CEMACH 2003–5 Saving Mothers’ Lives: lessons for anaesthetists

Huda Al-Foudri FCARCSI
Euan Kevelighan FRCOG DipMedEd
Sue Catling BA (Cantab) MB BS FRCA

Key points

Systolic hypertension ≥ 160 mm Hg should be urgently and effectively treated.

Placenta percreta is an increasing cause of major obstetric haemorrhage. Women who have had a previous Caesarean section must have placental localization.

Widespread adoption of modified early obstetric warning scoring charts may help earlier recognition of critical illness.

Regular training is required in management of maternal collapse.

Obese parturients require expert management and monitoring—fatal airway complications can occur even with regional techniques.

The Confidential Enquiry into Maternal and Child Health (CEMACH) reviews every death in the UK occurring during pregnancy and up to 42 days post-partum. Originally titled Confidential Enquiries into Maternal Deaths (CEMD), this series of Reports is the longest running audit in the world. It covers every triennium since 1952, with sequential Reports having established the leading causes of maternal mortality and recommended change where appropriate. Up to 1985, only deaths in England and Wales were considered; thereafter, the whole UK was included.

The maternal mortality rate (MMR) is defined as the number of maternal deaths per 100,000 maternities. This has markedly reduced over the last 50 yr. In 1952–4, there were 1094 maternal deaths (MMR 53.29), whereas the latest Report 2003–5 records 295 deaths (MMR 13.95) of which 132 were direct and 163 were indirect. However, most of this dramatic improvement in MMR occurred early and was mainly attributable to advances in public health and medicine (Fig. 1).

Unfortunately, if we compare total deaths (223) and MMR (9.83) for the 1985–7 Report with those in the current Report 2003–5, we see that over the last 20 yr, there has been an increase in both total deaths and MMR.

Suggested explanations for the failure of MMR to decline since the last (2000–2) CEMACH Report include increasing maternal age at delivery, obesity, poor overall health status, difficulty in accessing maternity care, and the increase in immigrant mothers. One in four mothers now delivering in the UK has been born outside the UK. Whether these factors can explain the increase in MMR since 1985 is not known. In addition, the Scottish morbidity data indicate that for every maternal death, there are 60 ‘near-misses’ (rate of severe morbidity 5.3/1000 maternities).

CEMACH historically divides maternal deaths into direct deaths from conditions directly related to pregnancy and indirect deaths from conditions unrelated to pregnancy but exacerbated by it. Deaths occurring during pregnancy or within 6 weeks of birth, which were not due to or affected by pregnancy are called coincidental deaths. Deaths from any cause that occurred between 6 weeks and 1 yr after delivery are termed late deaths. Most late deaths are unrelated to pregnancy and therefore termed late coincidental deaths. This complex structure sometimes obscures causality. A ‘late’ death beyond the arbitrary 42-day post-natal period is often not counted in the quoted figures, even though the death is clearly attributable to the initial event after the patient has survived for a protracted period on the intensive therapy unit (ITU). Figure 2 therefore shows the less familiar—but more informative—leading causes of maternal deaths as direct/indirect plus relevant late deaths, with the proportion receiving major substandard care (defined as treatment—or lack of—which contributed significantly to the death of the mother, and where different treatment may have altered the outcome).

Anaesthesia as a continuing cause of maternal death

Anaesthesia was the seventh most common direct cause of maternal death in this Report and the ninth most common overall (Fig. 2). One hundred and fifty of the 295 patients who died received an anaesthetic and six of these deaths were directly caused by fatally substandard care in the management of the anaesthetic (MMR 0.28). In a further 31 cases, the anaesthetic care was considered to be poor. The Report highlights problems of obesity,
inappropriate postoperative respiratory monitoring, and drug administration errors.

The six anaesthetic deaths included four obese patients, one drug error (i.v. bupivacaine), and one haemothorax secondary to central venous pressure (CVP) line insertion. There were no deaths caused by unrecognized oesophageal intubation.

The causes of death in the above patients were:

(i) Failure of reintubation for severe bronchospasm post-extubation after laparoscopic surgery for ectopic pregnancy in an asthmatic patient.
(ii) Cardiac arrest secondary to ventilatory depression after a large dose of fentanyl before extubation. There was a delay in the arrival of the anaesthetist who had left the patient with the recovery staff after extubation.
(iii) Postoperative respiratory failure secondary to severe bronchospasm in a patient who underwent a Caesarean section (CS) using spinal anaesthesia. The patient was later returned to the ward with inadequate monitoring and treatment.

These three patients died from fatal airway complications related to their obesity and inadequate postoperative monitoring. It is important to note that this can occur even when regional anaesthesia is used. The incidence of failed tracheal intubation in the obstetric population is much greater than in the general population (one in 280 compared with one in 2230). This incidence increases dramatically in the morbidly obese parturient and figures as high as 33% have been quoted in the literature. Tracheal extubation is also an important skill to acquire and is currently part of the initial test of competence in the CCT syllabus.

(iv) Cardiac arrest in a septic patient who underwent a general anaesthetic a few weeks post-partum for removal of a septic focus from the kidney. The cause of death was unknown and one possibility was arrhythmia secondary to electrolyte disturbances.

(v) The maternal death due to drug error was caused by 150 ml of 0.1% bupivacaine being accidentally administered via an i.v. line instead of the epidural catheter, resulting in a ventricular fibrillation arrest from which the patient could not be resuscitated. The NPSA has published guidelines in order to reduce the risk of such errors. Treatment with intralipid is now recommended for the treatment of local anaesthetic toxicity; it should be immediately available in all obstetric units. An initial i.v. bolus of intralipid 20% of 1.5 ml kg$^{-1}$ (100 ml bolus for a 70 kg patient) can be repeated twice every 5 min, followed by an i.v. infusion of 0.25–0.5 ml kg$^{-1}$ min$^{-1}$ (400 ml over 10–20 min).

(vi) The sixth anaesthetic death was caused by a haemothorax secondary to repeated attempts at CVP line insertion in a patient with pectus excavatum and fulminant pre-eclampsia. This is the third death from a CVP line complication since the introduction of CEMACH. The current Report encourages CVP monitoring when there is clear benefit, and CVP insertion should take due account of the potential risks. It is safer to avoid central line insertion in patients with a higher risk of complications, for example, abnormal anatomy, obesity, and coagulopathy. Where technical difficulty is not anticipated and CVP monitoring is thought to be justified, the risk of complications may be reduced by the use of ultrasound guidance.

The top 10 recommendations: lessons for anaesthetists

For the first time, the current Report identifies 10 priority recommendations for improving care and encourages their introduction and implementation with audit (Table 1). Several of these recommendations are public health and educational issues (recommendations 1–4) and are not within the control of
160 mm Hg should be treated. This represents improved care. There has been no improvement in the past 20 yr. Ten of those dying from intracranial haemorrhage secondary to pressor response to intubation and extubation is re-iterated, with a welcome statement that anaesthetists should ‘be allowed’ time to achieve this, even in the face of a grade 1 CS. If the patient has laboured with an epidural, this can be topped up if time allows, otherwise spinal anaesthesia remains the technique of choice in pre-eclampsia (unless the patient has abnormal coagulation, e.g. platelet count \( < 80–100 \times 10^9 \) depending on locally agreed protocols), and general anaesthesia should only be used if regional block is contraindicated. The risk of producing a spinal haematoma in a coagulopathic patient must be balanced against the risk of intracerebral haemorrhage secondary to pressor response to laryngoscopy.

Care should be taken with the use of oxytocics in pre-eclampsia. Syntocinon should be diluted and given by slow i.v. bolus injection. Both ergometrine and syntometrine are contraindicated in hypertensive patients. However, the clinical balance between preventing bleeding from an atomic uterus and exacerbating hypertension with uterotonics can be a difficult decision and should be made by a senior anaesthetist (see 6b below).

### CEMACH recommendation 6a: ‘indications for CS should be appropriate’

The Report states: ‘Whilst recognising that for some mothers and/or their babies, CS may be the safest mode of delivery, mothers must be advised that caesarean section is not a risk-free procedure and can cause problems in current and future pregnancies’. Patients should have the opportunity to discuss labour analgesia with an anaesthetist antenatally, either in classes or an anaesthetic clinic, so that fear of pain in labour does not inappropriately influence the patient’s decision for Caesarean.

### CEMACH recommendation 6b: ‘any patient with a previous CS should have placental localization’

In the management of haemorrhage, the Report states that ‘In particular, there were questions concerning the most appropriate management of women with placenta percreta, a problem likely to become more prevalent due to its emerging relationship with previous caesarean section scars. There were also apparent failures in recognising the signs and symptoms of intra-abdominal bleeding especially after caesarean section. Lastly, ergometrine often seems to have been forgotten as a useful oxytocic drug’.

### Haemorrhage

Seventeen women died from haemorrhage including genital tract trauma (MMR 0.8), compared with 16 in the 1985–7 Report (MMR 0.7). There has been no improvement in the past 20 yr. Ten of those who died in this triennium received substandard care (59%), mainly

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### Table 1: Top 10 CEMACH recommendations

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<tr>
<th>CEMACH 2003–5: Top 10 recommendations</th>
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<tbody>
<tr>
<td>1. Pre-conception counselling for patients with poor medical or mental health</td>
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<td>2. Providing easy access to maternity services with the first booking completed by week 12</td>
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<td>3. Late bookers (( &gt; 12 ) weeks) should be given priority on accessing maternity services</td>
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<td>4. Full medical history and examination of all migrant women at booking</td>
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<tr>
<td>5. Systolic hypertension ( \geq 160 ) mm Hg should be treated</td>
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<td>6. (a) Indications for CS should be appropriate and (b) any patient with a previous CS should have placental localization</td>
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<td>7. Constructive reflection by staff on any critical event or serious untoward incidents</td>
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<td>8. Ensuring regular training (obstetric skills and drills)</td>
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<td>9. Implementation of an early warning scoring system specific for obstetrics (MEOWS)</td>
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<td>10. Urgent guidelines are required for obesity, sepsis, and pain/bleeding in early pregnancy</td>
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anaesthetists. However, some recommendations (5 and 6b) give new guidelines in the familiar anaesthetic areas of pre-eclampsia and haemorrhage or highlight the need for new guidelines (10). Other recommendations (6a, 7, 8, and 9) invite anaesthetists to consider their team roles in training, reflective practice, and the timely recognition of serious maternal illness.

