BJMP 2008:1(1) 15-23

Review

Anaesthetic management of obese parturient

Nimit Shah and Yaqub Latoo

SUMMARY

Obesity has become a ticking time bomb. The population of obese people and so also obese pregnant patients is increasing worldwide and it won't be long before when anaesthetists will be more commonly faced with managing obese parturients with a large spectrum of comorbidities. The last confidential enquiry into maternal and child health (CEMACH) 2002 - 2005 report stressed obesity as a major risk factor associated with maternal mortality and following suit of its recommendation, we write this review article on management of obese parturients, highlighting the problems in obese parturients and recommending guidelines for management of such patients. As the use of regional anaesthesia in obstetrics anaesthesia has increased, the trainee anaesthetists are relatively less skilled to provide general anaesthesia. General anaesthesia with all the airway management problems has been the major reason of maternal mortality in the previous CEMACH reports. An epidural block though technically difficult, provides optimal analgesia and can be extended for caesarean section if required. Hence obese parturient should be assessed and consulted by a senior anaesthetist as early as 28 weeks of gestation in the pregnancy for formulating a plan for labour analgesia and anaesthesia for caesarean section if required. Epidural analgesia should be provided in early labour prophylactically to avoid general anaesthesia. Early anaesthetic assessment, prophylactic epidural block, ensuring its effectiveness, alternative plan for failed regional block along with preparation for general anaesthetic and difficult intubation, involving senior help in the management and multidisciplinary approach are advocated to mitigate potential anaesthetic risks.

Abbreviations: BMI - Body mass index

Obesity has become a major health problem of modern society and increasing globally at nearly epidemic proportions especially in western and European countries [1,2,3,4].

DEFINING OBESITY

Obesity can be simply defined as a condition in which body fat is in excess beyond a point incompatible with physical and mental health and normal life expectancy [5] or as a metabolic disorder that is primarily induced and sustained by an over consumption or underutilization of caloric substrate. There are 2 types of obesity; Android obesity which is truncal distribution of fat associated with high incidence of cardiovascular disorders and Gynecoid obesity where fat is distributed to thighs and buttocks associated with pregnancy and not tightly linked to cardiovascular problems [6, 7].

Indices used to for obesity are Ideal body weight in kilograms (Broca's Index), and more commonly the BMI or body mass index (also called Quetelet's index).

1) Ideal body weight = height in centimeters - 100 for men (105 for women). Overweightness is 20% more than ideal body weight and morbid obesity is twice the Ideal Body weight.
2) BMI = weight in kgs/ square of height in meters

PREVALENCE

In the US more than 60 million adults can be classified as either overweight or obese with morbid obesity affecting more than 9 million adults. Approximately 30 – 40 % of females are obese and it is estimated that 50 per cent of women will be obese by 2050. A study looking at trends in pre-partum obesity in nine states of the United States found an increase in pre-partum obesity from 13% in 1993–1994 to 22% in 2002–2003 [8].

WHO CLASSIFICATION OF OBESITY [9]

Classification	Body mass index (kg/m2)	Associated health risks
Underweight	<18.5	Low
Normal range	18.5–24.9	Average
Overweight	>25.0	
Preobese	25.0–29.9	Increased
Obese class I	30.0–34.9	Moderately increased
Obese class II	35.0–39.9	Severely increased
Obese class III	>40	Very severely increased

In the UK, 56% of all women are over the recommended BMI, with 33% of them classified as overweight (BMI > 25) and 23% obese (BMI > 30). The Health Survey of England published in 2002 gives data about the prevalence of obesity in England. Females in the reproductive age group (16 – 44 years) have shown a dramatic increase in BMI. The percentage of women with BMI above 30 increased from 12% in 1993 to 18.3% in 2002. Also alarming is that the percentage of morbidly obese women has doubled in the last decade [10]. The dramatically increasing rate of obesity in the general population also extends to women of reproductive age.

PATHOPHYSIOLOGICAL CHANGES IN OBESE PREGNANT PATIENT

Obesity compounds most of the physiological changes in pregnancy

Airway - Obesity and pregnancy each increase the chance of difficult airway. Limited mouth opening and limited neck movements are common in obesity. There is narrowing of the pharyngeal opening due to excess adipose tissue and on airway examination, the airway will have more commonly high of mallampati grades. In pregnancy, particularly in pregnancy induced hypertension, the mucous membranes in the airway are oedematous and hence more prone to bleeding. Breast enlargement in pregnancy also predisposes to difficult airway.

Respiratory system – There are significant changes in an obese parturient and most of them are additive. In early pregnancy , in a non obese parturient, even before the uterus is large enough to affect respiratory function, women begin to have a sensation of dyspnea. This sensation likely occurs from the increased alveolar ventilation, probably secondary to progesterone effects on the respiratory center in the brainstem [11,12]. By the fifth month of pregnancy, the growing uterus begin to cause a progressive decrease in expiratory reserve volume (ERV), residual volume (RV) and functional residual capacity (FRC), which at term are about 15–20% less than those of the non-pregnant state[13]. Obesity in non-pregnant subjects is associated with a decrease in expiratory reserve volume (ERV), residual volume (RV) and functional residual capacity (FRC), most likely caused by the added weight of excess fat on the chest and abdomen and decreased chest compliance[13 – 16]. Eng et al.[13] showed, however, that obese parturients did not have a significant additional reduction in functional residual capacity (FRC) compared to normal-weight parturients, which might be partially explained by the fact that the study was performed with the parturients in the sitting position. Another possible explanation is progesterone which has a relaxing effect on smooth muscle and decreases airway resistance, thus reducing some of the negative effects of obesity on the respiratory system [12, 17].

Dempsey et al.[18] have showed that excess body weight in obesity increases oxygen consumption and CO2 production in a linear fashion. The work of breathing is increased in obese parturients due to chest wall weight and they typically show a rapid and shallow breathing pattern [18]. This leads in turn to a higher ventilatory requirements and work of breathing [12,19]. The supine, lithotomy, induction of general anaesthesia and especially the Trendelenburg position worsen lung volumes significantly. The functional residual capacity (FRC) is further reduced and the closing capacity (CC) encroaches on the funtional residual capacity (FRC) resulting in small airway collapse, ventilation perfusion mismatch, shunting and hypoxemia[20]. These physiologic changes make the obese parturient particularly prone to rapid desaturation, stressing the importance of adequate denitrogenation ('pre-oxygenation') before induction of general anaesthesia.

