Successful delivery in a morbidly obese patient after failed intubation and regional technique

Editor—We read with interest the letter describing a Caesarean section under local anaesthetic infiltration in a patient with a BMI of 49 after failed intubation and subsequent failed neuraxial blockade.1 Although successful, we cannot agree with the authors that local infiltration is an appropriate de novo technique to carry out a Caesarean section in the UK in any patient, but particularly in a morbidly obese woman.

The most recent report into maternal mortality in the UK 2003–05, ‘Saving mothers’ lives’, highlights the risks associated with obesity in the obstetric population.2 Ideally, such patients should be referred for anaesthetic review antenatally. On admission to the labour ward, they should be reviewed by the duty anaesthetist and an anaesthetic management plan should be made in case operative delivery is required. Regional anaesthesia is preferred in obesity but requires a skilled anaesthetist and appropriate equipment. A back-up plan to convert to general anaesthesia must always be considered and advanced airway skills, including awake fibreoptic, should be available.

If the need for a Category 1 Caesarean section arises in such a woman, the duty obstetrician, midwife, and anaesthetist must be aware that there may be a delay in establishing anaesthesia and senior help may be required. During this time, attention must be paid to optimizing fetal condition using intraterine resuscitative measures.3 In most Category 1 Caesarean sections, there is no evidence that the most rapid anaesthetic technique (general anaesthesia with rapid sequence induction) improves neonatal outcomes, as indicators of fetal distress lack specificity.4

In general, it is held that the mother’s life must not be deliberately endangered in deference to the baby. Embarking on a Caesarean section under local infiltration commits the mother to the dangers of abdominal surgery with a high risk of anaesthetic failure. In such circumstances, manipulation of viscera is likely to cause pain, nausea, vomiting, and loss of patient co-operation. Morbid obesity increases these risks. As the authors had already established that they were in a ‘can’t intubate/can’t ventilate’ scenario, we would ask what their ‘Plan B’ would have been had complications arisen.

We would suggest that local infiltration should be reserved as a possible mode of supplementing inadequate regional blocks in some circumstances and to provide post-operative analgesia after general anaesthesia. It is not, as the authors suggest, an appropriate ‘life-saving technique in emergency Caesarean section in morbidly obese patients’.

M. J. Scrutton*
M. Kinsella
I. Gardner
N. Wharton
Bristol, UK
*E-mail: mark.scrutton@ubht.nhs.uk

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situation and were keen to go ahead with it, and to have delivery of a live baby. As we understand, there is no evidence that Caesarean section under local anaesthesia has an increased incidence of mortality than any other form of anaesthesia.

Finally, we agree with Dr Scrutton, in that normally, local anaesthesia should be supplementary to other forms of anaesthesia. This situation was extraordinary. We reiterate, we do not recommend local anaesthesia as a sole technique for Caesarean section, but would like to share our experience of a successful delivery under local anaesthetic infiltration. In our case with no complications to either baby or mother.

S. Patil*
P. Sinha
S. Krishnan
Swansea, UK
*E-mail: drsangy@hotmail.com

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Tension enterothorax

Editor—Diaphragmatic herniation of abdominal contents is a recognized complication of transhiatal surgery. We report a case of late tension enterothorax after an Ivor Lewis oesophagectomy complicated and exacerbated by massive haemorrhage into the herniated bowel.

A 61-yr-old man presented with sudden onset severe abdominal pain, initially epigastric in origin but rapidly spreading to the whole abdomen. He had an Ivor Lewis oesophagectomy 10 months previously for adenocarcinoma. Significant examination findings were tachycardia (98 beats min\(^{-1}\)), hypertension 142/114 mm Hg, and capillary refill time 5 s. Initial investigations revealed haemoglobin (Hb) of 14.6 g dl\(^{-1}\), white cell count 26.3 \(\times\) 10\(^{-9}\) litre\(^{-1}\), and a metabolic acidosis with pH 7.3, base deficit 4.9 mmol litre\(^{-1}\), and lactate 2.9 mmol litre\(^{-1}\). Chest X-ray showed some shadowing in the left lower and midzones. Abdominal X-ray was unremarkable. Before further imaging with an abdominal CT scan, his condition deteriorated and his pulse rose to 110 beats min\(^{-1}\) and arterial pressure dropped to 80/40 mm Hg. His Hb was noted to have decreased to 6.8 g dl\(^{-1}\). His neck veins had become distended and a central venous catheter was easily inserted. It was thought at this stage that he was having a massive gastrointestinal haemorrhage, transfusion of blood commenced, and upper gastrointestinal endoscopy performed which demonstrated absence of bleeding down to the third part of the duodenum. By the end of the endoscopy, his Hb had dropped further to 5.8 g dl\(^{-1}\), despite transfusion of 4 units of packed red cells. A left upper quadrant mass was now apparent and a ruptured spleen or splenic artery aneurysm was suspected.

He was transferred to theatre for a laparotomy where anaesthesia was induced with a modified rapid sequence induction with propofol 20 mg, alfentanil 500 \(\mu\)g, and rocuronium 50 mg and the trachea intubated. His systolic pressure fell to 40 mm Hg requiring an initial bolus of adrenaline 500 \(\mu\)g followed by an infusion of 0.5 \(\mu\)g kg\(^{-1}\) min\(^{-1}\). Gelofusine, blood, and fresh frozen plasma infusions, guided by laboratory results, were given with the adrenaline to maintain a mean arterial pressure above 65 mm Hg. Laparotomy revealed no intra-abdominal bleeding but a bulging left hemidiaphragm through which blood was aspirated. A new diagnosis of ruptured thoracic aneurysm was made and the patient’s abdomen was closed and he was transferred for a thoracic CT scan with contrast which revealed a tension enterothorax with mediastinal shift to the right side (Fig. 1). Thoracotomy revealed hugely dilated loops of infarcted small bowel with associated haemorrhage. The herniated bowel was reduced and infarcted bowel resected. The adrenaline requirement immediately decreased to 0.1 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) upon opening the chest. Ventilation was maintained via a single lumen tube throughout the whole procedure and anaesthesia maintained with sevoflurane in oxygen to maintain

Fig 1 CT scan of the thorax demonstrating herniated bowel loops and mediastinal shift.