### CEMACH recommendation 5: pre-eclampsia: ‘systolic hypertension \( \geq 160 \) mm Hg should be treated’

The Report states: ‘In the current triennium the single most serious failing in the clinical care provided for mothers with pre-eclampsia was the inadequate treatment of their systolic hypertension’. There were 18 deaths from pre-eclampsia/eclampsia (MMR 0.85), 10 of these dying from intracranial haemorrhage associated with uncontrolled hypertension. Although this is a reduction from the 1985–1987 figures, there has been no reduction compared with the last two Reports. Substandard care is identified in eight cases and ‘remediable factors’ (‘areas where patient care might have been improved’) in 13. There is now clear evidence that systolic arterial pressure (SAP) of 160 mm Hg should be treated. This represents a new and important guideline, as previous recommendations have focused on mean AP of 125 mm Hg as the threshold for treatment. This change will need to be rapidly incorporated into local protocols and should be clearly identified as an action threshold on a modified early obstetric warning scoring chart (MEOWS, see below). Treatment should be instituted at lower levels of SAP if there are signs of rapid deterioration. Treatment of hypertension in pre-eclamptic toxemia includes oral treatment with labetalol or nifedipine in divided doses. If this fails to achieve an AP \( < 160 \) mm Hg systolic, then i.v. labetalol should be administered as a slow i.v. bolus followed by a labetalol infusion. If \( \beta \)-block is ineffective or contraindicated, then hydralazine as a slow i.v. bolus over 20 min may be used. Hydralazine is a vasodilator, so plasma volume expansion with a 250 ml colloid bolus is recommended. However, fluid loading in pre-eclampsia should always be controlled. Hydralazine has a slow onset of action, so the effect of each bolus should be thoroughly evaluated over 20 min in order to avoid over treatment or too rapid AP reduction. Hydralazine infusion can be utilized for maintenance. The advice to obtund the pressor response to intubation and extubation is re-iterated, with a welcome statement that anaesthetists should ‘be allowed’ time to achieve this, even in the face of a grade 1 CS. If the patient has laboured with an epidural, this can be topped up if time allows, otherwise spinal anaesthesia remains the technique of choice in pre-eclampsia (unless the patient has abnormal coagulation, e.g. platelet count \( < 80–100 \times 10^9 \) depending on locally agreed protocols), and general anaesthesia should only be used if regional block is contraindicated. The risk of producing a spinal haematoma in a coagulopathic patient must be balanced against the risk of intracerebral haemorrhage secondary to pressor response to laryngoscopy.

Care should be taken with the use of oxytocics in pre-eclampsia. Syntocinon should be diluted and given by slow i.v. bolus injection. Both ergometrine and syntometrine are contraindicated in hypertensive patients. However, the clinical balance between preventing bleeding from an atomic uterus and exacerbating hypertension with uterotonics can be a difficult decision and should be made by a senior anaesthetist (see 6b below).
due to failure to identify and manage intra-abdominal bleeding, uterine atony, and placenta percreta. In contrast, the Scottish morbidity data show that major haemorrhage accounted for 69% of the total cases of serious morbidity, with a rate of major obstetric haemorrhage of 3.66 per 1000 maternities. Suboptimal care was identified in only 3%, and most of the cases were well managed on the ward or high dependency unit (HDU). The critical care chapter emphasizes that recognition of critical physiological compromise such as that caused by major intra-abdominal bleeding can be assisted by the use of early warning charts (see below), and that resuscitation skills can be improved by training on appropriate courses [Management of Obstetric Emergencies and Trauma (MOET) and Advanced Life Support in Obstetrics (ALSO), see below] and the use of simulation scenarios. The importance of preventing dilutional coagulopathy in massive transusion is highlighted, with a suggestion that there may be a role for ‘whole blood’, or for the use of 1 U of fresh frozen plasma for each pack of red cells transfused. Bedside estimation of haemoglobin (Hb) concentration using the Haemocue device is valuable, and there may be a role for near-patient coagulation testing.

Anterior placenta praevia with a previous CS scar requires multidisciplinary consultant care, and the placental site must be determined with ultrasound or magnetic resonance imaging well in advance of planned delivery. Atonic uterus is common and can often be anticipated. Syntocinon 5 U (repeated if indicated) and ergometrine 500 μg given slowly i.v. should be the first choices to prevent and treat atony of the uterus. Additionally, a syntocinon infusion should be started immediately after delivery of the baby, with 40 U of syntocinon in 500 ml of saline to run i.v. over 4 h. Both CEMACH and the UK Obstetric Surveillance System emphasize that both ergometrine and syntocinon are being withheld inappropriately in obstetric haemorrhage. Since a previous CEMACH Report where a rapid i.v. 10 U bolus of syntocinon was associated with the death of a hypotensive patient, this drug is now given by slow i.v. 5 U bolus. Any decision to withhold uterotonic in obstetric haemorrhage due to uterine atony (e.g. if the patient is also pre-eclamptic/hypertensive) must therefore be very carefully considered and made at senior level. Second-line drugs to control haemorrhage unresponsive to oxytocin and ergometrine include synthetic prostaglandin analogues carboprost (15-methyl PGF2alpha) 250 μg i.m. repeated every 15 min to a maximum dose of 2 mg, and misoprostol (PGE1) 600–1000 μg p.r. Patients who refuse blood transfusion, such as Jehovah’s Witnesses, should be actively treated with haematinics to optimize their Hb and should be offered cell salvage where appropriate.

**CEMACH recommendation 8: ‘ensuring regular training (obstetric skills and drills)’**

The management of many of the patients who died was considered suboptimal due to poor communication skills, poor resuscitation skills, and care being provided by unsupervised and inexperienced trainees. The latter factor may be exacerbated by the introduction of shorter training programmes. Lack of experience can be mitigated by attendance at resuscitation courses such as ALSO and MOET. The Report states that all staff involved in the care of acute obstetric admissions should have current ALS or ILS certification. Regular ‘skills and drills’ practice sessions on the use of major haemorrhage protocols and simulation-based teamwork training are also useful. Simulation can provide practical training for rare conditions that are rarely encountered clinically and it has been shown to improve knowledge and clinical team performance. For example, in amniotic fluid embolism (AFE; incidence 1.8/100 000 maternities), survival largely depends on adequate and effective resuscitation. In this triennium, there were 19 (17 direct and two late) deaths (MMR 0.89), with seven patients receiving substandard care, mainly relating to inadequate resuscitation efforts from first responders.

The management of maternal cardiac arrest includes perimortem CS. This should be started within 4 min of the diagnosis of cardiac arrest and completed within 5 min using minimal equipment and the most appropriate technique. The number of perimortem CSs has almost doubled compared with the last Report. There has been previous misunderstanding about the role of perimortem CS—emptying the uterus relieves aorta-caval compression and increases venous return and thereby helps to restore cardiac output. It makes ventilation easier and permits cardiopulmonary resuscitation (CPR) in the supine position. It is part of the maternal resuscitation algorithm and is not performed to save the baby, the survival of which is a welcome bonus. In this Report, there were 52 perimortem CS and 20 babies survived. These findings indicate that with improved resuscitation techniques, more babies are surviving perimortem CSs, particularly where the women collapsed in an already well-staffed and equipped delivery room or operating theatre. However, they also highlight the very poor outcome for babies delivered in Emergency Departments, especially for women who arrive after having undergone CPR for a considerable length of time. The babies who survived were born to mothers who were near or at term, and who suffered a cardiac arrest while already undergoing active treatment in the Emergency Department.

Trainees must know their limitations and should not hesitate to call for help, which should be readily available. The management of patients should be multidisciplinary and teamwork should be encouraged.

**Recommendation 9: ‘implementation of an early warning scoring system specific for obstetrics (MEOWS)’**

The Report states: ‘In many cases, the early warning signs of impending maternal collapse went unrecognised’. The Report strongly recommends the introduction of an MEOWS system to aid the early detection of life-threatening illness such as haemorrhage, pre-eclampsia/eclampsia, AFE, and sepsis. In pregnancy, the increase in physiological reserve may mask the signs of critical
illness, resulting in a delay in diagnosis and appropriate management.

The obstetric modifications to the usual early warning chart parameters (ventilatory frequency, temperature, heart rate, SAP, mental response, urine output, and oxygen saturation) add specific obstetric indicators such as proteinuria, diastolic AP, and amniotic fluid or lochia consistency. Figure 3 shows an example used by Stirling Royal Infirmary, which was published in the current Report and can be adopted by Trusts. Such a chart should be introduced in all areas dealing with pregnant patients. It should be noted that while a carefully completed MEOWS chart can indicate deterioration requiring action, a robust mechanism to ensure that the appropriate and prompt action actually occurs is also required to alter the outcome. The rate of obstetric admission to ITU is less than 1 per 1000 obstetric admission, but fortunately, these patients have a high survival rate (96%).1

**Thrombosis/thromboembolism**

This is the third most common cause of maternal death, with a total of 45 deaths (41 direct and four late, MMR 2.12), with substandard care identified in 23 cases. Again, this is an increase compared with the 32 cases in 1985–7 (MMR 1.14), with predisposing factors such as obesity and increasing maternal age. The dose of low molecular weight heparin should be increased in the morbidly obese parturient, and current RCOG Clinical Green Top Guideline No. 3713 gives weight-specific dosage advice shown in Table 2.

**Sepsis**

Twenty-two deaths (including one in early pregnancy and three late deaths) occurred from genital tract sepsis in the current triennium (MMR 1.04), compared with nine deaths in the 1985–7 Report (MMR 0.4)—this is a substantial and worrying increase. Substandard care was identified in 15 cases. Maternal tachycardia, constant severe abdominal pain and tenderness are important early features of genital tract sepsis that should prompt urgent medical review. Pyrexia and raised white cell count are not always present. Anaesthetists are familiar with the diagnosis and management of septic shock and should be proactive in leading the labour ward team where necessary. If sepsis is suspected, regular frequent observations should be made and the use of the MEOWS chart is crucial (see recommendation 9 above). The Critical Care section of the CEMACH Report highlights the use of the Sepsis Resuscitation Care bundle.14 Risk factors for sepsis include obesity, cervical cerclage, gestational diabetes, wound haematoma, retained products, and impaired immunity.