In non-obese parturients, physiologic changes during pregnancy are thought to protect from obstructive sleep apnea, due to high circulating levels of progesterone, which is a ventilatory stimulant [18]. However, obesity increases the risk for obstructive sleep apnea significantly and this syndrome is not uncommon in the obese parturients. Obesity hypoventilation syndrome (OHS , Pickwickian syndrome) is seen in 8% of population of obese parturients characterized by morbid obesity, alveolar hypoventilation and daytime somnolence. In response to chronic hypoventilation and hypoxemia, they develop polycythaemia, increased cardiac output, cardiomegaly, pulmonary hypertension and eventually right heart failure. There is a significant increase in morbidity and mortality. They are more to obstructive sleep apnoea. Pulmonary embolism and pneumonia are also common in these patients [25].

Cardiovascular system – Cardiac output increases in pregnancy, with a significant increase in cardiac output, becoming detectable by the third week of pregnancy and a 35 - 40% increase by the end of the first trimester. Cardiac output continues to rise throughout the second trimester until it reaches a level that is approximately 50% more than that in the non-pregnant state. For the remaining pregnancy, cardiac output remains relatively stable around that level. During labour, cardiac output increases further by approximately 10% in the early first stage, 25% in the late first stage and 40% in the second stage. Uterine contractions increase cardiac output by further 10–15% and in the immediate post-partum period the cardiac output peaks at as much as 75% above prepartum values [19]. Obesity increases cardiac output even further because of extra amount of fat. Every 100 g of fat increases the cardiac output by 30–50 ml/min[22]. Blood volume is increased in pregnancy and even more when pregnancy is complicated by obesity. In non-obese parturients, there is a significant reduction in afterload [22]. In obese pregnant parturients, however, afterload reduction may be impaired due to increased peripheral resistance and greater conduit artery stiffness [23]. Additionally, obesity is associated with a higher prevalence of hypertension, diabetes mellitus, hyperlipidemia and poor cardiac function and it is one of the leading risk factors for coronary artery disease and cerebrovascular accidents [24]. Due to hyperdynamic circulation, there ensues left ventricular hypertrophy and diastolic dysfunction. Systolic function might remain normal but progressively systolic dysfunction may ensue. Pulmonary blood volume increase due to increased cardiac output. Pulmonary hypertension can develop and is exacerbated by supine position, airway obstruction and hypoxemia can develop. In obesity hypoventilation syndrome, right ventricular failure can develop. Increased number of peripartum cardiomyopathy cases are seen in obese pregnant parturients but it is unclear if obesity is a risk factor [25].

The obese pregnant parturients are at an increased risk of supine hypotension syndrome (SHS) due to compression of major abdominal vessels. This is exacerbated by large panniculus which adds to the uterine compression. Tseuda et al. have reported two cases of sudden death on assuming the supine position in morbidly obese patients [26].

Gastrointestinal system – Obesity further decreases lower oesophageal tone which is already decreased in pregnancy and increase the risk of aspiration of gastric contents and Mendelson's syndrome [27, 28]. Hiatus hernia is increased in obese patients. Roberts and Shirley studied obese and non obese pregnant parturients in labour; the gastric volume in obese parturients is five times greater than in the controls [29,30,31]. Obese population have a higher incidence of diabetes, which can cause delayed gastric emptying, increasing the risk for aspiration. Also,

it is well known that obesity predisposes to difficult or failed intubation, both of which are associated with a higher incidence of aspiration.

Others - Gestational diabetes is common. Obesity metabolic syndrome includes dyslipidemia, impaired endothelial function, high blood pressure, increased inflammatory mediators, insulin resistance and hyperinsulinemia even in absence of diabetes [25].

PHARMACOKINETICS AND PHARMACODYNAMICS CHANGES

Obesity increases both fat and lean masses; however, the percentage of fat tissue increases more than does the lean mass, affecting the apparent volume of distribution of anaesthetic drugs according to their lipid solubility. Thiopental sodium and propofol dosages are calculated on total body weight (TBW). Benzodiazepine loading doses should be adjusted on actual weight, and maintenance doses should be adjusted on ideal body weight. The loading dose of lipophilic opioids is based on total body weight (TBW), whereas maintenance dosages should be cautiously reduced because of the higher sensitivity of the obese patient to their depressant effects. Pharmacokinetic parameters of muscle relaxants are minimally affected by obesity, and their dosage is based on ideal rather than total body weight (TBW). Minimum alveolar concentration is decreased. Inhalation anaesthetics with very low lipid solubility, such as sevoflurane and desflurane, allow for quick modification of the anaesthetic plan during surgery and rapid emergence at the end of surgery, hence representing very flexible anaesthetic drugs for use in this patient population. Drug dosing is generally based on the volume of distribution for the loading dose and on the clearance for maintenance. In the obese patient, the volume of distribution is increased if the drug is distributed both in lean and fat tissues whereas the anaesthetic drug clearance is usually normal or increased [32]. Albumin binding of drugs is unchanged in the obese, but levels of fatty acids, triglycerides, and a1-acid glycoprotein are increased and may influence plasma protein binding. In pregnancy, the volume of distribution is increased, albumin concentration decreased and the renal clearance is increased. Net effect is unpredictable. Pseudocholinesterase levels are decreased in pregnancy.

LOCAL ANAESTHETIC REQUIREMENTS

Lower dose of local anaesthetic is required (less by 25%) when injected neuraxially. Proposed mechanisms are pregnancy induced hormone related changes in the action of spinal cord neurotransmitters, potentiation of the analgesic effect of the endogeneous analgesic systems, increased permeability of the neural sheath [25] and decreased dilution by decreased volume of cerebrospinal fluid (CSF) Hodgkinson et al [33] have shown an increased cephalad spread of local anesthetics in obese patients. Hogan et al.[34] found a lower average cerebrospinal fluid (CSF) volume in obese subjectsI, which could explain the decreased local anesthetic dose requirements due to decreased anaesthetic dilution. Since similar changes were noticed with external abdominal compression and abdominal pressure increases linearly with increased body weight [35], increased abdominal pressure is probably the cause. Some [36] have also attributed the decrease in cerebrospinal fluid (CSF) volume to compression of the dural sac due to engorgement of the epidural venous plexus and increased epidural space pressure, resulting from compression of the inferior vena cava by gravid uterus with redistribution of the venous return from the lower limbs and pelvis. Hogan et al.[34], however, suggested that the mechanism by which increased abdominal pressure decreases the CSF volume is probably inward movement of soft tissue (mostly fat) in the intervertebral foramen, which displaces CSF. This hypothesis is based on their findings that the greatest change in CSF volume during abdominal compression was found at sites in which intervertebral foramina were present. Greene suggests that larger buttocks of obese patients place the vertebral coloumn in the Trendelenburg position, exaggerating the cephalad spread of the local anaesthetic [25, 37]

