This article describes emergencies with which an anaesthetist may become involved on the obstetric unit. Traditionally, over half the anaesthetics administered on the delivery suite were considered to be emergencies because they were unplanned. Attempts have been made to subdivide unplanned procedures, in line with the Confidential Enquiries into Perioperative Deaths (CEPOD).

Lucas and colleagues considered five methods of classification: a visual analogue scale, a numerical rating scale, suitable anaesthetic technique, maximum time-to-delivery and a clinical classification. The clinical classification (Table 1) performed best. It was further assessed prospectively to classify the urgency of over 400 Caesarean sections (CS) and produced agreement between anaesthetists and obstetricians in 90% of cases. This classification has been adopted by the Confidential Enquiries into Maternal Deaths (CEMD) and the Confidential Enquiries into Stillbirths and Deaths in Infancy (CESDI). At a national level, it should assist in comparative audit. It is hoped that this classification will aid communication on the delivery suite, help to prioritise work and allocation of manpower, minimise panic and avoid unnecessary general anaesthesia (GA).

Obstetric emergencies

Using the new classification, the clinical diagnoses in the immediate category fall into three groups: (i) prolapsed cord (especially with cord compression); (ii) profound and persistent fetal distress; and (iii) haemorrhage.

Cord prolapse

Cord prolapse is an uncommon event associated with small infants, premature labour and breech presentation. The cord is usually detected in the vagina but may be occult. Delivery is always urgent but needs to be immediate in cases with cord compression.

Fetal distress

The detection of other forms of fetal distress is fraught with difficulties. Fetal heart rate monitoring gives both false positive and negative results. However, if the fetal heart rate is consistently 70 or below and/or the fetal scalp pH is < 7 then immediate delivery is advised. Prior to immediate or urgent delivery, it is important to optimise the intrauterine environment. Placental perfusion can be improved by relieving aorto-caval compression through repositioning the mother in the lateral or knee elbow position and treating maternal hypotension with fluids and ephedrine. Exogenous oxytocic should be stopped and consideration given to the use of a tocolytic, as intense, frequent and or prolonged uterine contraction may be contributing to fetal distress by reducing uterine blood flow. Supplementary maternal oxygen may improve oxygenation of the distressed fetus in the presence of reduced fetal blood flow.

Major haemorrhage

Major haemorrhage can be defined as the acute loss of > 40% of blood volume or > 2 litre at term. There may be problems detecting the loss, as early signs of bleeding mimic the normal physiological changes of pregnancy, e.g. dilutional anaemia,

Table 1 Classification for the urgency of Caesarean section

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
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<tbody>
<tr>
<td>1. Immediate threat to the life of the woman or fetus</td>
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<tr>
<td>2. Maternal or fetal compromise which is not immediately life-threatening</td>
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<tr>
<td>3. Needing early delivery but no maternal or fetal compromise</td>
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<td>4. At a time to suit the woman and the maternity team</td>
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Key points

Fetal distress: improve oxygen delivery with fluids, vasoconstrictors, lateral tilt, supplementary oxygen and cessation of uterine stimulate on route to prompt delivery.

Maternal haemorrhage: record and replace losses, treat underlying cause, deliver the baby and consider hysterectomy or uterine artery embolotomy.

Maternal collapse: assess and treat ABCs, early intubation, use a lateral tilt during external cardiac massage and prompt delivery of the baby.

Eclampsia and PET: treat fits, control blood pressure, check clotting and deliver the baby.

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increased pulse and respiratory rate. The loss may be concealed or underestimated on drapes or in amniotic fluid.