**Lessons not included in the top 10 recommendations**

**Cardiac disease: the leading cause of maternal death**

Cardiac disease is now the leading cause of maternal death, with a total of 82 deaths (MMR 3.87) of which 23 (28%) had substandard care (Fig. 1). Interestingly, the top 10 recommendations do not include any comment on cardiac disease, other than migrant women should have a full medical history and examination at
Cardiac deaths include 16 myocardial infarcts (mostly associated with obesity, older age at childbirth, smoking, and poor diet), 12 peripartum cardiomyopathies (soon to be reclassified as direct maternal deaths), and nine thoracic aortic dissections.

CEMACH/CEMD Reports over the past 30 yr show that only a minority of cardiac deaths occur in women with known disease, (mainly congenital defects) and that the increase is entirely accounted for by women who either had identifiable risk factors at booking (BMI >30, diabetes, smoking, hypercholesterolaemia,
hypertension, and family history) or in which the problem arose entirely de novo during pregnancy. These latter two categories mainly include ischaemic heart disease, cardiomyopathy, aneurysm, and myocarditis. Patients with known disease will usually already be under the care of Grown Up Congenital Heart (GUCH) multidisciplinary teams and may require referral to a specialist centre for delivery depending on the exact lesion. Most repaired lesions, uncomplicated shunts, and mild valve disease represent a low risk (0.1–1.0% mortality), with the highest risk group comprising pulmonary hypertension, Marfan’s with aortic involvement, and significant aortic stenosis or ventricular dysfunction (5–30% mortality). Most cardiac patients are now managed by vaginal delivery, with CS usually reserved for appropriate obstetric indications. Labour requires effective pain relief with careful, slowly established low-dose epidural block and monitoring with ECG, pulse oximetry, and direct AP. Systemic vascular resistance should be maintained and vasodilators such as oxytocin should be avoided—diluted ergometrine may be preferable. Phenylephrine is the vasopressor of choice. Particular care should be taken to avoid bleeding, pulmonary oedema, arrhythmias, thromboembolism, air embolism with shunts, and bacterial endocarditis. Anaesthesia for CS may be regional or general, and the care with which each technique is administered is more important than the choice of technique in most cases. A clear understanding of the precise nature of the lesion, the presence or absence of pulmonary hypertension, and the degree of anticoagulation will guide the decision. Close observation on HDU/ITU should extend into the post-delivery period.

Conflict of interest
None declared.

References
4. Royal College of Anaesthetists. Initial Assessment of Competency. CCT in Anaesthesia II: Competency Based Basic Level Training and Assessment. 2007. Available from www.rcoa.ac.uk
Amniotic fluid embolism is one of the most catastrophic complications of pregnancy. Passage of amniotic fluid into the maternal circulation was first reported by Meyer\(^1\) in 1926, and the syndrome was first described by Steiner and Lushbaugh\(^2\) in 1941. However, the condition is exceedingly rare and the exact pathophysiology is still unknown. Common presenting symptoms include dyspnea, nonreassuring fetal status, hypotension, seizures and disseminated intravascular coagulation (DIC). Early recognition of amniotic fluid embolism is critical to a successful outcome. However, despite intensive efforts at resuscitation, outcomes are frequently poor for both infant and mother. Aggressive management with the use of recombinant factor VIIa (rFVIIa) and a ventricular assist device,\(^3\) inhaled nitric oxide,\(^4\) cardiopulmonary bypass,\(^5\) and intraaortic balloon pump with extracorporeal membrane oxygenation (ECMO)\(^6\) have recently been reported with successful outcomes.

To better understand the risk factors and clinical presentation of women with AFE, two registries were developed, one by Clark et al.\(^7\) in the United States and one by Tuffnell\(^8\) in the United Kingdom (UK). Entry criteria were the same in both registries and included: 1) acute hypotension or cardiac arrest, 2) acute hypoxia, 3) coagulopathy, and 4) onset during labor, cesarean delivery or dilation and evacuation or within 30 min of evacuation of the uterus. Both of these registries have limitations since they depend on self-reporting and limited numbers of patients were entered, 46 in the United States registry published in 1995 and 44 in the UK registry published in 2005.

### Incidence and Outcome

The true incidence of AFE is unknown but is estimated to occur between 1 in 8000 and 1 in 80,000 deliveries,\(^7\)\(^–\)\(^9\) with reported mortality rates in older reports as high as 60% even with aggressive and immediate treatment.\(^7\) Maternal morbidity is also high and only 15% of survivors may be neurologically intact. More recent data suggest a lower mortality rate can be achieved, 27% in a population-based study performed in 1999\(^10\) and 37% in the UK registry from 2005.\(^8\) It is unclear if the improved mortality rate is related to better critical care management or an artifact related to different reporting techniques yielding a larger denominator. Neonatal outcome is generally poor with a mortality rate of 20%–25% and, of the survivors, only 50% may be neurologically intact.\(^7\)\(^,\)\(^8\) There are no proven risk factors to the development of AFE and its onset cannot be predicted.

### Pathophysiology

The pathophysiology of AFE is poorly understood as human study is obviously limited. Experimental animal studies into the pathogenesis and treatment of AFE have produced mixed results. Some models have failed to reproduce the syndrome even with direct intravascular injection of amniotic fluid,\(^11\)\(^,\)\(^12\) and some have only been able to do so when the injected amniotic fluid was stained with meconium.\(^13\)\(^,\)\(^14\)
AFE, as a clinical syndrome, was first characterized by the presence of amniotic fluid debris in the maternal pulmonary circulation, and the amniotic fluid was thought to cause an embolic phenomenon. Steiner and Lushbaugh, in their original description of AFE, reported the presence of mucin, amorphous eosinophilic material and squamous cells in women with AFE, consistent with the presence of amniotic fluid. They hypothesized that amniotic fluid was forced into the maternal circulation during contractions leading to the embolic event. The theory was supported by analyzing the women in their series who developed AFE. Most cases of AFE occurred in multiparous women who delivered vaginally during a tumultuous or hyperstimulated labor. However, knowledge of uterine physiology and results of other studies have called this theory into question. During contractions, especially forceful ones, when uterine pressure increases above maternal venous pressure, maternal-placental exchange decreases or ceases, but certainly does not increase. Therefore, Clark et al. speculated that the least likely time for transfer of amniotic fluid is during tumultuous labor or during uterine tachysystole. Furthermore, Lee et al. demonstrated that fetal squamous cells can be found in the pulmonary circulation of women without clinical evidence of AFE, and other investigators could not reproduce the syndrome in two separate animal models, mini-pigs and monkeys, by injecting amniotic fluid directly into their circulation. Finally, Clark et al. found that 19% of women in their registry first manifested symptoms during cesarean delivery when there is no tumultuous labor.

Clark et al. recognized that the clinical course and hemodynamic changes of AFE were similar to patients with anaphylactic shock and proposed that AFE was more of an immunologic than embolic phenomena. Amniotic fluid contains many vasoactive and procoagulant substances, such as platelet activating factor, cytokines, bradykinin, thromboxane, leukotrienes and arachidonic acid, and entrance of even minute amounts of these substances into the maternal circulation could cause the syndrome. This would explain why fetal cells were not always found in women who suffered AFE. They further recommended changing the name of the syndrome from AFE “to the anaphylactoid syndrome of pregnancy.” In support of this “immunologic” theory is the finding that AFE seems to be more common in women carrying male fetuses, and these women are also at increased risk for Rh isoimmunization, another immunologic-based condition. Some have suggested that plasma tryptase levels, a mast cell enzyme, may be helpful in the diagnosis of AFE. Further support for an immune basis is that complement activation, another component of the immune response, may play a role in the pathogenesis of AFE. Specifically, C3 and C4 levels are markedly decreased in women with AFE.

### Table 1. Signs and Symptoms of Amniotic Fluid Embolism

<table>
<thead>
<tr>
<th>Signs or symptoms</th>
<th>Frequency</th>
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<tbody>
<tr>
<td>Hypotension</td>
<td>100%</td>
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<tr>
<td>Fetal distress</td>
<td>100%</td>
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<tr>
<td>Pulmonary edema or ARDS</td>
<td>93%</td>
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<tr>
<td>Cardiopulmonary arrest</td>
<td>87%</td>
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<tr>
<td>Cyanosis</td>
<td>83%</td>
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<tr>
<td>Coagulopathy</td>
<td>83%</td>
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<tr>
<td>Dyspnea</td>
<td>49%</td>
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<tr>
<td>Seizure</td>
<td>48%</td>
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<tr>
<td>Uterine atony</td>
<td>23%</td>
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<tr>
<td>Bronchospasm</td>
<td>15%</td>
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<tr>
<td>Transient hypertension</td>
<td>11%</td>
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<tr>
<td>Cough</td>
<td>7%</td>
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<tr>
<td>Headache</td>
<td>7%</td>
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<tr>
<td>Chest pain</td>
<td>2%</td>
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ARDS = adult respiratory distress syndrome.


### Clinical Presentation

AFE usually occurs intrapartum or in the immediate postpartum period. The symptoms are often sudden and protean. Clark et al. found the most common presenting signs and symptoms were hypotension and signs of nonreassuring fetal status (100%), pulmonary edema or respiratory symptoms (93%), cardiac arrest (87%), cyanosis (83%), and coagulopathy (83%) (Table 1). Tuffnell found that the most common presentation was a sudden change in either the maternal or fetal condition, but he did not specify the exact symptom.

Clark et al. proposed a biphasic model of the hemodynamic consequences of AFE. The initial response is acute pulmonary hypertension and vasospasm leading to right ventricular failure, hypoxia, and cardiac arrest. If one survives this initial insult, then the pulmonary hypertension is generally not sustained and may be replaced with left ventricular failure and pulmonary edema. Increased pulmonary artery pressure has not been consistently reported probably because this finding may be short-lived. When cardiac pressures are measured early in the process, pulmonary and right ventricular pressures have been found to be elevated. Hankins et al. demonstrated in a goat model that injecting 2.5 mL/kg of homologous amniotic fluid IV increased right heart and systemic vascular resistance. They also demonstrated that the presence of meconium was needed to produce left heart failure and hypoxia. These findings were confirmed by Petroianu et al. and might explain a role for meconium as the putative cause for mast cell degranulation and inflammatory response. This would explain why in Clark et al.’s series the worst maternal outcomes were in those with meconium-stained amniotic fluid. It is also possible that meconium does not cause the syndrome, but is simply present because it is a nonspecific marker of either maternal or fetal stress.