CONSEQUENCES OF OBESITY IN PREGNANCY

Obesity severely complicates pregnancy. It affects both the mother and foetus

Maternal consequences

There is increased risk of gestational diabetes and type 2 Diabetes. There is 3 - 10 fold higher risk of gestational diabetes (type 1 insulin dependent diabetes mellitus)[38]. Studies have shown when pregnancy is complicated by gestational diabetes, there is a higher risk of developing type 2 diabetes mellitus in later life [39]. There is a 2 – 4 fold increased risk of preeclampsia. The risk is almost 5 times greater in the morbidly obese group; typically a BMI > 35 [38, 40]. But there is no increased risk of HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome [31]. Obesity is an independent risk factor for hypertension [41].

It is reported [42] that there is a higher chance of failure to progress, prolonged second stage of labour and a failed induction of labour in obese compared to non obese parturients and this is secondary to soft tissue dystocia. There is a higher risk of instrumental delivery of up to 18% in women with a BMI between 35 and 40 and up to 34% in patients with BMI greater than 40. Also there is an increased risk of failed instrumental delivery leading to caesarean section. There is 3 times higher risk of caesarean section in a obese parturient. This is due to fetal macrosomia, higher risk of shoulder dystocia and/or failed cervical dilatation [38, 43, 44]. About two thirds present as emergency caesarean section [45]. The obese parturient is at a higher risk of having a prolonged incision to delivery time, blood loss greater than 1000 ml and prolonged operative times. There is an increased risk of wound infections and endometritis and dehiscence [46, 47]. There is an increased risk of major postpartum haemorrhage. The risk of postpartum haemorrhage rises with increasing BMI and is about 30% more frequent for a moderately raised BMI and about 70% more frequent for a highly raised BMI compared with the normal BMI group [45,48]. There is an increased risk of thromboembolism – obesity and pregnancy are each independent risk factors for deep vein thrombosis. Both pharmacological and mechanical methods should be used for thromboprophylaxis.

Obese women spend an average of 5 more days in hospital resulting in 5 fold increase in cost of care due to potential complications such as wound infections and postpartum haemorrhage [49].

Fetal consequences

Maternal obesity is associated with large for gestational age infants. There is increased risk of a macrosomic foetus, independent of maternal diabetes [43, 50]. There is an increased risk of shoulder dystocia upto three times more common in the morbidly obese parturients. The risk of foetal macrosomia and shoulder dystocia increases with increase in BMI [44].

There is an increased risk of Infant birth defects. Since 1994 a number of studies have shown an association between maternal obesity and infant birth defects. Anomalies include neural tube defects such as anencephaly, anomalies of the heart and intestinal tract, omphaloceles, orofacial clefts, and multiple congential anomalies of the central nervous system [43, 51, 52].

There is an increased risk of stillbirth, a three times increase in antepartum stillbirth was found in morbidly obese parturients compared with women of normal BMI [52, 53].

Due to the depth of maternal adipose, foetal monitoring by intermittent or continuous Electronic Foetal Monitoring using external transducers may be technically difficult. The use of fetal scalp electrodes and intrauterine pressure catheters to ensure an acceptable standard of fetal monitoring may be needed.

In a study 'Maternal obesity and pregnancy outcome: a study of 287213 pregnancies in London' (N J Sebire, et all, International journal of obesity (2001) 25, 1175 - 82) complications such as gestational diabetes mellitus proteinuric pre-eclampsia; induction of labour; delivery by emergency caesarean section; postpartum haemorrhage; genital tract infection; urinary tract infection, wound infection; birth weight above the 90th centile, and intrauterine death were significantly higher in obese pregnant parturients than non obese pregnant parturients. However, delivery before 32 weeks gestation and breastfeeding at discharge were significantly less likely in the overweight groups. In all cases, increasing maternal BMI was associated with increased magnitude of risk [38]. Weiss et al.[54] found for nulliparous patients a caesarean delivery rate of 20.7% in the control group compared with 33.8% in the obese and 47.4% in the morbidly obese group.

The Confidential Enquiry into Maternal and Child Health 2004 reported that 35% of all maternal deaths occurring in the triennium 2000–2002 were in obese women with BMI > 30. The most recent CEMACH reports in the United Kingdom reported that obesity was a cofactor in a significant number of the maternal deaths between 2003 and 2005. Twenty seven percent of the women who died had BMI > 30. Of these women, 12% had a BMI between 30 and 34, 7% had values between 35 and 39 and 8% had a BMI of 40 or more. 295 women who died 119 were overweight and 64 of those were morbidly or super-morbidly obese. In 30 per cent of women who experienced a stillbirth or perinatal death, the maternal BMI was recorded at more than 30.

Furthermore, Cedergren et al found a 3 fold increased rate of stillbirths, 5 fold increased risk of preeclampsia and a 3 fold increased risk of caesarean section. The success rate for a vaginal delivery in obese parturient with a previous caesarean section is less than 15% [38].

ANAESTHETIC MANAGEMENT

Obese parturients have severely limited physiological reserve and a higher risk of emergency surgical intervention. Hence the anaesthetic risks increase greatly. Obesity and pregnancy each has multisystem effects, many of which are additive. A thorough understanding of the physiology, associated conditions and morbidity, available options for anaesthesia and possible complications is important.

Senior anaesthetist must be involved early in multidisciplinary approach for patient care as early as 28 weeks of gestation. The preoperative assessment include evaluation of airway, respiratory and cardiovascular system and pregnancy associated problems such as pregnancy induced hypertension, gestational diabetes etc and also should include patient education. An examination of the back should be done.

