorders of pregnancy, diabetes, delivery by Caesarean section (both primary and repeat) are well documented [28–30]. Although obese parturients are at significant risk of pre-eclampsia, they do not seem to be at increased risk of HELLP (Haemolysis, Elevated Liver enzymes, Low Platelets) syndrome [31]. As discussed before, dyslipidaemias, lipid peroxidation, endothelial damage, platelet aggregation and inflammatory mediators make direct and indirect contributions towards the pathophysiology of the hypertensive disorders of pregnancy [21]. Complications during labour such as intrapartum fetal distress, meconium aspiration, failure to progress, abnormal presentation, shoulder dystocia and an increased rate of instrumental delivery are more common [28, 32, 33]. Moreover, the success of vaginal birth after Caesarean section has been shown to decrease stepwise with increasing maternal BMI [32, 34]. Postoperative endometritis and wound infection are significantly higher in obese women [33–35]. Obesity is an intrinsic risk factor for both increased operative blood loss and postpartum haemorrhage [36]. Carter *et al.* have demonstrated a significant association between BMI, eating attitudes and symptoms of anxiety and depression in the postpartum period that are not present during pregnancy [37]. Overall, the literature suggests that obese pregnant women have a 14–25% incidence of pre-eclampsia, a 6–14% incidence of gestational diabetes and a 30–47% likelihood of Caesarean delivery [27, 28].

**Fetal morbidity**

Maternal obesity is associated with fetal macrosomia, increased risk of birth defects and fetal deaths [38–40]. Various studies have revealed an increased risk of a variety of conditions such as neural tube defects, especially spina bifida, omphalocele and heart defects, in particular ventricular outflow tract defects or multiple anomalies [39]. Moreover, increasing maternal size represents a major risk factor for failure of ultrasound to diagnose fetal anomalies [41]. Chauhan *et al.* reported a higher incidence of umbilical arterial acidosis (pH < 7.10) among obese women regardless of whether they had a trial of labour or repeat Caesarean delivery [29]. The higher incidence of fetal compromise in morbidly obese women could be the result of an inability to assess the fetus adequately because of size, underlying medical complications such as chronic hypertension, pregestational diabetes or that traditional intrauterine resuscitative efforts are inadequate in overweight patients. Obese women face difficulties with both lactogenesis and initiation of breast-feeding because of a decreased prolactin response to suckling [42, 43]. Breast-feeding is protective of childhood obesity [44]. The worries about propagation of a vicious circle are being confirmed by the longitudinal study of British cohort from 1958 that concludes maternal weight rather than birth weight as an important risk factor for childhood obesity and adult BMI [45].

**Weight gain and changes**

Pregnancy is normally associated with weight gain that consists of the growing fetus and enlarging maternal-fluid and soft-tissue compartments including fat. On average, a 3.5 kg weight gain is mostly due to maternal storage of fat during pregnancy [46]. Ledermann *et al.* showed that women who are overweight or obese and gain weight as recommended during pregnancy appear to have a small or negligible increase in total body fat [17]. The main components of weight gain in these heavier women are fluid and non-fat soft tissues. Community based studies have also reported that maternal obesity is not associated with increased weight gain after pregnancy, though they are generally at risk of gaining more weight [47].

**Anaesthetic considerations**

Obesity has been identified as a significant risk factor for anaesthesia related maternal mortality [48, 49]. The increased incidence of operative procedures, both elective and emergency, and the concurrent medical and antenatal problems may contribute to the risk. Postoperative complications such as wound infection, deep vein thrombosis, atelectasis and chest infection are more prevalent [49–51]. In addition to the associated medical problems, the anaesthetist is challenged by these patients with technical difficulties of airway management and insertion of regional blocks. No anaesthetic technique is without special hazards in grossly obese patients.