Coagulation disorders are a prominent feature of the amniotic fluid syndrome. DIC is present in more
than 83% of patients with AFE. The onset can occur as quickly as 10–30 min from onset of symptoms or may be delayed by as many as 4 h. Amniotic fluid contains tissue factor that acts as a procoagulant and may account for the coagulopathy. Tissue factor binds with Factor VII and activates the extrinsic coagulation pathway. Alternatively, the coagulopathy may be related to fibrinolysis due to increased levels of plasminogen activation inhibitor 1 in amniotic fluid.

The differential diagnoses of AFE include obstetric, nonobstetric and anesthetic etiologies (Table 2). Although each of the disorders listed has symptoms consistent with AFE, sudden onset of dyspnea in the face of cardiovascular collapse and DIC should lead the clinician to suspect AFE and initiate treatment. Since squamous cells have been found in the circulation of patients with and without the AFE syndrome, the diagnosis is one of exclusion based on presenting symptoms and clinical course, not based on laboratory or pathology findings.

Management

Early recognition of AFE is critical to a successful outcome. Management is primarily resuscitative and should be directed toward controlling the airway, maintaining vital signs and correcting coagulopathy. AFE is always associated with hypoxia. Therefore, control of the airway with tracheal intubation and administration of 100% O2 with positive pressure ventilation should be performed as soon as possible. Venous access with large bore IV catheters should be accomplished without delay. Arterial catheterization should also be considered for accurate arterial blood pressure monitoring and frequent blood sampling.

If the presentation is before delivery, providers should consider expeditious delivery of the fetus. Early delivery of the fetus in the resuscitation process may increase the chances of perinatal survival without neurologic sequelae. Also, delivery of the fetus aids in the maternal resuscitation efforts by improving venous return to the right heart.

Echocardiography is a sensitive tool to evaluate cardiac function and intravascular volume status. Both transthoracic and transesophageal modalities have been used by some clinicians in the diagnosis and management of AFE. Verroust et al. documented the presence of amniotic fluid in the right heart by echocardiography, which had a different appearance on echocardiography than blood. Care must be taken when inserting the transesophageal echocardiography probe in the coagulopathic patient.

Vasopressors and inotropic support are generally needed to varying degrees in AFE. Central venous access should be established for vasopressor infusion and monitoring. Choice of vasopressor drug depends on the clinical scenario. Phenylephrine, a pure α1 agonist, is often an excellent choice early in the treatment of AFE because at that time point systemic vasodilation is the most prominent circulatory abnormality. Later in the course of the process, inotropic support is commonly needed, and drugs, such as norepinephrine, epinephrine, and dopamine, should be considered. Vasopressin may be used as primary therapy or as an adjunct to other inotropic therapies and has the benefit of sparing the pulmonary vasculature from vasoconstriction, especially at low doses. In the face of right heart failure, milrinone or other phosphodiesterase inhibitors should be considered.

Blood and blood products, including fresh frozen plasma, platelets and cryoprecipitate, must be available and administered early in the resuscitation phase of AFE. The successful use of rfVIIa has been reported, although it has also been associated with massive intravascular thrombosis. Aprotinin has also been effective in reducing hemorrhage with AFE. However, after the results of the Blood Conservation Using Antifibrinolytics in a Randomized Trial (BART) study of patients undergoing coronary artery bypass surgery that demonstrated an increased mortality with aprotinin as compared with lysine analogues, the drug is no longer available. Other antifibrinolytic drugs, such as aminocaproic acid and tranexamic acid, have been described in the management of obstetric hemorrhage and menorrhagia and might also be considered during AFE. However, the authors are unaware of published reports in which these drugs were used specifically for treatment of the AFE-associated coagulopathy.

Other novel approaches for the treatment of AFE have been successfully used. Inhaled nitric oxide has been used in the treatment of right-sided heart failure and pulmonary hypertension. The use of cardiopulmonary bypass and placement of an intraaortic balloon pump counter pulsation with ECMO have also been described in the management of severe hypoxia and left heart failure associated with AFE. We recently reported the successful management of AFE with a right ventricular assist device and rfVIIa.

Table 2. Differential Diagnosis of Amniotic Fluid Embolism

<table>
<thead>
<tr>
<th>Obstetric causes</th>
<th>Acute hemorrhage</th>
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<tbody>
<tr>
<td>Placental abruption</td>
<td>Eclampsia</td>
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<tr>
<td>Uterine rupture</td>
<td>Peripartum cardiomyopathy</td>
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<td>Uterine atony</td>
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<td>Anesthetic causes</td>
<td>High spinal anesthesia</td>
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<tr>
<td></td>
<td>Aspiration</td>
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<td>Local anesthetic toxicity</td>
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<td>Nonobstetric causes</td>
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<td>Air embolism</td>
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<td></td>
<td>Anaphylaxis</td>
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<tr>
<td></td>
<td>Sepsis/septic shock</td>
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</table>

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CONCLUSION

AFE is a rare but often fatal complication of pregnancy and its onset can neither be predicted nor prevented. Early recognition of AFE with prompt intervention is paramount to a successful outcome. Management is resuscitative, geared toward maintaining vital signs and treating hemodynamic and coagulopathic derangements as they occur. A team approach among obstetrician, anesthesiologist and intensivist is necessary for a successful outcome. Despite early intervention, maternal and fetal mortality remain high. Aggressive management with novel products and devices, such as rFVIIa, cardiopulmonary bypass, ventricular assist device and ECMO, has been reported and should be considered.

REFERENCES

34. Stroup J, Haraway D, Beal JM. Aprotinin in the management of coagulopathy associated with amniotic fluid embolism. Pharmacotherapy 2006;26:689–93
Patients with severe tetanus in developed countries would typically be given deep sedation, paralysing drugs, and artificial ventilation (ie, are given a general anaesthetic until the disease wanes). For reasons of safety, Thwaites and colleagues enrolled spontaneously breathing patients who had severe tetanus and a tracheostomy. The fact that patients with severe tetanus in developing countries receive a tracheostomy, are given sedatives, hypnotics, and muscle relaxants, and are left breathing spontaneously (ie, are not necessarily ventilated) shows how different medicine can be in different settings. Is low-dose curare given to spontaneously breathing patients in developing countries for want of respirators? Irrespective of the reason for such treatment, it reflects expertise in finely tuning the use of curare, in controlling tetanic spasms while leaving the diaphragm unaffected (the last muscle to succumb to curare).

The crucial question now is: might magnesium sulphate reduce the need for tracheostomy in patients who would otherwise have had this procedure in settings such as those where Thwaites and colleagues’ study was done? Progress has been made since the time of Hippocrates: immunisation can prevent tetanus, and intensive care can control the symptoms. What else can be done apart from extend these advances to all patients with tetanus worldwide? Remember the so-called soothing plasters offered by the iatros in Hippocrates’ writing: might magnesium sulphate also reduce tetanus-associated pain?

Bruno Simini
Anestesia, Rianimazione e Terapia Antalgica, Ospedale di Lucca, Servizio Sanitario Nazionale, Regione Toscana, 55100 Lucca, Italy brunosimini@virgilio.it
I declare that I have no conflict of interest.


Amniotic fluid embolism: on the trail of an elusive diagnosis

In today’s Lancet, Michael Kramer and colleagues1 examine associations between amniotic fluid embolism and suspected risk factors in a large population-based retrospective cohort. They found an incidence of such embolism of about one in 17 000 singleton pregnancies in Canada between 1991 and 2002, which accords with previous estimates.2 However, the case-fatality rate of 13% is about half that in a population study from California.3 The researchers report an association between amniotic fluid embolism and medical induction of labour, the primary risk factor they investigated. Additionally, they identified several other risk factors, including uterine rupture or laceration, placenta praevia or abruption, advanced maternal age, caesarean or instrumental vaginal delivery, polyhydramnios, and eclampsia.

Amniotic fluid embolism is a rare but catastrophic syndrome that typically presents during labour and delivery or immediately postpartum. It is characterised by acute onset of a constellation of clinical features, including hypoxia, hypotension with shock, altered mental status, and disseminated intravascular coagulation. In our experience at a large tertiary referral centre for obstetric patients in southwestern Pennsylvania, a fulminant and rapidly progressive clinical course for patients is the rule rather than the exception. On the basis of data from observational studies and animal models, this course probably results from severe pulmonary vasoconstriction, left-ventricular impairment, and systemic inflammation with variable contributions of each over time (figure).

Early diagnosis of amniotic fluid embolism is an integral step in the delivery of timely and appropriate care. Substantial efforts have been made to describe the subset of pregnant women who are particularly at risk, on the basis of findings of population-based cohort studies. Unfortunately, estimation of the incidence of the disorder and identification of associated risk factors has been difficult because of the absence of a laboratory or clinical gold-standard for diagnosis. Efforts to develop
a diagnostic test are ongoing; however, amniotic fluid embolism remains a clinical diagnosis dependent on bedside judgment and exclusion of other diseases in the broad differential diagnosis (panel). Detection of patients with the disorder in large cohort studies has historically been based on the International Classification of Diseases 9th revision code 673.1, without other verifying evidence. Criteria typically used to make the diagnosis include the clinical features mentioned above and many suspected risk factors reported in population-based studies and an analysis of a national registry.

The absence of a gold-standard for diagnosis is an unavoidable source of error in epidemiological studies of amniotic fluid embolism. Despite this difficulty, Kramer and colleagues have been able to establish anew or strengthen associations between the disorder and many of the risk factors investigated. A few of the risk factors for which an association is reported, however, need to be scrutinised further. Cohort studies are vulnerable to misclassification bias, leading to both over-reporting and under-reporting with unknown frequency. As the researchers suggest, non-fatal amniotic fluid embolism might have been overdiagnosed in their study. Moreover, fatal cases of the disorder are less likely to be misdiagnosed because the signs and symptoms of amniotic fluid embolism (corresponding to multiorgan system failure) in conjunction with its fulminant and rapidly progressive nature distinguishes it from other causes of maternal death. Thus the association between amniotic fluid embolism and a given risk factor is most reliable when a death is recorded.