Airway

The obese parturients need thorough pre-operative assessment for difficult airway as incidence of failed intubation is 8 times higher than non obese patients. In the obstetric population, between one in 280 and one in 750 attempted tracheal intubations fail [45], compared to one in 2230 in the general population [17, 55,56]. In contrast, the incidence of difficult intubation in obese population, is as high as 15.5% [57]. Dewan [58] found the incidence as high as 33% in morbidly obese parturients. A 6-year review of failed intubations in parturients in a United Kiingdom region reported 36 cases of failed intubation and it was found that the average BMI of these women was 33 [57]. So it is evident that incidence of difficult or failed tracheal intubation in obese parturients is very high and emphasizes optimal assessment and management of the airway. An airway assessment should include mallampati classification, thyromental distance, neck extension (atlanto-occipital joint extension), mouth opening (vertical dimension). The combination of two tests (mallampatti and thyromental distance), though in a small study of 80 parturients 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 minute; hence they are also useful in an emergency scenario. Other features shown to be of significance are short neck, receding mandible and protruding incisors [60]. It is of interest to note that neck circumference, not BMI, is more predictive of a difficult intubation in morbidly obese patients [61]. A study has shown a gestational weight gain of more than 15 kgs is associated with three times increase in suboptimal laryngoscopic view as compared to that in non obese parturients of the same age [57, 62]. This means weight gain in pregnancy should be limited in obese parturients and if an obese parturient presents who has gained more than 15 kgs of weight in pregnancy, she will be more likely to have a difficult airway [62]. A plan of airway management should be formulated in case of an emergency for all women regardless of the primary obstetric and anaesthetic plan. Although rapid sequence intubation with proper positioning and back up equipment may be adequate for most women, an alternative airway plan should be considered. A history of snoring, diagnosis of sleep apnoea, lack of teeth, and large breasts all increase risk of difficult intubation and awake fibreoptic intubation should be considered in all patients with limited range of neck, head or jaw movements, short neck, neck

circumference of 15 inches and above, and mallampati score of 3 and above [25]

Respiratory System

Usually a complete history and chest examination and routine investigations including an ECG is adequate for a preoperative anaesthetic fitness. However chest X ray, arterial blood gas, pulmonary function tests can be done to aid further evaluation of respiratory reserve. Measurement of oxygen saturation by pulse oximetry in sitting and then supine can provide evidence of airway closure during normal tidal volume ventilation, thereby identifying candidates for post operative oxygen administration [17].

Women with obesity are more likely to have obstructive sleep apnoea but the prevalence is unknown in pregnancy. Sleep disturbances and day time fatigue are normal at the end of pregnancy and so obstructive sleep apnoea may go undiagnosed. J Mhyre [25] has suggested women with a BMI > 35, neck circumference of greater than 16 inches, symptoms of suspected airway obstruction during sleep (include frequent or loud snoring, observed pauses in breathing during sleep, frequent arousals from sleep or arousal with a choking sensation) should be screened by polysomnography for obstructive sleep apnoea and advised continuous positive airway pressure (CPAP) if required.

If obesity hypoventilation syndrome is suspected arterial blood gas is useful to screen hypoxia, hypercarbia and acidosis and echocardiogram should be done to evaluate cardiac function and patient should be referred to cardiologist [25,63]

Cardiovascular system

Cardiovascular co-morbidities such as hypertension, ischaemic heart disease and heart failure can co-exist in obese parturients. Nearly 40% of the obese population experience angina without demonstrable coronary artery disease [64]. Pulmonary hypertension can be present. Hence cardiologists should be involved early in the care of symptomatic morbidly obese parturients to investigate and optimise the disease status wherever appropriate [17]. Echocardiogram may be useful.

The obese parturients cannot be accurately stratified for perioperative risk using the usual screening indices such as Goldman's index etc as obesity and pregnancy are not included as risk factors in these indices and they might be classed in the lower risk group despite having significantly increased risk.

Others

Patients should be assessed for pregnancy associated problems such as pregnancy induced hypertension and gestational diabetes mellitus etc.

Peri-operative issues such as transfers, beds, intravenous access, central venous access, difficulty in measuring non invasive blood pressure, arterial cannulation, different size regional anaesthesia kit should be anticipated, discussed and planned for.

Post operative intensive care management /high dependency care should be sought for. Deep vein thrombosis prophylaxis must be put in place. The management plan should be liaised with whole team including consultant anaesthetists, consultant obstetricians, consultant intensivists, midwives, operating department practioners (ODP's) and physiotherapists

ANALGESIA FOR LABOUR

Each of the risk factors of fetal macrosomia and shoulder dystocia which are increased in obese parturient result in more painful contractions and complicated labour [65]. Although there are various modalities of pain relief, analgesia using neuroaxial blockade has been shown to be the most effective [66]. The anticipated technical difficulties should not preclude the use of epidural analgesia in obese parturients. It is been shown effective pain relief during labour can improve maternal respiratory function and attenuate sympathetically mediated cardiovascular responses [67, 68]. Available evidence shows that the rate of caesarean delivery does not increase with epidural analgesia during labour [66], 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 problems include appropriate positioning of the patient, identification of the midline and epidural space, and dislodgement of catheters [45, 69, 70]. The initial failure rate for epidural catheter placement can be very high (42%) [45] 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 [71]. The knee–chest position required for doing epidural in the lateral position is difficult to obtain in the obese. One study has shown that cardiac output decreased more in the lateral decubitus position with maximal lumbar flexion compared with the sitting position [72]. 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 [73]. Overall, the sitting position is preferable and should be used.

STEPS AND CAVEATS

Early placement and confirmation of optimal epidural analgesia even before onset of labour (when a term patient presents before labour) is prudent. This allows sufficient time to manage a failed epidural block (because not only the incidence of failed initial epidural catheter placement is high in obese parturients, but the incidence of failed epidural during labour due to migration of epidural catheter in the fatty subcutaneous tissues is also high)[17, 74, 75]. Re-evaluate the airway and cardiorespiratory status.

Senior anaesthetist preferably a consultant anaesthetist should be involved. Ensure wide intravenous cannula (preferably 14 or 16 gauge) in place. In case of problems with blood pressure cuff not measuring, cuff can be placed on calf/forearm; will help in getting the trends if no accurate reading. Invasive blood pressure monitoring might be needed. Ensure pulse oximetry monitoring and supplement oxygen by mask if required.

Perform in sitting position. Ensure midline position as even if slight deviation of the midline will lead to exaggerated directional errors due to increased length of epidural space from the skin and hence failure of epidural. Midline might be not possible to palpate, in this case drop a line from C1 spinous process to lower skin crease and this may be guide as a midline. Strapping excess fat away from the midline might be necessary.

If highest points of iliac crests are palpated for the Tuffier's line then because of fat pads on the sides, higher spaces might be inadvertently selected and increased chance of spinal cord damage. In case of difficulty, lower thoracic space may be selected.