**Airway**

The incidence of failed tracheal intubation is approximately 1 in 280 in the obstetric population compared to 1 in 2230 in the general surgical population [52–54]. This
contrasts with an incidence of difficult intubation in an obese population as high as 15.5% [55]. In his institutional experience, Dewan [7] reports the incidence as high as 33% in morbidly obese parturients. Interestingly, a 6-year review of failed intubations in obstetric patients in a UK region reported 36 cases of failed intubation and the average BMI of these women was found to be 33 [53]. So it is evident that difficult or failed tracheal intubation in obese parturients is very high and optimal assessment and management of the airway cannot be overemphasised in this population.

Though there are no bony differences between the pregnant and non-pregnant population, obese and non-obese patients, fat deposition in obese and soft tissue changes during pregnancy do influence the airway. Operational factors such as poor head positioning, cricoid pressure [56] and anxiety contribute to the difficulty on occasion. In addition, pregnancy induced hypertension, upper respiratory tract infection, stridor and voice changes may suggest the presence of airway oedema. Weight gain in excess of 15 kg during pregnancy has been shown to be associated with an increase in suboptimal laryngoscopic views [57].

Although not totally reliable, assessment of oropharyngeal structures using the Mallampatti classification has been shown to strongly correlate with the prediction of difficult intubation in obstetric anaesthesia [58, 59]. Other features shown to be significant include short neck, receding mandible and protruding maxillary incisors [58]. The combination of two tests (Mallampatti and thyromental distance), albeit in a small study of 80 obstetric patients receiving general anaesthesia, has been shown to be 100% sensitive with 70% positive predictor value [59]. These tests can be done in less than 1 min; hence they are also useful in an emergency scenario. A previous uneventful general anaesthetic for either obstetric or non-obstetric reasons may not be helpful because of weight gain. However, previous anaesthetic charts inform of laryngoscopic view. In cases of documented difficult intubation, further airway imaging (X-ray and computerised tomography) is questionable as the combined clinical and radiological measurements only improve predictability of a difficult airway by 0.04% compared to clinical assessments alone [60]. Anaesthesia for both emergency and elective scenarios should be planned in advance. It is appropriate to involve patients in the decision-making process for safe delivery of the fetus.

**Respiratory system**
The likelihood of OSA has been alluded to, but it is often under-diagnosed in women of childbearing age [11]. Normal women in late pregnancy have difficulty with sleep maintenance and spend less time in the supine position. It is possible that, inasmuch as complaints of difficulty in sleeping and daytime fatigue are common, women suffering from OSA are not identified. Careful history taking may help diagnose OSA. Prompt diagnosis by polysomnography and treatment with continuous positive airway pressure may be beneficial. Pulmonary hypertension and right heart failure need to be excluded in parturients with OSA [12, 61]. Measurement of oxygen saturation by pulse oximetry, both in sitting and supine positions, may provide evidence of airway closure during normal tidal volume ventilation, thereby identifying candidates for postoperative oxygen administration. Pre-operative arterial blood gas examination provides information regarding the current status of ventilation and oxygenation. Detailed pulmonary investigations including chest X-ray should be reserved for those with more severe respiratory disease. Pre-operative antibiotics and chest physiotherapy are ideally reserved for patients with concurrent chest infection.

**Cardiovascular system**
Cardiovascular co-morbidities such as hypertension, ischaemic heart disease and heart failure dominate the clinical picture in the obese population and these can co-exist in obese parturients. Nearly 40% of the obese population experience angina without demonstrable coronary artery disease [62]. Hence, routine electrocardiograph recording may be useful. Cardiologists should be involved early in the care of symptomatic morbidly obese parturients to investigate and optimise the disease status wherever appropriate.

**Gastrointestinal and endocrine systems**
Gastro-oesophageal reflux and diabetes mellitus are the most commonly seen disorders [28]. Any previous laboratory investigations such as fasting blood glucose concentration and liver function tests should be noted. If there is any abnormality of liver function, HELLP syndrome should be ruled out.

Though aggressive prophylaxis against acid aspiration is advocated for all obese mothers undergoing Caesarean section [24, 63], there is a lack of conclusive evidence for starvation policies and prophylaxis during labour. Current evidence suggests avoiding solids and semisolids once a woman is in active labour or requests analgesia [64]. Sodium citrate and ranitidine remain the most commonly used drugs for acid aspiration prophylaxis in the UK [65].