Risk factors related to death from amniotic fluid embolism include medical induction of labour, uterine rupture or laceration, placenta praevia or abruption, and advanced maternal age. The odds ratio for these factors is higher for deaths alone than for total cases of the disorder, which lends support to the argument for association. By contrast, polyhydramnios and eclampsia are not linked to fatal amniotic fluid embolism. Because of the high likelihood of overdiagnosis of non-fatal disorder and because only a few patients have amniotic fluid embolism plus polyhydramnios and eclampsia, as little as one incorrectly identified case could result in an erroneous association being drawn. Although relations with amniotic fluid embolism seem to be established for medical induction of labour and several other risk factors, more research is needed to ascertain whether an association exists for polyhydramnios and eclampsia.

Irrespective of the limitations described above, Kramer and colleagues’ study is important for the people who will be affected by this rare but deadly disease. The researchers have identified definitively the association of medical induction of labour with amniotic fluid embolism.

Panel: Differential diagnosis of amniotic fluid embolism

- Pulmonary thromboembolism
- Air embolism
- Haemorrhage
- Aspiration of gastric contents
- Anaesthetic complications
- Anaphylaxis
- Sepsis or systemic inflammatory response syndrome
- Myocardial infarction
- Cardiomyopathy
- Eclampsia
- Transfusion reactions

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embolism, and delineated the small but important effect this association can have on future obstetric patients. They have also strengthened the evidence for the association of the disorder with other risk factors for which data were previously inconsistent.

Information from Kramer’s and other population studies will hopefully lead to the development of a standard clinical definition of amniotic fluid embolism, until a definitive diagnostic test can be discovered. The standard could be developed with a set of weighted clinical and epidemiological criteria, and by validation of a numerical score to indicate presence of disease. Such a definition will permit accurate estimates of incidence and will serve to expedite the bedside care of women with amniotic fluid embolism in the future.

Jason Moore
Department of Critical Care Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA 15261, USA
moorje@ccm.upmc.edu

I declare that I have no conflict of interest


WHO Director-General election: public-health infrastructures

There is a growing recognition among governments, and their citizens of the value of health. A healthy population is more likely to foster economic, educational, and social development, contributing to gains in economic productivity and national prosperity.1

In recent years there has been an extraordinary increase in health initiatives designed to improve health outcomes, particularly in low-resource countries, including bilateral and multilateral assistance, private foundations, academic institutions, non-governmental organisations, and others. These initiatives, usually vertical in nature and targeted towards a specific disease or population, have done much to alleviate challenges in the developing world.

Yet, even with these successes, public-health infrastructure has serious gaps in many countries. These gaps lessen the ability of governments to implement comprehensive public-health initiatives and to improve health outcomes. WHO’s Eleventh General Programme of Work discusses the challenges of closing the gaps in international health responses. The plan says that “Rather than to implement programmes itself, WHO’s role is to contribute to building sustainable institutional capacity”.2

In many countries, especially those that are low in resources, the over-arching problem is a lack of a focused and functional public-health system that can identify, prioritise, address, and assess disease-control programmes. Without such a system, progress is restricted. If governments do not have the physical infrastructure, capital investment, and human resources, public-health functions are limited in scope, taken on piecemeal by donors or not at all. Sadly, there are scenarios in which donor funds, resources, or services go unused because countries do not have the capacity to absorb them or the networks to distribute them.

The discussions at the recent HIV/AIDS conference in Toronto highlighted the importance of good public-health

MCQ QUESTIONS (True / False)

Before reading this tutorial, try to answer the following questions. The answers can be found at the end of the article.

1. Non-obstetric surgery in pregnancy:-
   a) occurs in approximately 5%
   b) appendicectomy is the most common procedure performed
   c) is associated with increased congenital abnormalities
   d) is associated with increased spontaneous abortion
   e) laparoscopy is contraindicated

2. During pregnancy:
   a) MAC decreases by 10%
   b) plasma cholinesterase levels increase
   c) suxamethonium has a clinically prolonged duration of action
   d) platelet consumption decreases
   e) Functional Residual Capacity (FRC) may be less than alveoli closing volume

3. Concerning foetal safety during non-obstetric surgery:
   a) teratogenicity is the most serious risk
   b) hyperoxia is not dangerous
   c) foetal heart rate monitoring perioperatively improves foetal outcome
   d) ketamine is the anaesthetic drug of choice
   e) maternal hypercarbia should be avoided

INTRODUCTION

During pregnancy, surgery for non-obstetric procedures occurs in up to 2% of women. This figure may be considerably higher in the first trimester as pregnancy may go undetected at the time of surgery. Approximately 42% of procedures occur in the first trimester, 35% during the second and 23% during the third.

The range and incidence of procedures are similar to the non-pregnant group of young women. Acute abdominal problems are most common, with appendicectomy ranking first followed by cholecystectomy. Pregnancy predisposes to cholelithiasis and approximately 3% of pregnant women develop gallstones, however only a limited number require surgery. Other common problems include adnexal disease (e.g. ovarian cysts which may rupture or become torted) and trauma. Much less
Anaesthetists who care for pregnant patients undergoing non-obstetric surgery must provide safe anaesthesia for both the mother and the foetus. To maintain maternal safety the physiological and anatomical changes of pregnancy must be considered and anaesthetic techniques and drug administration modified accordingly. Foetal wellbeing is related to avoidance of foetal asphyxia, teratogenic drugs and preterm labour.

MATERNAL SAFETY

Physiological / anatomical changes

Cardiovascular changes
Maternal cardiac output increases in pregnancy by 50% and peaks by the end of the 2nd trimester. This is due to a combination of an increased heart rate (25%) and stroke volume (30%). The increase in heart rate is a reflex response to a lowered systemic vascular resistance (SVR) caused by circulating oestrogen and progesterone. Left ventricular hypertrophy and dilatation facilitate the increase in stroke volume but myocardial contractility remains unchanged.

ECG changes that occur in pregnancy and are entirely normal include left axis deviation and minor ST / T wave changes. Heart murmurs are also common due to turbulence associated with increased blood flow.

As the enlarging uterus moves out of the pelvis it can compress the inferior vena cava and the descending aorta in the supine position. The compression of the inferior vena cava causes decreased venous return and hence preload, which reduces cardiac output by up to 20%. This is known as supine hypotension syndrome. Pregnant patients compensate for hypotension by an increase in sympathetic tone causing vasoconstriction and tachycardia. This may divert blood away from organs such as the uterus, with subsequent foetal distress. The compression of the aorta can cause a further reduction in uterine blood flow. Aortocaval compression becomes clinically relevant from approximately 20 weeks gestation. It can be relieved by a left lateral tilt of 15 degrees, which is therefore essential in all pregnant patients in the supine position after 20 weeks. This is especially important to remember when a patient is under regional anaesthesia/analgesia since hypotension may be potentiated by a sympathetic block.

There is an increase in blood volume in pregnancy of between 35-50% at term. There is both an increase in plasma volume and red cell volume, but a greater increase in plasma volume which leads to a dilutional anaemia. The reduced blood viscosity aids flow through the uteroplacental circulation and the increase in volume serves as a protective measure against haemorrhage at delivery. It must be remembered that because of the increase in blood volume, along with a resting tachycardia, there may be delay in the onset of the classical symptoms and signs of hypovolaemia.

Pregnancy is a hypercoaguable state with an increase in most clotting factors. The platelet count may fall but there is actually an increase in production and consumption. Pregnancy is a significant risk factor for thromboembolism and therefore thromboprophylaxis is essential in the postoperative period when the risk is further increased by immobility and the hypercatabolic state.
Respiratory changes
The respiratory changes of pregnancy are perhaps the most important for anaesthetists to note.

There is an increased oxygen demand of up to 60% at term. This is met by an increased cardiac output and an increase in minute ventilation (MV). MV increases early due to an increase in respiratory rate and tidal volume and is up by 45% by term. This increase in MV is mediated by progesterone which acts as a respiratory stimulant. The increased MV causes a mild respiratory alkalosis (PaCO$_2$ decreases by 1kPa). The increase in pH is limited by increased renal bicarbonate excretion. Relative hypocapnia should be maintained when artificially ventilating pregnant patients. An increase in maternal PaCO$_2$ limits the gradient for CO$_2$ diffusion from foetal to maternal blood leading to foetal acidosis.

The functional residual capacity (FRC) is the main oxygen reserve in the apnoeic patient and is decreased in pregnancy due to the enlarging uterus displacing the diaphragm upwards. This is further exacerbated in the supine position and increases as the pregnancy progresses.

Airway management may be challenging during pregnancy. Bag-mask ventilation may be more difficult due to increased soft tissue in the neck. Laryngoscopy can be hindered by weight gain and breast engorgement. Increased oedema of the vocal cords due to increased capillary permeability can hinder intubation and increase the risk of bleeding. This may make further attempts at intubation more difficult and increase the incidence of failed intubation. Increased maternal oxygen consumption and reduced FRC results in rapid oxygen desaturation during attempts at intubation. Smaller sized endotracheal tubes may be needed and all anaesthetists should be familiar with a failed intubation drill. Nasal intubation should be avoided due to increased vascularity of mucous membranes.

Given the combination of these changes, careful pre-oxygenation is essential prior to induction of anaesthesia. This should be confirmed if possible by monitoring the end tidal oxygen fraction. In a well pre-oxygenated patient this should be >0.9. Pre-oxygenation can be less efficient in the term parturient in the supine position because the closing volume of the alveoli may be greater than the FRC. Pre-oxygenation in a slightly head up position may help this.

Gastrointestinal changes
Circulating progesterone reduces the lower oesophageal sphincter (LOS) tone, increasing the incidence of oesophageal reflux. This is further exacerbated by anatomical changes. The gravid uterus is displaced upwards and to the left pushing the intra-abdominal part of the oesophagus into the thorax in most pregnant women. This often makes the LOS incompetent and lowers the barrier pressure. These factors, along with a lowered stomach pH, increase the risk and severity of aspiration pneumonitis under general anaesthesia.

It is recommended that from 16 weeks gestation patients undergoing general anaesthesia should be given prophylaxis against aspiration pneumonitis. This usually includes a non-particulate antacid such as sodium citrate 0.3M 30ml and an H2 receptor antagonist e.g. ranitidine 150 mg orally or 50mg intravenously. Some anaesthetists may also choose to give a prokinetic such as metoclopramide. Induction of anaesthesia should be by a rapid sequence technique with cricoid pressure and a fast acting muscle relaxant such as suxamethonium. A cuffed endotracheal tube should be used. At the end of the procedure patients should be extubated fully awake in the lateral position.