There are problems associated with pregnancy, which can make the treatment of haemorrhage more difficult. Antenatally, haemorrhage will exacerbate supine hypotension and may impair uteroplacental blood flow. Pregnant women have an increased risk of acid aspiration and disseminated intravascular coagulation (DIC). In addition, the haemorrhage may be severe. It is the cause of 11% of direct maternal deaths in the UK and 20–25% of perinatal deaths. Just under half of these maternal deaths are associated with DIC. The maternal deaths are equally divided between the antenatal and postnatal period. Antenatally, the major causes of haemorrhage are placenta praevia and abruption. Caesarean section and uterine rupture are the commonest causes. Postpartum atonic uterus, retained placenta and trauma to the birth canal are all capable of giving rise to significant haemorrhage.

Placental abruption occurs in 1–1.5% of pregnancies, is responsible for 20–25% of antepartum haemorrhage and gives rise to 15–20% of perinatal mortality. It can be classified according to the amount lost and the losses may be partially or completely concealed. In severe abruption, where the baby is dead and > 2.5 litre of blood may have been lost, there is a 30% risk of DIC and vaginal delivery is preferred. If the baby is still alive then probably < 2 litre will have been lost. In these cases, DIC is much less likely (10% risk) and urgent CS is essential. Placental abruption is one of the many triggers of DIC associated with pregnancy. Clinically, DIC may present as anything from a minor chemical abnormality to haemostatic failure. The management of DIC involves maintaining circulation, sending blood for analysis of the problem and cross matching and transfusion of blood, platelets and clotting factors as necessary. However, success of the treatment depends on removal of the trigger.

Placenta praevia is slightly less common, occurring in 0.5–1% of pregnancies, and is responsible for a similar percentage of antepartum haemorrhage (15–20%) but only causes 5% of perinatal mortality. The classification relating the placenta to the cervical os and the risk of vaginal delivery is unhelpful because most women with placenta praevia are delivered by CS. There is an increased risk of haemorrhage due to incision of large vessels or the placenta, a poorly formed lower segment or a morbidly adherent placenta. The risk of the latter increases with the number of previous CS (risk > 25% if more than one previous CS). There is debate about the best way to anaesthetise women with placenta praevia. However, the combination of hypovolaemia, active bleeding, previous CS and a grade IV anterior placenta is an indication for GA in many units.

The management of major haemorrhage involves a team and every unit should have up-to-date guidelines. There should be regular opportunities for obstetric unit staff to practise their response. The obstetric management will involve delivery of the fetus, emptying the uterus and getting the uterus to contract. The latter may be assisted by mechanical stimulation, emptying the bladder, removing uterine relaxants and giving uterine stimulants. If these measures fail, a hysterectomy with or without arterial obliteration may be required.

**Maternal collapse**

Maternal collapse is an event for which the anaesthetist is called to give emergency care based on assessment and treatment of airway, breathing and circulation (ABC), while the diagnosis and subsequent management is determined.

**Cardiac arrest and heart disease**

Ischaemic heart disease is becoming more prevalent on labour wards with the increasing age of parturients. Cardiac arrest can occur as a result of primary cardiac pathology (congenital or acquired, e.g. cardiomyopathy) or secondarily to other causes of maternal collapse. Management is stabilisation of the mother prior to urgent delivery, except in a cardiac arrest situation where delivery is recommended after 5 min of unsuccessful cardiopulmonary resuscitation. In pregnancy, it is wise to intubate early (risk of aspiration) and use lateral tilt (prevent aorto-caval occlusion).

**Embolism**

Over 25% of maternal deaths in the UK are caused by thrombotic, amniotic or air emboli. Thromboemboli usually follow venous stasis in the lower limb and pelvic veins. Pregnancy is a hypercoaguuble state but other risk factors include immobility, smoking, surgery, hypovolaemia, hypotension, hypothermia, malignancy and inherited disorders of the fibrinolytic system. Amniotic fluid emboli are less common and occur as a result of direct communication between uterine veins and amniotic fluid. Risk factors include precipitant labour or delivery, a large baby, increased maternal age or parity, placenta praevia and use of uterine stimulants. Air emboli are rare but can occur during removal of a retained placenta if air enters the open uteroplacental veins. Presentation may include dyspnoea with chest pain and occasionally haemoptysis. Examination usually reveals cyanosis, hypotension, tachycardia, poor peripheral perfusion and raised jugular venous pressure. In severe cases, convulsions, coma and cardiac arrest may be the presenting features. The patient needs resuscitation and treatment for the underlying condition.
Obstetric emergencies and the anaesthetist