A recent study in pregnant patients has shown a positive correlation between BMI and the distance to skin to the lumbar puncture [76]. 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 cms [73, 77]. Hence it seems appropriate to use a standard needle to identify the epidural space on the first attempt. In morbidly obese patient ultrasound technique has been found valuable in establishing epidural [78, 79]

In case of difficulty in insertion, a deliberate spinal with 25 guage needle might be performed (no injection of drugs) to assess the midline and depth of epidural space. There is an increased risk of dural tap [25], but decreased risk of postdural puncture headache [75]. In case of dural tap, epidural can be converted to continuous spinal catheter analgesia with extreme caution. Also there is an increased risk of Intravenous placement of epidural catheter due to engorged epidural veins and decrease in epidural space. The meniscus drop (negative pressure) test is not reliable as epidural pressure may be high [25]. Minimum 5 cms of catheter in space should be left. To minimize catheter displacement, it should be secured on assumption of upright or preferably lateral position from the initial flexed position. The epidural should be checked with a test dose and a functioning epidural should be ensured. Sometimes a longer epidural needle might be required. There is an advantage to titrate block height. Minimum local anaesthetic concentration (MLAC) is lower in obese pregnant patients compared to non pregnant patients [80]

If epidural is contraindicated or impossible to site then entonox is an useful adjunct. Intramuscular opioids are not reliable. Patient controlled analgesia can be used but cautiously as increased chance of sedation and respiratory depression. Remifentanyl, an ultra short acting opioid, has favourable pharmacokinetics to be used as an opioid for patient controlled analgesia but not enough data is available for its use in obese parturients. It is metabolized by red blood cells and tissue esterases both in mother and foetus and hence does not accumulate and is easily antagonized if required. However it is a potent respiratory depressant and hence should be used very cautiously in obese parturients who would be susceptible to its sedative side effects and hence they should be managed in high dependency unit with appropriate monitoring and one to one nursing by skilled midwife and under observation of a highly skilled anaesthetist. The dosage should be carefully titrated individually and naloxone and difficult airway trolley ready. Patients with obstructive sleep apnoea would be very susceptible to its sedative side effects and hence should be avoided. Proper training of patients is required as its peak effect is 2 -3 minutes and if the button of patient controlled analgesia is pressed at the onset of contraction it would be less effective. The duration of its use should be minimized as much as possible.

ANALGESIA/ANAESTHESIA FOR CAESAREAN SECTION

Obesity and Caesarean section have been identified as independent risk factors for maternal morbidity and mortality [44]. Analysis of direct maternal deaths due to anaesthesia, in the confidential enquiries report on maternal mortality in the United Kingdom from 1979 to 2005, reveals that the majority of deaths occurred under general anaesthesia, compared with regional anaesthesia [17]. Most parturients who die of complications of general anaesthesia die of airway management problems, including aspiration, failed intubation, inadequate ventilation, and respiratory failure.[54, 55]

Factors that play a role in general anaesthesia being more likely to be associated with maternal mortality than regional anaesthesia are unexpected airway difficulties, pulmonary aspiration of gastric contents, emergency general anaesthesia (including conversion of a failed regional), peripartum haemorrhage, and embolism necessitating general anaesthesia, and resident lack of experience in general anaesthesia for caesarean section [28 , 81].

In Why Mothers Die 2000–02, 35% of all the women who died were obese, 50% more than in the general population [82]. Hence regional anaesthesia preferably epidural should be opted unless contraindicated or difficult.

Direct maternal deaths due to anaesthesia by types of anaesthesia in United Kingdom 1979–2005. Derived from CEMD reports. Since 1979, maternal deaths are reported as direct and indirect.

Year	Total(n)	GA(n)	RA(n)	Other (n)
2002-05	6	4	1	1
2000-02	6	6	0	0
1997-99	3	2	1	0
1994-96	1	0	1	0
1991-93	8	7	1	0
1988-90	4	3	1	0
1985-87	8	7	1	0
1982-84	18	17	1	0
1979-81	22	22	0	0

GA, general anaesthesia; RA, regional anaesthesia; Other – eg Central VP line insertion

REGIONAL ANAESTHESIA FOR CAESAREAN SECTION

Different techniques can be used. Epidurals are reliable but have high failure rate, spinal is a familiar technique while combined spinal epidural has minimal side effects such as headache, high block , hypotension and can be used post operatively as well as for redo surgery.

Use 25% less local anaesthetic dose compared to non obese patient due to altered neuro-axial physiology and anatomy.

Epidural

A working epidural as above can be continued for caesarean section and it also provides post operative pain relief. However it may be inadequate in more than 25% of these patients, mainly because of difficulty in blocking the sacral roots, resulting in visceral pain upon stimulation of the bladder [83].

Spinal

An obese woman is a candidate for spinal anaesthesia if the airway examination is normal, cardiopulmonary derangements are minimal and the obstetricians aim to complete the surgery within 90 minutes [25]. In obese parturients spinals can be technically difficult requiring varied needle lengths and being unable to titrate to block for surgery and surgical duration. If the spinal wears off, general anaesthesia with all its inherent risks, will be required. Tuohy needle can be used as an introducer for the spinal needle [25]. Spinal opioids can provide post operative analgesia but respiratory monitoring becomes essential.

Combined Spinal Epidural

Success of combined spinal epidural will depend on familiarity with technique. It is more versatile to titrate the block and dose and also a faster onset compared to epidural alone. This technique can be useful for post operative analgesia and re-operative anaesthesia [84]. There is higher rate of success for surgical anaesthesia compared to spinal or epidural alone. Several studies have shown that catheters inserted as a part of combined spinal epidural technique produce anaesthesia /analgesia more reliably than those inserted via a standard epidural technique [85 – 88]. The appearance of cerebrospinal fluid indirectly confirms correct epidural needle placement and increase the chance of functional epidural catheter. There is a possible flaw when spinal injection alone produces the desired block and epidural remains untested; when epidural is required and fails, general anaesthetic might be needed [25] and hence a small dose intrathecally might be used to establish the analgesia to make mother pain free (which therefore also decreases the risk of hypotension) and then epidural should be used to make sure it is working for the complete surgical anaesthesia.

Continious Spinal anaesthesia

Operators need to be familiar with technique. Continuous spinal anaesthesia must be done always with consultant anaesthetist. It is occasionally used in patients who have accidental dural puncture. It may be used when epidural is indicated and difficult to site. It provides reliable and predictable block and allows to titrate the block to desired level and duration. It provides surgical anaesthetic level within minutes in emergency situations with incremental doses. It is important to flush the catheter before placement to avoid introducing air into the spinal space which could cause pneumoencephalus headache [25]. It is also very important to mark it as an intrathecal catheter and to be used by anaesthetist only. This can be used for analgesia as well as anaesthesia.