- Remember left lateral tilt to prevent aortocaval compression
- Remember meticulous pre-oxygenation to prevent hypoxia
- Remember antacid prophylaxis and rapid sequence induction to reduce risk of aspiration
Drugs: Altered pharmacokinetics / pharmacodynamics

Pharmacokinetic and pharmacodynamic profiles are altered in pregnancy and drugs should be titrated accordingly.

The minimum alveolar concentration (MAC) of volatile agents is reduced by 30% under the influence of progesterone and endogenous endorphins. There is a decrease in plasma cholinesterase levels by 25% from early pregnancy, but prolonged neuromuscular blockade with suxamethonium is uncommon due to increased blood volume causing an increased volume of distribution. Non-depolarising muscle relaxants have a prolonged duration of action. Neuromuscular monitoring with a nerve stimulator is recommended. The increased blood volume causes a physiological hypoalbuminaemia. This alters the plasma protein binding and increases the free or unbound fraction of drugs. An example of this is local anaesthetics. As well as decreased plasma protein binding, there is also increased neural tissue sensitivity. These factors decrease the therapeutic doses and also the toxic plasma levels of local anaesthetic agents.

The volume of the epidural and subarachnoid spaces is reduced due to the gravid uterus compressing the inferior vena cava causing distension of the epidural venous plexus. This leads to a more extensive spread of local anaesthetic agents administered during central neuraxial blockade and also increases the risk of inadvertent intravascular injection. Careful aspiration prior to injection should always be performed.

FOETAL SAFETY

Prevention of foetal asphyxia

One of the most serious risks to the foetus during maternal surgery is intrauterine asphyxia. This must be avoided by maintaining maternal oxygenation and haemodynamic stability. It is extremely important to avoid hypoxia, extreme hyper and hypocarbia, hypotension and uterine hypertonus. Maternal hypoxaemia causes uteroplacental vasoconstriction and decreased perfusion, causing foetal hypoxia, acidosis and ultimately death.

There is a linear relationship between maternal and foetal PaCO₂. Maternal hypercarbia limits the gradient for CO₂ diffusion from foetal to maternal blood and leads to foetal acidosis. Therefore end-tidal carbon dioxide monitoring should be used to guide ventilation and arterial blood gas analysis should be considered during prolonged or laparoscopic surgery. Hypocarbia is also problematic, potentially causing uteroplacental vasoconstriction and foetal acidosis, although the mild hypocapnia that occurs with the physiological changes of pregnancy should be maintained (PaCO₂ around 4kPa).

Uteroplacental circulation is not autoregulated and hence perfusion is entirely dependant on the maintenance of an adequate maternal blood pressure and cardiac output. Hypotension can be caused by anaesthetic drugs, central neuraxial blockade, hypovolaemia or aortocaval compression. Maternal hypotension needs to be treated aggressively by ensuring left lateral tilt and boluses of IV fluids. Additional vasopressors may be required and currently it is felt alpha agonists such as phenylephrine and metaraminol produce a better foetal acid balance than indirect sympathomimetic agents such as ephedrine. Ephedrine also has a relatively slow onset and long duration of action and tachyphylaxis can occur making titration difficult.

Drugs and teratogenicity

Teratogenicity is defined as the observation of any significant change in the function or form of a child secondary to prenatal treatment. The teratogenicity of a drug depends upon the dose administered, the route of administration, the timing of foetal exposure and the species administered to. During the first
two weeks of human gestation the teratogens have an all or none phenomenon; the foetus is lost or is preserved fully intact. The period from the 3rd to the 8th week of gestation, represents the most important time for organogenesis during which drugs can exert their most serious teratogenic effects. After this, drug exposure should not cause organ abnormalities, but foetal growth retardation may occur.

Although most anaesthetic drugs are known teratogens in certain species, most agents are safe in humans. The foetus is at more risk from asphyxia than the teratogenic effect of anaesthetic drugs. Studies looking at the outcomes of women who underwent surgery during pregnancy suggest no increase in congenital anomalies in their offspring but an increase in foetal loss, growth restriction and low birth weight attributed to the requirement for surgery (not anaesthetic administration). There is some concern from animal and epidemiological studies that exposure to general anaesthetic agents may cause neurodevelopmental delay in infants. It is difficult to extrapolate animal findings to humans and in epidemiological studies it is difficult to distinguish the potential confounding effects of anaesthesia, reason for surgery and underlying medical conditions.

Nitrous oxide inhibits methionine synthetase, and therefore there is concern it could affect DNA synthesis in the developing foetus. It has also been shown to be teratogenic during peak organogenesis in rodents, but there is no evidence in humans. Anaesthesia can be safely delivered without nitrous oxide and therefore many would avoid its use during non-obstetric surgery in the pregnant woman.

Another drug of concern is ketamine. This causes increased uterine tone and foetal asphyxia and should not be used in the first two trimesters. The effect is not seen in the third trimester.

Benzodiazepines have been associated with a cleft lip and palate in animal studies. The association in humans is controversial. A single dose has not been associated with teratogenicity. Long term use should be avoided as neonatal withdrawal may occur. Single doses may be useful to provide anxiolysis preoperatively.

Prevention of pre-term labour / foetal monitoring

Surgery during pregnancy increases the risk of spontaneous abortion, preterm labour and preterm delivery. This risk is increased with intra-abdominal procedures. Uterine manipulation should be kept to a minimum and drugs that increase uterine tone (e.g. ketamine) should be avoided. Prophylactic tocolytic therapy is controversial as there are associated maternal side effects and efficacy during non-obstetric surgery has not been proven.

Perioperative foetal monitoring is also an area of controversy. From 18-22 weeks foetal heart rate (FHR) monitoring is feasible and from 25 weeks heart rate variability can be observed. Continuous monitoring may be technically difficult during abdominal operations or in cases of maternal obesity. Anaesthetic agents reduce both baseline FHR and FHR variability and therefore interpretation is difficult and may lead to unnecessary interventions. Anaesthetic agents do not cause decelerations or persistent foetal bradycardia and these changes may indicate foetal distress. Monitoring may enable swift action to be taken such as the optimisation of maternal haemodynamics, oxygenation and ventilation.

Although perioperative foetal monitoring has not been shown to improve foetal outcome, a sensible approach would be to use cardiotocography (CTG) monitoring where possible and practical when the foetus is of a viable age. The obstetricians and neonatologists should be informed, appropriately trained personnel available to interpret the CTG and a prior action plan in place for when there is evidence of foetal distress unresponsive to conservative measures.

If the foetus is not of a viable age or perioperative CTG monitoring is not possible / practical, FHR monitoring should occur pre and post-operatively and staff should be alert to the signs of premature labour.
LAPAROSCOPIC SURGERY

There were previous concerns regarding foetal safety during laparoscopic surgery. These included fears of direct uterine and foetal trauma, foetal acidosis due to absorbed carbon dioxide and decreased maternal cardiac output secondary to the increased intra-abdominal pressure and positioning with a subsequent decrease in uteroplacental perfusion.

There are advantages to laparoscopic surgery for both the mother and the foetus such as decreased post-operative pain (and therefore less need for analgesics), shorter recovery times and a lower risk of thromboembolic events.

A Swedish study compared laparotomy and laparoscopy performed in pregnancy in over 2 million deliveries. Premature delivery, growth restriction and low birth weight were more common in both groups compared to the general population but there were no differences between the laparotomy and laparoscopy groups.

Pregnancy should therefore not be seen as a contraindication to laparoscopic surgery if surgery is required. Certain precautions should be taken. Pneumatic stockings should be used to promote venous return and the lowest pressure pneumoperitoneum (<12mmHg) should be used where possible. Aortocaval compression should be avoided and changes in position should be undertaken slowly. PaCO₂ should be closely monitored by the routine use of end tidal carbon dioxide monitoring and consideration of arterial blood gas analysis in selected cases. FHR monitoring may be advisable to detect foetal compromise early allowing optimisation of maternal haemodynamics. FHR changes may indicate the need for temporary deflation of the pneumoperitoneum.

POSTOPERATIVE CARE

As previously stated pregnancy induces a hypercoaguable state and the risk of thromboembolic disease is further increased by postoperative venous stasis. Attention to thromboprophylaxis is therefore essential. This should include early mobilisation, maintaining adequate hydration, TED stockings and other calf compression devices and consideration of pharmacological prophylaxis (for example subcutaneous low molecular weight heparin).

Analgesia

Adequate analgesia is important as pain will cause increased circulating catecholamines which will impair uteroplacental perfusion. Analgesia may mask the signs of early preterm labour and therefore tocometry is useful to detect contractions. This will enable tocolysis to be administered without delay. If a pregnancy continues beyond the first postoperative week the incidence of premature labour is no higher than the non-surgical pregnant patient.

The FDA (United States Food and Drug Administration) introduced a classification system in 1979 of drug risk to the foetus. This runs from Category A (safest) to Category X (known danger).
Table 1. FDA classification of foetal harm risk from drugs.

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category A</td>
<td>Adequate and well controlled studies have failed to demonstrate a risk to the foetus in the first trimester of pregnancy (and there is no evidence of risk in later pregnancies).</td>
</tr>
<tr>
<td>Category B</td>
<td>Animal reproduction studies have failed to demonstrate a foetal risk but there are no controlled studies in pregnant women, OR animal reproduction studies have shown an adverse effect, but adequate well controlled studies in pregnant women have failed to demonstrate a risk to the foetus in any trimester.</td>
</tr>
<tr>
<td>Category C</td>
<td>Animal reproduction studies have shown an adverse effect on the foetus and there are no adequate well controlled studies in humans, or studies in animals and humans are not available. Potential benefits of drugs may warrant use of drug in pregnant women despite potential risks.</td>
</tr>
<tr>
<td>Category D</td>
<td>There is positive evidence of human foetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (e.g. life threatening situation or serious disease for which safer drugs are not available).</td>
</tr>
<tr>
<td>Category X</td>
<td>Studies in animals or humans have demonstrated foetal abnormalities, or evidence based on human experience, and the risk of use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.</td>
</tr>
</tbody>
</table>

There are other classification systems from other countries. The FDA requires a relatively large amount of high quality data for a drug to be classified as Category A. As a result many drugs classified as Category A in other countries are classified as Category C by the FDA.