**Practical considerations**
Obese parturients share a battery of technical difficulties along with their non-pregnant counterparts. Blood pressure measurement will require an appropriate sized cuff; otherwise both systolic and diastolic readings will be...
overestimated. There is a strong argument in favour of invasive blood pressure monitoring peri-operatively if there are difficulties in getting the appropriate size cuff and positioning of the arm, as commonly used regional blocks demand frequent monitoring of blood pressure [66]. Venous access can be difficult and extravasation may not be immediately apparent. Patients should be warned about the difficulties and the possible need for central venous access and arterial cannulation in the peri-operative period. When regional anaesthesia is contemplated, examination of needle insertion sites and spinal bony landmarks might predict difficulty [67].

Many of the other vital accessories are related to the physical size of parturients and need to be considered well in advance of the delivery. The weight bearing capacity of delivery beds and operating tables should be adequate to accommodate super-morbidly obese parturients. Suitable equipment (trapezes, lifters and hoover mats) and sufficient numbers of personnel for safe transfer of women within the hospital must be available. Sufficiently large blood pressure cuffs should be made available wherever blood pressure readings might be taken, including clinics, wards and the delivery suite. Mechanical devices for thrombo-prophylaxis such as stockings of appropriate size and sequential compression devices are other requisites for these patients.

**Anaesthetic management**

**Analgesia for labour**

Analgesia during labour is of significant importance for the positive experience of childbirth for many mothers. Increased prepregnancy weight is associated with an increased incidence of fetal macrosomia and labour abnormalities such as shoulder dystocia. Each of these is a known risk factor for more painful contractions and complicated labour. Melzack *et al.* reported a positive correlation of BMI with the severity of labour pain [68]; however, this has been questioned by Ranta *et al.* [69]. Although there are various modalities of pain relief, analgesia using neuroaxial blockade has been shown to be the most effective [70]. The anticipated technical difficulties should not preclude the use of epidural analgesia in obese parturients. Effective pain relief during labour can improve maternal respiratory function and attenuate sympathetically mediated cardiovascular responses [71, 72]. Available evidence shows that the rate of Caesarean delivery does not increase with epidural analgesia during labour [70], though obesity increases the need for Caesarean section. Hence, placing a functional epidural catheter is advantageous should any operative intervention be required. In addition, epidural analgesia can be extended into the postoperative period where adequate pain relief can optimise care.

The challenges for the anaesthetist should not be underestimated. Technical difficulties include positioning the patient correctly, identification of the midline, identification of the epidural space, and dislodgement of catheters [51, 66, 73]. The initial failure rate for epidural catheter placement can be very high (42%) [51] and multiple attempts of catheter placement are common. Jordan *et al.* noted 74.4% of massively obese parturients needed more than a single attempt and 14% needed more than three attempts for successful epidural placement [50]. The knee–chest position required for siting epidurals in the lateral position is difficult to obtain in the obese. One study found that cardiac output decreased more in the lateral decubitus position with maximal lumbar flexion compared with the sitting position [74]. Moreover, in the lateral position, gravity can drag down the pad of fat obscuring the midline. Another study found the depth of the epidural space from skin to be greater in patients where the epidural was inserted in the lateral decubitus position [75]. Overall, the sitting position is preferable. Various techniques have been reported to identify the midline in difficult scenarios. They include using the prominence of the seventh cervical vertebrae and the gluteal cleft to identify the midline and using a 26-G 8.5 cm spinal needle to probe subcutaneous tissue and delineate the position of a posterior process above and below a lumbar interspace [76]. Where it is difficult to identify the space, parturients can assist verbally in directing needle to the midline [66] and tilting the table towards the anaesthetist can encourage opening of interspinous spaces by forcing the parturient to bend forwards. The difficulty in location of the midline increases the likelihood of lateral projection of the needle apparently increasing the depth of the space and mal-positioning of the catheter [77]. The paramedian approach to identify the epidural space is sometimes popular in difficult cases. With the back pad of fat and pregnancy induced softening of the tissues, there is a higher incidence of false positives in identifying the epidural space. It has been argued that the loss of resistance technique is more reliable than the techniques that are dependent on subatmospheric pressure in the obese. The depth of epidural space at the L3–4 interface positively correlates with BMI [75, 78].