Incidence of headache and infection is higher with this technique compared to other regional techniques but overall incidence of post dural puncture headache in obese parturients is lower [89, 90]. Final density and level are proportional to the dose in mgs, not the volume delivered

GENERAL ANAESTHESIA FOR CAESAREAN

Consultant anaesthetist should be involved as early as possible. Strategy should be to avoid need for emergency general anaesthesia by being proactive and establishing effective regional analgesia and anaesthesia as early as possible. Airway assessment regarding difficult airway must be done. Preparation for general anaesthesia and difficult intubation (ensure lower sized endotracheal tube and a laryngeal mass airway) must be in place including awake fibre optic laryngoscope. Anti-aspiration prophylaxis must be given before conduct of anaesthesia

Collins et al. [91] investigated the effect of the position of the patient on the view obtained during laryngoscopy in 60 morbidly obese patients. They found that the 'ramped' position, accomplished by arranging blankets underneath the patient's upper body and head until horizontal alignment is achieved between the external auditory meatus and the sternal notch, clearly improves the laryngeal view when compared with the standard 'sniff' position. HELP (Head elevated laryngoscopy position) should be given to make sure that airway is in alignment.

After all monitoring including foetal monitoring is in place, patient must be prepared awake and draped. Adequate preoxygenation { 8 vital capacity breaths of 100% oxygen [92]} is ensured as otherwise they rapidly desaturate. Baraka et al. [92] showed that pre-oxygenation achieved by eight vital capacity breaths within 60 s at an oxygen flow of 10 liters/min not only results in a higher partial pressure of arterial oxygen (PaO2), but also in a slower hemoglobin desaturation when compared with the four deep breaths technique. Use standard rapid sequence induction with cricoid pressure and left lateral tilt in patients with no anticipated difficult airway. For general anaesthesia make sure drug doses for (thiopentone, suxamethonium, atracuruim) are calculated before hand keeping in view altered distribution and elimination in obese patients. Dewan suggests that at least 4 mgs/kg of thiopentone (up to a maximum dose of 500 mgs) should be used if chosen, to avoid the risk of maternal awareness, hypertension and decreased uterine blood flow during light anaesthesia [93]. Administration of a larger dose may be associated with delayed arousal in the event of failed intubation. For suxamethonium, dose based on 1 - 2 mgs/kg of actual bodyweight up to maximum of 200 mgs [93].

Tracheal intubation should be confirmed by capnography in addition to auscultation. Endobronchial intubation should be promptly recognized and managed. 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 suxamethonium 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 foetal wellbeing or risk of regurgitation [17].

Patients will need suitable ventilators for adequate ventilation. They need large tidal volumes of 10–12 mls/kg and positive end expiratory pressure (PEEP) may be avoided [25], as though it increases partial pressure of oxygen in blood (PaO2) [25], it might decrease cardiac output and oxygen delivery to foetus [25]. Extubation must be done when awake in left lateral position or semi upright position after adequate reversal of muscle relaxant

Antibiotic prophylaxis is a must as high incidence of wound infection in these patients [46, 47]. There is increased risk of post operative respiratory failure and hence morbidly obese parturients are best managed in intensive care management or high dependency care post operatively after general anaesthesia [75]. Adequate pain control (Patient controlled analgesia / patient controlled epidural analgesia (PCA/PCEA) to assure post op deep breathing. Infiltrative analgesia at the end of surgery can be carefully used to decrease requirement of post op analgesia. Post operative oxygen should be given and continuous positive airway pressure if required.

Thromboprophylaxis should be given after liasing with the obstetricians as to the dose and frequency required. Both pharmacological and mechanical methods and early mobilization should be used for thromboprophylaxis. It has been suggested that low molecular weight heparin (LMWH) dosing should be based on actual body weight [94].

The anticoagulation status of the patient becomes particularly important for the anesthesiologist when the patient has a spinal or an epidural catheter. According to European guidelines (when a single daily dosing of low molecular weight heparin (LMWH's) is used), catheters can be removed 10–12 hrs after the last dose of low molecular weight heparin (LMWH) and 4 hrs before the next dose.

Subcutaneous and Intramuscular routes of drug administration should be avoided as they are less reliable.

CONFLICT OF INTERESTS
None declared

AUTHOR DETAILS
NIMIT SHAH, FRCA, FFARCSI, DNB. Specialist registrar, Norfolk and Norwich hospital, Norwich NR4 7UY.
YAQUB LATOO FRCA. Consultant Anaesthetist, Bedford hospital, Bedford, MK42 9DJ.

CORRESPONDENCE: Dr Y Latoo, Consultant Anaesthetist, Bedford hospital, Kempston Road, Bedford, MK42 9DJ.
Email: yaqublatoo@aol.com