The table below shows how many of the common analgesic drugs used are classified by the FDA and their safety in breastfeeding.

**PUERPERIUM**

The puerperium covers the 6 week period following childbirth during which time the various changes that occurred during pregnancy revert to the non pregnant state. The cardiovascular system and blood volume return to normal by the end of 2 weeks. After delivery of the placenta the uterus is the size of a 20 week pregnancy and decreases by 1 finger breadth each day, so that by day 12 it is no longer palpable. It is wise to avoid elective surgery in the initial 6 week post partum period to allow the body to return to its normal physiological function.

If anaesthesia is undertaken in this time or for operative delivery of the foetus women may wish to know the effects on breastfeeding. Administration of drugs to the breastfeeding mother can inhibit lactation or cause direct harmful effects to the infant due to excretion in breast milk. For many medications there is insufficient evidence available to provide accurate guidance on drug safety during breastfeeding. When prescribing or administering drugs consider:

- Is the medication really needed?
- Minimise drug exposure by administering just after breastfeeding.
- Breastfeeding is the gold standard for infant nutrition. Balance the risk of drug excretion in milk with advantages of continued breastfeeding.
Table 2. Common analgesics used in pregnancy / puerperium.

<table>
<thead>
<tr>
<th>Drug</th>
<th>FDA Risk Category</th>
<th>Foetal Risk</th>
<th>Breastfeeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ibuprofen</td>
<td>C</td>
<td>No adequate human data. Studies in animals show adverse foetal effects. Use in 3rd trimester can cause constriction of ductus arteriosus.</td>
<td>Minimal amounts excreted into milk. Safe in breast feeding.</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>B</td>
<td>No adequate human data but studies in animals do not indicate risk to foetus. Can cause constriction of ductus arteriosus in 3rd trimester.</td>
<td>Excreted into milk. Considered safe.</td>
</tr>
<tr>
<td>Codeine</td>
<td>C</td>
<td>No adequate human data. High doses at term can cause neonatal withdrawal / respiratory depression.</td>
<td>Excreted into milk in insignificant amounts, compatible with breast feeding.</td>
</tr>
<tr>
<td>Morphine</td>
<td>C</td>
<td>Chronic maternal use causes neonatal withdrawal and respiratory depression.</td>
<td>Excreted into milk. Considered safe in therapeutic doses.</td>
</tr>
<tr>
<td>Tramadol</td>
<td>C</td>
<td>Human data is lacking. When used in labour may cause fewer maternal side effects and lower neonatal respiratory depression than other opioids.</td>
<td>Excreted into milk. Unknown effect on infants. Safely used by many mothers (although avoidance recommended by manufacturers)</td>
</tr>
</tbody>
</table>

Breast milk production is dependant on adequate maternal hydration and regular stimulation (either by the baby feeding or by the mother expressing). If scheduled for anaesthesia / surgery encourage the mother to breastfeed as near as possible to the procedure.

**General anaesthesia**

Propofol and thiopentone are found in breast milk in insignificant amounts, as are levels of volatile agents. As neuromuscular blocking agents are large, ionised and water soluble they are not excreted into breast milk. After general anaesthesia women can be advised to express and discard the first sample of milk and to resume infant feeding after this. Many argue that no portion of milk needs to be wasted. All of the commonly used antiemetics are advised to be used ‘with caution’ or ‘only if essential’ by manufacturers.

**Regional anaesthesia**

Local anaesthetics are not excreted into breast milk in amounts sufficient to be harmful. Therefore breast feeding can continue as normal after regional anaesthesia.

**Analgesics**

See above table for commonly used analgesics. The American Academy of Pediatrics (AAP) published a statement on drug transfer into human milk and possible effects on the infant. The AAP considers paracetamol, most non-steroidal anti-inflammatory drugs and morphine compatible with breastfeeding.
SUMMARY

Non-obstetric surgery during pregnancy is not uncommon and anaesthetists should be aware of the implications for management. The physiological changes of pregnancy need to be considered, especially the avoidance of aortocaval compression, antacid prophylaxis and adequate preoxygenation. The airway needs careful evaluation preoperatively.

The main risk to the foetus is asphyxia. This can be avoided by ensuring adequate maternal oxygenation and ventilation, avoiding hypotension and avoiding drugs that increase uterine tone. This should ensure adequate uteroplacental perfusion. Perioperative foetal heart rate monitoring may be useful if trained staff are available and it is practically possible. Regional anaesthesia is likely to have benefits over general anaesthesia. Attention should be paid to thromboprophylaxis, analgesia and signs of preterm labour in the postoperative period.

When caring for pregnant ladies undergoing non-obstetric surgery a multidisciplinary team is essential. This should include surgeons, anaesthetists, obstetricians, midwives, nurses and neonatologists where available. Elective surgery should be postponed until 6 weeks postpartum when possible. Non-elective surgery should be delayed until the 2nd trimester when organogenesis has occurred and the risk of teratogenicity decreases but this may not always be possible.

- Remember the physiological and anatomical changes of pregnancy
- Prevent foetal asphyxia by maintaining maternal oxygenation, ventilation and haemodynamic stability
- Remember postoperative thromboprophylaxis

ANSWERS TO QUESTIONS
1. FTFTF
2. FFFFT
3. FTFFT

REFERENCES / FURTHER READING
Anesthesia for non-obstetric surgery in the pregnant patient

M. VAN DE VELDE, F. DE BUCK

Department of Anesthesiolog, University Hospitals Gasthuisberg, Catholic University of Leuven, Leuven, Belgium

**ABSTRACT**

Surgery during pregnancy is relatively common. The present review of the literature will focus on relevant issues such as maternal safety during non-obstetric surgery in pregnancy, teratogenicity of anesthetic drugs, the avoidance of fetal asphyxia, the prevention of preterm labor, the safety of laparoscopy, the need to monitor the fetal heart rate and will finally give a practical approach to manage these patients.

**Key words:** Non-obstetric surgery - Pregnancy - Anesthesia - Teratogenicity - Fetal asphyxia.

Non-obstetric surgery during pregnancy is relatively common. Several older studies have found that between 0.15% and 2% of pregnant women underwent non-obstetric surgery. The European Union has 380 million inhabitants. Assuming a birth rate of approximately 10/1 000, 3.8 million pregnancies enter the (European Union) EU statistics each year. This means that in the EU, each year between 5 700 and 76 000 pregnant women undergo non-obstetric surgery. This figure may be a serious underestimation as many women of childbearing age may present for surgery with an unrecognized pregnancy. Several studies suggest this occurs in 0.3% to 2.4% of women presenting for surgery. However, routine pregnancy testing in women of childbearing age is not routinely recommended, since many of these pregnancies might be identified following a detailed history.

The most common indications for surgery during pregnancy are either pregnancy related or non-pregnancy related. Pregnancy related surgery includes interventions for cervical incompetence and surgery for ovarian cyst problems. Increasingly popular is also fetal surgery with an estimated 250 to 500 cases performed now each year in the EU. The most common non-pregnancy related indications are acute abdominal problems (most commonly appendicitis and cholecystitis), maternal trauma and surgery for maternal malignancies. Of course any type of emergent surgery may be carried out during pregnancy.

Surgery may be indicated during any stage of pregnancy. In a Swedish registry of 5 405 patients who had surgery during pregnancy, 42% had an intervention during the 1st trimester, 35% during the 2nd trimester and 23% during the 3rd trimester. When caring for pregnant women undergoing non-obstetric surgery, anesthetists must provide safe anesthesia for both mother and child. Maternal safety is related to the physiologic adaptations associated with pregnancy, which enforce anesthetists to adapt their standard anesthetic techniques. Fetal safety relates to teratogenicity, avoidance of fetal asphyxia and avoidance of preterm labor and delivery. Each of these issues will be discussed in this manuscript.
Maternal safety: maternal physiologic adaptations to pregnancy

The pregnant woman undergoes significant physiologic adaptations to pregnancy. Pregnancy induced changes pose hazards to mother and fetus during anesthesia and surgery. Most of these changes are due to the mechanical effects of the enlarging uterus, hormonal changes associated with pregnancy, increased metabolic demands and the low resistance placental circulation. The most important changes for the anesthetist are summarized below as well as their impact on anesthetic practice in this patient population.

Cardiovascular changes

Cardiac output increases gradually beginning at 8 weeks of gestation and reaching its maximal increase by the end of the 2nd trimester. Both heart rate and stroke volume are increased, resulting in a 50% increase of cardiac output by the end of the 2nd trimester. Myocardial contractility remains unchanged, but systemic vascular resistance is decreased. This is primarily due to progesterone as well as the presence of the low resistance placentas.

Of concern to the anesthetist is aortocaval compression. Compression by the gravid uterus of the vena cava results in a reduced cardiac preload, reduced cardiac output and maternal hypotension. Aortocaval compression becomes apparent from the 2nd trimester onwards. It occurs when the patient is supine and can be relieved by assuming the lateral position. During surgery left lateral tilt should be used.

Mild cardiovascular signs, such as mild tricuspid regurgitation, mild pericardial effusion, left ventricular hypertrophy, accentuation of the first heart sound and electrocardiographic changes are perfectly normal during pregnancy.

Respiratory changes

Minute ventilation is increased up to 70% by term. This results in chronic respiratory alkalosis with a decreased PaCO₂ of 28 mmHg to 32 mmHg and a slight increase in pH to 7.44. This is obvious from the 1st trimester onwards. Bicarbonate is increasingly excreted by the kidneys. Oxygen consumption increases, but PaO₂ does not change.

Functional residual capacity is decreased by approximately 20% and even more in the supine position. The pregnant patient is, therefore, at increased risk for hypoxia when periods of apnea occur. Pregnancy also results in anatomical changes in the airway, making endotracheal intubation more difficult. This is further complicated by the increased vascularization resulting in more bleeding during intubation attempts. Also mask ventilation is sometimes much more difficult.

Gastro-intestinal changes

Although gastric emptying is normal during pregnancy, the risk of gastric content aspiration is increased in pregnancy, because of reduced barrier pressures at the level of the lower esophageal sphincter. This already is obvious from the 15th week onwards, especially in patients with heartburn. This is further accentuated by distortion of the gastric and pyloric anatomy.