Some of the difficulties can be circumvented by using ultrasound to identify the epidural space and calculate its depth [79, 80]. Ultrasound studies have confirmed the significant changes in spinal anatomy in pregnancy and found the skin–epidural distance to be greater in parturients. They have also revealed that the safety zone between transfixation of the ligamentum flavum and
inadvertent dural puncture is smaller [81]. Although the epidural space may be deeper in overweight people, the majority of studies report that only a few have an epidural space deeper than 8 cm [75, 78]. Hence it seems appropriate to use a standard needle to identify the epidural space on the first attempt. A successfully placed catheter can easily be dislodged by the drag of the back pad of fat when parturients move around in bed. Wasson postulates that a 6 cm lateral movement of skin site relative to the spine would drag 2 cm of catheter out of the epidural space [82]. Moreover, the catheter is subjected to a drag when the flexed back is relaxed immediately after a successful placement. These should be taken into consideration when deciding the length of catheter to be left in the space for a successful working epidural. The other common reasons for a successfully placed catheter to fail include inadequate dose of local anaesthetic, poor drug spread, misplacement of the catheter, septae within the epidural space and fetal malpositioning leading to more stimulation. With increased operative intervention in obese parturients, it is prudent to make sure that the epidural is working well and, if not, to replace the catheter at the earliest possible opportunity. Hodgkinson et al. have shown that BMI and weight are the major determinants of cephalad spread of epidural anaesthesia and that in obese patients the sitting position reduced cephalad spread [83, 84]. However, Milligan et al. showed no difference in the spread of epidural analgesia between obese and non-obese patients requesting labour analgesia, either in the sitting or in the lateral position [85].

Entonox can be a useful adjunct. Other forms of inhalational analgesia involving isoflurane and desflurane provide better analgesia when compared to nitrous oxide alone but cause more sedation and amnesia. Whereas intramuscular opioid injections may be unreliable, patient controlled intravenous analgesia has been used successfully in obese patients. However, opioids in labour have been associated with maternal and fetal side-effects [86]. All these methods potentially cause maternal drowsiness and lead to airway obstruction, thus inherently carrying a risk to the obese parturients.

Anæsthesia for Caesarean section

Obesity and caesarean section have been identified as independent risk factors for maternal morbidity and mortality [28]. In Why Mothers Die 2000–02, 35% of all the women who died were obese, 50% more than in the general population [49]. Analysis of direct maternal deaths due to anaesthesia reported in the confidential enquiries from 1979 to 2002 reveals the dominance of deaths under general compared to regional anaesthesia (Table 4 [49, 87–93]). Hawkins, in her analysis of maternal deaths in

<table>
<thead>
<tr>
<th>Year</th>
<th>Total (n)</th>
<th>GA (n)</th>
<th>RA (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000–02</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>1997–99</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>1994–96</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>1991–93</td>
<td>8</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>1988–90</td>
<td>4</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>1985–87</td>
<td>8</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>1982–84</td>
<td>18</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>1979–81</td>
<td>22</td>
<td>22</td>
<td>0</td>
</tr>
</tbody>
</table>

GA, general anaesthesia; RA, regional anaesthesia.

Figure 2 Anaesthesia-related maternal deaths by types of anaesthesia, United States 1979–90 [94]. Reprinted with permission from Anesthesiology.