REFERENCES

1. Seidell JC. Epidemiology of obesity. Semin Vasc Med 2005; (1):3–14.
2. Friedman N et al. Overweight and obesity: an overview of prevalence, clinical impact, and economic impact. Dis Manag 2004; 7 (Suppl 1):S1–S6.
3. Malecka-Tendera E et al. Childhood obesity: a pandemic of the twentyfirst century. Int J Obes 2006; 30 (Suppl 2):S1–S3.
4. Deitel M. Some consequences of the global obesity epidemic. Obes Surg 2005; 15:1–2.
5. National Institutes of Health Consensus Development Conference Statement. Health implications of obesity. Ann Int Med 1985; 103: 147–51
6. Bray GA (1985) complications of obesity. Ann Intern Med 103:1052 – 1062
7. M Tanaka et al – Anesthetic management in obese parturient, J of Anesthesia (1999), 13:217 - 229
8. Kim SY, Dietz PM, England L et al. Trends in pre-pregnancy obesity in nine states, 1993–2003. Obesity (Silver Spring) 2007; 15: 986–93.
9. World Health Organization. Obesity: Preventing and Managing the Global Epidemic. Report on a WHO consultation, WHO Technical Report Series 894. Geneva: WHO, 2000
10. Department of Health, London. Health survey for England 2002 – trends [WWW document].
11. Naimark A, Cherniak R. Compliance of the respiratory system and its components in health and obesity. J Appl Physiol 1960; 15: 377–82.
12. Unterborn J. Pulmonary function testing in obesity, pregnancy, and extremes of body habitus. Clin Chest Med 2001; 22: 759–67.
13. Eng M, Butler J, Bonica JJ. Respiratory function in pregnant obese women. Am J Obstet Gynecol 1975; 123: 241–5.
14. Cullen JH, Formel PF. The respiratory defects in extreme obesity. Am J Med 1962; 32: 525–31.
15. Lee JJ, Larsen RH, Buckley JJ et al. Pulmonary function and its correlation to the degree of obesity in 294 patients. Anesth Rev 1981; 8: 28–32.
16. Vaughan RW, Cork RC, Hollander D. The effect of massive weight loss on arterial oxygenation and pulmonary function tests. Anesthesiology 1981; 54: 325–8.
17. Saravanakumar K etal. Obesity and obstetric anaesthesia. Anaesthesia 2006; 61:36–48.
18. Lefcourt LA, Rodis JF. Obstructive sleep apnea in pregnancy. Obst Gynecol Surv 1996; 51: 503–6
19. Chang AB. Physiologic changes of pregnancy. In: Chestnut DH, ed. Obstetric anesthesia: principles and practice, 3rd edn. Philadelphia: Elsevier Mosby, 2004: 15–36.
20. Øberg B, Poulsen TD. Obesity: an anaesthetic challenge. Acta Anaesthesiol Scand 1996; 40: 191–200.
21. Stephansson O et al. Maternal weight, pregnancy weight gain and the risk of antepartum stillbirth. American Journal of Obstetrics and Gynecology 2001; 184: 463–9.
22. Veille JC, Hanson R. Obesity, pregnancy, and left ventricular functioning during the third trimester. Am J Obstet Gynecol 1994; 171: 980–3.
23. Vasan RS. Cardiac function and obesity. Heart 2003; 89: 1127–9.
24. Tomoda S, Tamura T, Sudo Y et al. Effects of obesity on pregnant women: maternal hemodynamic change. Am J Perinat 1996; 13: 73–8.
25. J Mhyre – anaesthetic management of morbidly obese pts
26. Tsueda K et al, Obesity supine death syndrome: reports of two morbidly obese patients. Anesthesia and Analgesia 1979; 58: 345–7.
27. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. Am J Obstet Gynaecol 1945; 52: 191–204.
28. Olsson GL, Hallen B, Hambraeus-Jonzon K. Aspiration during anaesthesia: a computer-aided study of 185358 anaesthestics. Acta Anaesthesiol Scand 1986; 30: 84–92.
29. Brock Utne JG, Dow TG et al gastric and lower oesophageal pressure in early pregnancy. Br J Anaestheisa 1981; 53: 381 -384
30. Obrien TG Jr; LOS pressure and oesophageal function in obese humans J clin gastroenterology. 1980; 2:145 -148
31. Roberts RB, Shirley MA – reducing the risk of acid aspiration during caesarean section, anaesthe analgesia 1974; 53: 859 - 868
32. A . Casati , M . Putzu, Anesthesia in the obese patient: Pharmacokinetic considerations . Journal of Clinical Anesthesia , Volume 17 , Issue 2 , Pages 134 - 145
33. Hodgkinson R, Husain FJ. Obesity and the cephalad spread of analgesia following epidural administration of bupivacaine for cesarean section. Anesth Analg 1980; 59: 89–92.
34. Hogan QH, Prost R, Kulier A et al. Magnetic resonance imaging of cerebrospinal fluid volume and the influence of body habitus and abdominal pressure. Anesthesiology 1996; 84: 1341–9.
35. Hackney JD, Crane MG, Collier CC et al. Syndrome of extreme obesity and hypoventilation: studies of etiology. Ann Int Med 1960; 51: 541–52.
36. Greene NM. Distribution of local anesthetic solutions within the subarachnoid space. Anesth Analg 1985; 64: 715–30.
37. Greene NM Distribution of local anesthetics within the subarachnoid space. Anesthesia and Analgesia 1985;64:715 -30
38. Maternal obesity and pregnancy outcome: a study of 287 213 pregnancies in London N J Sebire et al International Journal of Obesity (2001) 25, 1175 - 82
39. Gabbe SG : Gestational Diabetes mellitus NEJM 315 (1986) 1025
40. Kumar AS – Pregnancy outcome in women with morbid obesity J Gynaecology Obstetric 97 (2001) 101