Convulsions

Convulsions are often caused by eclampsia. However, there are several other causes including pre-existing epilepsy, poorly controlled diabetes or local anaesthetic toxicity. Systemic or central nervous system infection and intracranial tumours can cause fits. Depletion of platelets and/or clotting factors through haemorrhage, idiopathic thrombocytopenia or DIC can lead to intracranial haemorrhage. Central venous sinus thrombosis is seen in pregnancy and can cause fits. Other pathological conditions (e.g. thrombotic thrombocytopenic purpura [TTP]), can cause convulsions through cerebral infarction.

Every effort to control convulsions must be made prior to CS. The anaesthetic induction agent of choice is the anticonvulsant thiopental.

Sepsis

Neutrophilia is evident by the third trimester, peaking during labour. However, cell mediated immunity is depressed. Infection often originates from the urinary-genital tract but may be respiratory, postoperative or as a complication of spinal/epidural anaesthesia. Severe sepsis can quickly result in collapse. Tissues should be sent for culture and sensitivity prior to starting appropriate antibiotic therapy. The most common organism responsible for life threatening obstetric infections in the UK is a beta-haemolytic streptococcus and first choice antibiotics are a penicillin and aminoglycoside. Resuscitation and stabilisation are the mainstay of management prior to delivery.

Uterine and cervical pathology

Uterine rupture usually occurs through a scar and can lead to a profoundly shocked patient. Immediate delivery is required. Uterine eversion can complicate a vaginal delivery and lead to maternal collapse. Anaesthetic input may be required to return the uterus to its correct position. Cervical problems relate to the vasovagal response where profound bradycardia and collapse can follow cervical manipulation or rapid dilatation. Fluid and parasympathetic antagonists may be required.

Pre-eclamptic toxaemia

In the absence of fetal distress or haemorrhage, severe pre-eclamptic toxaemia (PET) requires emergency medical rather than surgical management. Hypertension must be treated and an assessment made of maternal haemoglobin and platelet count before anaesthesia and surgery is commenced. Emptying of the uterus leads to resolution of the disease but rapid sequence induction in the presence of uncontrolled hypertension can result in maternal complications including intracranial haemorrhage, pulmonary oedema and myocardial infarction.

The anaesthetist is part of the team assessing and treating the mother with PET. It is a multisystem disease with very variable presentation. Indicators of severe disease include eclampsia or severe hypertension (systolic > 170 mmHg, diastolic > 110 mmHg) with proteinuria or moderate hypertension (systolic > 140 mmHg, diastolic > 90 mmHg) with proteinuria and other symptoms or signs of decompensation (headache, visual disturbance, papilloedema, epigastric pain, liver tenderness, clonus, platelet count < 100 × 10^9 litre^-1, alanine amino transferase > 50 IU litre^-1). The mother and fetus need to be closely monitored with one-to-one midwifery care. After initial assessment, records should be kept of physiological variables, investigations and treatments. Intravenous access should be obtained but input limited to 80 ml h^-1 (including drug infusions).

Hypotension associated with regional blockade should be treated with small boluses of ephedrine. A number of drugs have been used for the control of blood pressure in PET. The first line drug in our region is oral labetalol. If there is no response to oral therapy or if this route is unavailable, labetalol is given as an intravenous bolus followed by an infusion. If labetalol is contraindicated (e.g. asthma) or fails to control the blood pressure, then nifedipine 10 mg is given orally and repeated 6-hourly. Nifedipine is not licensed for use in pregnancy and experience with its use is limited. The fall in blood pressure may be rapid and profound and particular care should be taken when administering nifedipine with magnesium sulphate.