Table 4 Direct maternal deaths due to anaesthesia by types of anaesthesia in United Kingdom 1979–2002. Derived from CEMD reports. Since 1979, maternal deaths are reported as direct and indirect.

the United States, also reported that the absolute number of deaths due to complications of general anaesthesia, although small, is not decreasing over time [94] (Fig. 2). Regional anaesthesia is a safer option than general anaesthesia for delivery of the fetus [49, 94]. The principles in the anaesthetic management of these patients include

• regional anaesthesia unless contraindicated;
• care provided by experienced medical personnel (both anaesthetist and obstetrician);
• anticipation of problems and effective preparation in terms of equipment, monitoring and personnel;
• general anaesthesia, if required should be delivered with tracheal intubation and controlled ventilation;
• postoperative care that includes close monitoring, early mobilisation, and physiotherapy; a high dependency setting may achieve this most appropriately;
• judicious use of neuraxial, oral and intravenous opioids for postoperative pain relief.
It is not surprising that obese parturients undergoing Caesarean section are at increased risk of bleeding [36, 50] and require longer operating time [50, 51]. Panniculus retraction is another issue in morbidly obese parturients. Cephalad retraction of the panniculus can cause severe cardiovascular compromise. There has been at least one report of fetal death attributed to severe hypotension following panniculus retraction [95]. The dense regional block up to the fourth thoracic dermatome required for Caesarean section can compound cardiovascular compromise. Paralysis of intercostal muscles along with the supine position and cephalad retraction of the panniculus has the potential to increase the work of breathing substantially and provoke respiratory failure in this susceptible population. Suspending the abdominal panniculus vertically may alleviate some of these problems [95].

Regional anaesthesia

Single shot spinal anaesthesia remains the most common type of anaesthesia employed for delivery of the fetus by Caesarean section. The advantage of using subarachnoid block includes a dense reliable block of rapid onset. However, relevant issues include technical difficulties, potential for high spinal blockade, profound dense thoracic motor blockade leading to cardiorespiratory compromise and inability to prolong the blockade. Technical difficulties are the same as those for locating the epidural space. However, it is often easier to identify the epidural space with a large gauge stiff epidural needle than with a smaller gauge flexible spinal needle. Many anaesthetists use an epidural needle as an introducer for a spinal needle in difficult cases. It is widely believed that local anaesthetic requirements are less in pregnant patients, more so in obese patients. Proposed mechanisms for the enhanced neural blockade in pregnancy include hormone-related changes in the action of spinal cord neurotransmitters, potentiation of the analgesic effect of endogenous analgesic systems, increased permeability of the neural sheath or other pharmacokinetic/dynamic differences [96]. Both pregnancy and obesity increase intra-abdominal pressure and cause compression of the inferior vena cava, which leads to engorgement of the epidural venous plexus and increased epidural space pressure. This in turn reduces the volume of cerebrospinal fluid in the subarachnoid space. Magnetic resonance imaging has confirmed the decreased cerebrospinal fluid volume in obese parturients [97]. Greene suggests that obesity per se does not decrease local anaesthetic requirements, rather the larger buttocks of obese patients place the vertebral column in the Trendelenburg position, exaggerating the spread of spinal anaesthesia [98]. There is little evidence in the literature to suggest an exaggerated spread in obese patients for a given amount of local anaesthetic agent [99–102]. Whatever the mechanism, the consequences of extensive blockade in the presence of a potentially difficult airway dictate the amount of local anaesthetic agent given intrathecally. Duration of surgery may extend beyond the duration of single-shot spinal anaesthesia and in such cases intra-operative induction of general anaesthesia is undesirable and potentially hazardous. Although continuous spinal anaesthesia represents an alternative [103], the merit of using it routinely for Caesarean section is yet to be proved. Hence, while choosing single-shot spinal anaesthesia, the consequences of extensive blockade and prolonged surgery must be borne in mind.