41. Gross T etall - Obesity in pregnancy : risks and outcome. Obstet Gynaecol 56 (1980) 446
42. Johnson SR et al. Maternal obesity and pregnancy. Surg Gynaecol Obstet 164: 431 -7
43. Ehrenburg HM et al. The influence of obesity and diabetes on the prevalence of macrosomia. American Journal of Obstetrics and Gynecology 2004; 191: 964–8.
44. Weiss et all – Obesity, obstret complications and caesarean delivery rate American J Obstetr GYnaec 190 (2004) 1091 -7
45. Hood DD et al. Anesthetic and obstetric outcome in morbidly obese parturients. Anethesiology 1993; 79:1210–1218.
46. Usha Kiran TS et al. Outcome of pregnancy in a woman with an increased body mass index. British Journal of Obstetrics and Gynaecology 2005; 112: 768–72.
47. Myles TD et al. Obesity as an independent risk factor for infectious morbidity in patients who undergo cesarean delivery. Obstetrics and Gynecology 2002; 100: 959– 64.
48. Andreasen KR et al. Obesity and pregnancy. Acta Obstetricia et Gynecologica Scandinavica 2004; 83: 1022–9.
49. Galtier-Dereure F et al. Obesity and pregnancy: complications and cost. American Journal of Clinical Nutrition 2000; 71 (Suppl.): S1242–8.
50. Obesity in pregnancy. Yu CK, Teoh TG, Robinson S BJOG. 2006 Oct;113(10):1117-25. Epub 2006 Aug 10
51. Watkins ML et al. Maternal obesity and risk for birth defects. Pediatrics 2003; 111: 1152–8.
52. Stephansson O et al. Maternal weight, pregnancy weight gain and the risk of antepartum stillbirth. American Journal of Obstetrics and Gynecology 2001; 184: 463–9.
53. Cedergren Mi : Maternal morbid obesity and risk of adverse pregnancy outcome Obstetr Gynecol 103 (2004) 219
54. Weiss JL, Malone FD, Emig D et al. Obesity, obstetric complications and cesarean delivery rate – A population-based screening study. Am J Obstet Gynecol 2004; 190: 1091–7.
55. Munnur U et al , Airway problems in pregnancy. Crit Care Med 2005; 33 (10 Suppl):S259–S268.
56. Schneider MC. Anaesthetic management of high-risk obstetric patients. Acta Anaesthesiol Scand 1997; 41 (1 Suppl 111):163–165.
57. Juvin P, et al. Difficult tracheal intubation is more common in obese than lean patients. Anesthesia and Analgesia 2003; 97: 595 – 600.
58. D'Angelo R, Dewan DD. Obesity. In: Chestnut DH, ed. Obstetric Anesthesia: Principles and Practice, 2004: 893–903
59. Merah NA et al. Prediction of difficult laryngoscopy in a population of Nigerian obstetric patients. West African Journal of Medicine 2004; 23: 38–4.
60. Rocke DA et al,. Relative risk analysis of factors associated with difficult intubation in obstetric anesthesia. anesthesiology 1992; 77: 67–73.
61. Bell RL et al. Postoperative considerations for patients with obesity and sleep apnea. anaesthesiol clin N Am 2005; 23:493–500
62. Shankar KB et all – airway changes during pregnancy 1997 – anaesthesiology 87: A895
63. ACOG committee opinion number 315. Obesity in pregnancy – Obstetr Gynaecol 2005; 106:671 -75
64. Lean ME. Obesity and cardiovascular disease: the waisted years. British Journal of Cardiology 1999; 6: 269–73.
65. Hess PE, Pratt SD, Lucas TP et al. Predictors of breakthrough pain during labor epidural analgesia. Anesth Analg 2001; 93: 414–8.
66. Howell CJ. Epidural versus non-epidural analgesia for pain relief in labour (Cochrane Review). In: The Cochrane Library, Issue 4. Chichester: John Wiley & Sons, Ltd., 2004.
67. Von Ungern-Sternberg BS et al, The effect of epidural analgesia in labour on maternal respiratory function. Anaesthesia 2004; 59: 350–3.
68. Cascio M, et al. Labour analgesia with intrathecal fentanyl decreases maternal stress. Canadian Journal of Anaesthesia 1997; 44: 605–9.
69. Patel J. Anaesthesia for LSCS in a morbidly obese patient. Anaesthesia and Intensive Care 1999; 27: 216–9.
70. Chetty V et al - Difficult spinal anaesthesia for caesarean section in two obese pregnant patients. West African Journal of Medicine 2001; 20: 274–6.
71. Jordan H, Perlow MD, Mark A, Morgan MD. Massive maternal obesity and perioperative cesarean morbidity. American Journal of Obstetrics and Gynecology 1994; 170: 560–5.
72. Vincent RD, Chestnut DH. Which position is more comfortable for the parturient during identification of the epidural space? International Journal of Obstetric Anesthesia 1991; 1: 9–11.
73. Hamza J et al Parturient's posture during epidural puncture affects the distance from skin to epidural space. Journal of Clinical Anesthesia 1995; 7: 1–4.
74. Robinson HE, O'Connell CM etall: Maternal outcomes in pregnancies complicated by obesity. Obstet Gynecol 2005; 106: 1357 - 64
75. Manuel C Vallejo – Anaesthetic management of the morbidly obese parturient; Current opinion in Anaesthesiology 2007, 20: 175 - 180
76. Clinkscales CP et all An observational study of the relationship between lumbar epidural space depth and BMI in Michigan parturients IJOA 2007;16:323-7
77. Watts RW. The influence of obesity on the relationship between body mass index and the distance to the epidural space from the skin. Anaesthesia and Intensive Care 1993; 21: 309–10.
78. Wallace DH, Currie JM. Indirect sonographic guidance for epidural anesthesia in obese pregnant patients. Regional Anesthesia 1992; 17: 233–6.
79. Grau T, et Ultrasound imaging improves learning curves in obstetric epidural anesthesia: a preliminary study. Canadian Journal of Anesthesia 2003; 50: 1047–50.
80. Melzack R- Severity of labour pain : influence of physical as w ell psychological variables Can Med Asso J 1984; 130:579 -584
81. Hawkins JL. Maternal mortality: anesthetic implications. Int Anesthesiol Clin 2002; 40:1–11.
82. Lewis G (ed). Confidential Enquiries into Maternal and Child Health. Why Mothers Die. The Sixth Report of the United Kingdom Confidential Enquiries into Maternal Deaths in the United Kingdom. London. RCOG Press. 2004. www.cemach.org.uk
83. Rawal N, Holmstrom B, Crowhurst JA et al. The combined spinal–epidural technique. Anesth Clin North Am 2000; 18: 267–95.
84. Kuczkowski KM, Benumof JL. Repeat Caesarean section in a morbidly obese parturient: a new anaesthetic option. Acta Anaesthesiologica Scandinavia 2002: 46: 753 – 4
85. van de Velde M, Teunkens A, Hanssens M et al. Post dural puncture headache following combined spinal epidural or epidural anesthesia in obstetric patients. Anaesth Intensive Care 2001; 29: 595–9.
86. Pan PH, Bogard TD, Owen MD. Incidence and characteristics of failures in obstetric neuraxial analgesia and anesthesia: a retrospective analysis of 19,259 deliveries. Int J Obstet Anesth 2004; 13: 227–33.
87. Norris MC. Are combined spinal–epidural catheters reliable? Int J Obstet Anesth 2000; 9: 3–6.
88. Eappen S, Blinn A, Segal S. Incidence of epidural catheter replacement in parturients: a retrospective chart review. Int J Obstet Anesth 1998;7: 220–5.
89. Coker LL Continuous spinal anaesthesia for Caesarean section for a morbidly obese patient: a case report. AANA J 2002;70: 189 -192
90. Faure E et al Incidence of postdural puncture headache in morbidly obese parturients. Reg Anesth 1994; 19: 361-363
91. Collins JS, Lemmens HJ, Brodsky JB et al. Laryngoscopy and morbid obesity: a comparison of the 'sniff' and 'ramped' positions. Obes Surg 2004; 14: 1171–5.
92. Baraka AS, Taha SK, Aouad MT et al. Preoxygenation – comparison of maximal breathing and tidal volume breathing techniques. Anesthesiology 1999; 91: 612–6.
93. Bray GA (1985) complications of obesity. Ann Intern Med 103:1052 – 1062
94. Michota F, Merli G. Anticoagulation in special patient populations: are special dosing considerations required? Clev Clin J Med 2005; 72 (Suppl. 1): S37–42.