A full description of the management of PET is beyond the scope of this article but it will be reviewed in some detail in a future issue of the journal.

Anaesthetic emergencies

A review of anaesthesia for CS has been published recently in this journal (see key references). We consider the emergency situations which may arise under these circumstances.

General anaesthesia

General anaesthesia is now used in a minority of CS but is necessary in situations where regional anaesthesia (RA) is contraindicated (e.g. patient refusal, deranged clotting, infection, hypovolaemia, fixed cardiac output) or where RA cannot be performed (e.g. prolapsed cord with compression). Failed intubation is an emergency associated with GA; it is estimated to occur in 1 in 300 obstetric cases compared with 1 in 2230 in the non-obstetric population (Table 2). Hypoxaemia occurs more rapidly due to...
reduced functional residual capacity and higher basal metabolic rate. The risk of aspiration is also greater.

**Regional anaesthesia**

Regional anaesthesia has advantages for obstetric operative procedures including maintenance of airway reflexes and spontaneous ventilation. The addition of an opioid to the cerebrospinal fluid or epidural space can provide postoperative analgesia. Single shot spinal anaesthesia is the most common regional technique and, in experienced hands, is simple and quick to perform. The more technically challenging spinal catheter can provide a slower onset, more controlled spinal block. Previously sited epidural catheters can be ‘topped up’ to provide anaesthesia. A combined spinal-epidural technique, although taking longer to site, has the advantage of immediate anaesthesia with the reassurance of the ability to ‘top up’ the block. Local anaesthetic can also be infiltrated directly by the surgeon and is successfully used in many developing countries.

Adverse effects from regional anaesthesia include failed block, urinary retention, headache and rarely infection. Emergency situations are hypotension, high block, total spinal and local anaesthetic toxicity.

**Hypotension**

A fall in blood pressure results from the sympathetic blockade and subsequent vasodilatation. If severe and untreated, low oxygen delivery will occur to maternal tissues, including the placenta and the baby. Intravenous access must be secured prior to oxygen delivery will occur to maternal tissues, including the placenta and the baby. Intravenous access must be secured prior to any regional technique with fluids and vasoconstrictors to hand. Ephedrine is first choice because it maintains fetal blood supply.

**High block and total spinal**

High block is the result of extensive spread of local anaesthetic and usually presents as hypotension, nausea, vomiting, nasal congestion, bradycardia and loss of responsiveness. The situation becomes an emergency if respiration or airway reflexes are compromised. For respiratory failure to occur, cervical nerves 3, 4 and 5 become blocked and prevent diaphragmatic movement. Cranial nerves would have to be affected before airway reflexes are lost; this occurs more commonly with total spinal block. The latter can occur: (i) as a progression of a high block; (ii) after movement of the patient too soon after administration of a spinal anaesthetic before the local anaesthetic has become ‘fixed’ to the tissues; or (iii) after administration of an epidural dose into the CSF. Total spinal usually occurs within 5–10 min of inappropriate movement or injection. Slower and less complete high blocks are seen after subdural administration of epidural doses. Immediate management is to gain control and protection of airway and to ventilate the lungs. Fluids and vasoconstrictors will be needed to maintain an adequate blood pressure. Verbal re-assurance should be given to the mother while an anaesthetic or sedative is given as she may be aware.

**Local anaesthetic toxicity**

This results from accidental intravascular injection or absolute overdosage. Plasma concentrations are dependent on the dose given, speed of absorption and rate of distribution and metabolism. Symptoms of toxicity are firstly circumoral numbness and tingling, tinnitus, feeling faint or drowsy, followed by convulsions and loss of consciousness. Coma and apnoea are followed by cardiovascular collapse as myocardial depression and vasodilatation occur. Management is again ABC with the addition of anticonvulsants to control seizures and fluid, inotropes and vasoconstrictors to maintain circulation.