Compared with spinal anaesthesia, epidural anaesthesia offers several advantages, including an easily titratable local anaesthetic dose and level of anaesthesia, ability to extend the block for prolonged surgery, slower and more easily controllable haemodynamic changes, a decreased potential for excess motor blockade and its utilisation for postoperative analgesia. Hodgkinson and Hussain demonstrated that height of blockade for a given volume of local anaesthetic agent is proportional to BMI and weight but not height [83]. The decreased volume of the epidural space, for reasons discussed above, may contribute to excessive spread of local anaesthetic agents in obese parturients. However, incremental injection of local anaesthetic doses reduces the effect of obesity on the spread of epidural anaesthesia. Extending labour epidural analgesia for Caesarean section requires additional local anaesthetic agent at higher concentrations. An optimum dose for this remains undetermined.

A combined spinal-epidural technique represents an attractive alternative [104], combining the advantages of rapid onset and dense block with the ability to prolong the block and use postoperatively. Furthermore, use of less local anaesthetic agent intrathecally may circumvent the problems of sudden onset of hypotension. However, the technical challenge of correctly placing an epidural catheter remains.

General anaesthesia

In England, 5–19% of Caesarean sections are done under general anaesthesia in the majority of the units [105]. The anatomical and physiological changes caused by both obesity and pregnancy are less favourable to anaesthetists, resulting in an increased incidence of difficult intubation and rapid desaturation during the apnoeic phase. The potential for an unanticipated difficult airway, difficult mask ventilation and rapid desaturation emphasises the need for an additional pair of experienced hands when administering general anaesthesia. In known cases of difficulty, awake intubation by direct laryngoscopy, fibre optic bronchoscope or Bullard laryngoscope [106] is
an alternative method of securing the airway. Awake intubation poses its own problems. The nasal route is not recommended because of the characteristic engorgement of nasal mucosa during pregnancy. Hypertension and catecholamine release during the procedure could adversely affect uterine blood flow. Moreover, it is difficult to perform in urgent scenarios such as maternal haemorrhage or fetal distress [7]. In any of these circumstances, the mother’s life should not be endangered to save a compromised fetus.

In an otherwise normal airway, administration of general anaesthesia begins with effective denitrogenation of the lungs, as obese parturients desaturate rapidly compared to normal patients. Denitrogenation can be performed by either 3 min of tidal breathing with 100% oxygen or by four maximal breaths with 100% oxygen. There is little evidence to support one technique over the other [107, 108]. Hence, it is reasonable that urgency of Caesarean section dictates the technique of denitrogenation. The choice of intravenous induction agent is relatively unimportant if there are no co-existing medical problems. Dewan suggests that at least 4 mg.kg
-1 of thiopental (up to a maximum dose of 500 mg) should be used if chosen, to avoid the risk of maternal awareness, hypertension and decreased uterine blood flow during light anaesthesia [7]. Administration of a larger dose may be associated with delayed arousal in the event of failed intubation. Succinylcholine remains the muscle relaxant of choice for intubation. Bentley et al. observed increased pseudo-cholinesterase activity in obese non-pregnant patients. They recommended that anaesthetists should administer succinylcholine on the basis of total rather than lean body weight in adult patients [109]. However, pregnancy reduces pseudo-cholinesterase activity. Hence the dose of succinylcholine 1.0–1.5 mg.kg
-1 up to a maximum of 200 mg is reasonable [7]. Tracheal intubation should be confirmed by the repetitive and characteristic waveform of capnography in addition to auscultation. Endobronchial intubation should also be promptly recognised and corrected to avoid intra- and postoperative pulmonary complications. In the event of failure to intubate the trachea after rapid sequence induction, it is imperative to institute a failed intubation drill without delay. Repeated attempts and a second dose of succinylcholine are seldom beneficial and often detrimental. The primary objective in the management of failed intubation is to ensure adequate maternal oxygenation despite the concerns of fetal well-being or risk of regurgitation.

In morbidly obese patients, a relatively high-inspired oxygen concentration may be necessary compared to non-obese counterparts, necessitating use of high concentrations of volatile agents. Under general anaesthesia, functional residual capacity (FRC) decreases because of the supine position, use of volatile agents, muscle relaxants and cephalad retraction of a panniculus, leading to early closure of small airways, exaggerating hypoxaemia. Various techniques such as higher tidal volumes, high inspired oxygen and the use of positive end-expiratory pressure (PEEP) have been used to maintain adequate oxygenation. In high-risk cases, emptying of the stomach and administration of sodium citrate via an orogastric tube before extubation may be helpful. Extubation should be attempted only in awake patients with adequate reversal of neuromuscular blockade, as there have been incidences of failed extubation in obese parturients [50, 51]. A 30° head-up tilt is a more favourable position for extubation than supine in the obese population [110].

Postoperative care
Obese parturients are at increased risk of postoperative complications such as hypoxaemia, atelectasis and pneumonia, deep vein thrombosis and pulmonary embolism, pulmonary oedema, postpartum cardiomyopathy, postoperative endometritis and wound complications such as infection and dehiscence [50, 51]. Early mobilisation, thromboprophylaxis, aggressive chest physiotherapy and adequate pain control are the key to the success of effective postoperative care.

In the recovery room, critical respiratory events (desaturation, hypoventilation and airway obstruction) occur twice as commonly in the obese compared to non-obese [111]. Computerised tomography has demonstrated that obesity predisposes to the formation of pulmonary atelectasis per se and even more so under general anaesthesia, persisting into the postoperative period [112]. Moreover, even after spinal anaesthesia, there is a BMI dependent decrease in respiratory function [113]. Hence, these critical respiratory events may not be benign and can lead to postoperative pulmonary morbidity. Nursing in the reclined position and oxygen supplementation can potentially reduce critical respiratory events. Early mobilisation has been shown to improve the respiratory volumes in the immediate postoperative phase [113]. Interestingly, Hood and Dewan found that, in morbidly obese women, all postpartum complications occurred in those undergoing Caesarean section and not in those having vaginal delivery [51]. In general surgical patients, pre-operative pulmonary function tests have been shown to predict postoperative pulmonary complications in obese patients [77] and an extrapolation for obese parturients may be true.

Pain control should be adequate in the postoperative period to facilitate mobilisation and chest physiotherapy, as it is one of the determinants of postoperative maternal morbidity. Epidural analgesia has been shown to improve
postoperative respiratory function in patients undergoing abdominal surgery [114]. Epidural infusion of local anaesthetic with opioids improves the quality of dynamic postoperative pain relief [115]. Patient controlled intravenous opioids have also been successfully used for postoperative pain relief in the morbidly obese [116].

Thrombo-embolic episodes remain the leading cause of direct maternal deaths in the UK. Obesity is a known independent risk factor for deep vein thrombosis. Both pharmacological and mechanical strategies are used for thromboprophylaxis and an adequate dose of an anticoagulant for an appropriate duration is recommended [117]. Obesity cardiomyopathy is a well-recognised clinical entity and at least three cases of peripartum cardiomyopathy in obese patients have been reported [51, 118, 119]. Although not established yet, obesity may well be a risk factor for peripartum cardiomyopathy [119]. Wound complications occur more frequently in obese than in non-obese patients and often lead to prolonged recovery. They have been found to be increased with midline abdominal incision compared to a Pfannenstiel incision [120]. An increased incidence of postoperative complications and antepartum medical disease probably contributes significantly to longer hospitalisation for the morbidly obese. Hospital stay and costs have been found to be increased for morbidly obese patients after both vaginal delivery and Caesarean section [121].

References


56 Noguchi T, Koga K, Shiga Y, Shigematsu A. The gum elastic bougie eases tracheal intubation while applying cri- coid pressure compared to a stylet. *Anesthesia and Analgesia* 2003; **97**: 1331–5.

57 Sankar KB, Krishna S, Moseley HSL. Airway changes in Nigerian obstetric patients. *Anaesthesia* 2000; **55**: 38–42.


102 Taivainen T, Tuominen M, Rosenberg PH. Influence of obesity on the spread of spinal analgesia after injection of plain 0.5% bupivacaine at the L3–4 or L4–5 interspace. British Journal of Anaesthesia 1990; 64: 542–6.