Other important changes

As a result of an increased plasma volume, anemia occurs, despite an increase in red blood cell volume. Pregnancy is also associated with a benign leukocytosis. Pregnancy also causes a hypercoagulable state with increases in most coagulation factors. Platelet turnover is enhanced as well as clotting and fibrinolysis. Pregnancy is, thus, a state of compensated intravascular coagulation. Thrombocytopenia can occur in up to 1% of pregnancies without signaling pre-eclampsia. The hypercoagulable state puts the pregnant patient at high risk for postoperative thromboembolic complications.

Glomerular filtration rate increases by 50% during pregnancy and as a result creatinine clearance is increased by 50% as well. Serum concentrations of creatinine are, therefore, reduced by almost 1/3.

Anesthetic requirements are significantly reduced for both inhalational and intravenous anesthetic agents. Pregnancy is associated with an increased sensitivity to inhalational anesthetics with minimum alveolar concentration reductions up to 40% being reported. Similarly, the sensitivity to intravenous agents is also increased. The reduced anesthetic requirements are most
likely due to a progesterone effect. Less spinal
or epidural anesthetics are required to produce a
similar dermatomal spread in pregnancy as com-
pared to non-pregnant patients. This is due to hor-
monal as well as mechanical effects of the enlarg-
ing uterus. Non-depolarizing muscle relax-
ants have a prolonged duration of action, while
the duration of action of succinylcholine is una-
fected by pregnancy.

These physiologic changes of pregnancy
enforce anesthetists to adapt their routine anes-
thetic technique. Acid aspiration prophylaxis (in
my institution a combination of an H2-block-
ing agent, sodium citrate orally 30 mL 0.3 M
and metoclopramide) is, therefore, recom-
ended to reduce gastric content and increase gastric
pH. This clearly results in a reduced morbidity
and mortality when accidental aspiration occurs.
Adequate positioning with left lateral displace-
ment of the uterus (at least 20° left lateral tilt) is
required to avoid the supine hypotensive syn-
drome. This should be performed from the 2nd
trimester onwards. The pregnant patient is more
prone to hypoxia because of decreased function-
al residual capacity and increased oxygen con-
sumption. Careful denitrogenation prior to
induction of general anesthesia is, therefore, rec-
ommended. A rapid sequence induction should
be performed using cricoid pressure and a rap-
idly acting muscle relaxant. The drug of choice
remains succinylcholine. Rocuronium would
be an alternative. However, it exerts a signifi-
cantly prolonged duration of action which is diffi-
cult to reverse. Mild respiratory alkalosis (for
non-pregnant women) should be maintained
during artificial ventilation. Pregnant patients
are more prone to thromboembolic complica-
tions and adequate prophylactic measures should
be taken including prophylactic administration
of low molecular weight heparines.

Teratogenicity of anesthetic drugs

Drugs should only be administered to pregnant
patients if the benefits outweigh the risks. Anesthetic drugs affect intra- and intercellular sig-
naling and have known effects on cellular mitosis
and DNA synthesis. Such intracellular sys-
tems are involved in cellular differentiation and
organogenesis. Therefore, all anesthetic agents can
be potentially teratogenic. The teratogenicity of
a drug is determined by the dose administered,
the route of administration, the timing of exposure
to the fetus and the species which is exposed to
the drug. Timing of exposure is of crucial impor-
tance. During the first 15 days of human gesta-
tion an all or nothing phenomenon occurs: the
fetus is lost or the fetus is preserved fully intact.
During the time of organogenesis (15-56 days)
structural abnormalities may occur. After this peri-
od, functional changes can be observed, but struc-
tural abnormalities are rare.

Since prospective clinical studies are impracti-
cal, unethical and would require enormous num-
bers, most of our knowledge comes from animal
studies, accidental exposure and reports from series
of patients that underwent anesthesia whilst being
pregnant.

Although most anesthetics are known terato-
gens in certain species, when a high enough dose
is administered or when directly administered to
the fetus, most agents are, however, perfectly safe
in clinical circumstances. We now know that local
anesthetics, volatile anesthetics, induction agents,
muscle relaxants and opioids are not teratogenic
when used in clinical concentrations and when
normal maternal physiology is maintained. Indeed,
derangements in maternal physiology are terato-
genic themselves.

It is probably best to avoid nitrous oxide during
pregnancy because it is not necessary to use this
agent to provide safe and effective anesthesia.
Nitrous oxide has known effects on DNA synthe-
sis and has been shown to have teratogenic effects
in animals.

Avoidance of fetal asphyxia

The most important and serious risk to the fetus
of maternal surgery during pregnancy is that of
intrauterine asphyxia. The most challenging goal
of the anesthetist is, therefore, to avoid fetal asphyx-
ia by maintaining normal maternal oxygenation
and hemodynamics. Maternal oxygenation, mater-
nal carbon dioxide levels, maternal blood pressure
and uterine tonus are all factors that need to be
controlled during surgery to avoid fetal asphyxia. It is extremely important to avoid hypoxia, hypercarbia, hypocarbia, maternal hypotension and uterine hypertonus during non-obstetric surgery. This is probably much more important than concerns about teratogenicity of different anaesthetic drugs.

Mild periods of hypoxemia of short duration are well tolerated. However, prolonged or serious maternal hypoxemia causes utero-placental vasoconstriction and decreased utero-placental perfusion, resulting in fetal hypoxemia, acidosis and, ultimately, fetal death. Hyperoxia is not dangerous, contrary to what previously was thought. It has been clearly demonstrated that hyperoxia does not result in an increased uterine vascular resistance nor does it decrease fetal oxygenation as measured by fetal scalp blood gas analysis.

Maternal hypercarbia directly induces fetal respiratory acidosis. Severe fetal respiratory acidosis causes fetal myocardial depression. Hypercapnia also results in uterine artery vasoconstriction and reduced uterine blood flow. Similarly, hypocapnia as well results in reduced uterine blood flow and ultimately fetal acidosis.

To treat maternal hypotension, ephedrine was long considered to be the first choice. However, recent data suggest that phenylephrine is equally efficacious to maintain normal maternal blood pressure and that phenylephrine produces a significantly better fetal acid base balance, at least in term pregnancies undergoing C-section under regional anesthesia. Therefore, the current advice is to treat aggressively maternal hypotension with phenylephrine.

Several drugs used commonly in anesthesia, such as ketamine or IV local anesthetics, can cause uterine hyperactivity and should be avoided.

Fetal heart rate monitoring during surgery

From 18-22 weeks fetal heart rate (FHR) monitoring is feasible and from 25 weeks heart rate variability can be observed. I would recommend using FHR monitoring routinely when feasible. It certainly is a very good indicator of inadequate utero-placental perfusion. Unfortunately, there is no evidence to show that using intraoperative FHR monitoring improves fetal outcome. As a result some recommend not to use it. The issue remains controversial, but many obstetric textbooks do advice to monitor whenever feasible. Remember, however, that loss of variability is not always an indicator of fetal distress, but may also signal fetal anesthesia.

Laparoscopy

Many authors have raised concerns about fetal well being during laparoscopy. They fear direct uterine or fetal trauma and they also fear fetal acidosis from absorbed carbon dioxide. Finally, because of increased intra-abdominal pressure, maternal cardiac output and thus utero-placental perfusion might be reduced. Animal data have corroborated these concerns. However, clinical experience, using a careful surgical and anesthetic technique, has been favorable. Reedy et al. compared laparotomy and laparoscopy performed in pregnancy in over 2 million pregnancies in Sweden during a 20-year period. These authors included 2 181 laparoscopies and 1 522 laparotomies with a gestational age between 4 and 20 weeks. They compared 5 fetal parameters (birth weight, gestational duration, growth restriction, infant survival and fetal malformations) for each type of surgery with the overall outcome in the non-operated population. Premature delivery, growth restriction and low birth weight was more frequent in the operated groups as compared to the general
population. No differences between laparoscopy and laparotomy were identified.

Thus, the following guidelines were issued by the Society of American Gastrointestinal endoscopic surgeons regarding laparoscopic surgery during pregnancy. Whenever possible, surgery should be deferred to the 2nd trimester. One should obtain a preoperative obstetric consultation. Intermittent pneumonic compression devices to prevent thrombosis should be used. Fetal and uterine status should be monitored as well as end-tidal CO₂ and maternal arterial blood gases. To enter the abdomen an open technique should be used. Aortocaval compression should be avoided. Finally, low pneumoperitoneal pressures (<12 mmHg) should be used.

Practical approach

So how should we address practically the pregnant patient that needs surgery during pregnancy for non-obstetric reasons?

Ideally, surgery should be performed during the 2nd trimester. A laparoscopy is possible. Patients should always receive acid aspiration prophylaxis. Left lateral tilt is required to avoid aortocaval compression. The FHR should be measured continuously.

Whenever feasible, a regional technique should be used. However, if general anesthesia is mandatory a rapid sequence induction is required: adequate denitrogenation, cricoid pressure, a rapid sequence induction is required: adequate denitrogenation, cricoid pressure, and maternal arterial blood gases. T o enter the extradural venous plexus in the supine and lateral positions, as determined by magnetic resonance imaging. Br J Anaesth 1997;78:317-9.


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Address reprint requests to: M. Van de Velde, MD, PhD, Director Obstetric Anesthesia and Extra Muros Anesthesia, Department of Anesthesiology, University Hospitals Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium. E-mail: marc.vandevelde@uz.kuleuven.ac.be
Maternal haemorrhage

M. Walfish*, A. Neuman and D. Wlody

SUNY Downstate Medical Center, 450 Clarkson Ave., Box 6, Brooklyn, NY 11203, USA

*Corresponding author. E-mail: menachem.walfish@downstate.edu

Maternal haemorrhage is the leading cause of preventable maternal death worldwide and encompasses antepartum, intrapartum, and postpartum bleeding. This review highlights factors that predispose to severe bleeding, its management, and the most recent treatment and guidelines. Advances in obstetric care have provided physicians with the diagnostic tools to detect, anticipate, and prevent severe life-threatening maternal haemorrhage in most patients who have had prenatal care. In an optimal setting, patients at high risk for haemorrhage are referred to tertiary care centres where multidisciplinary teams are prepared to care for and deal with known potential complications. However, even with the best prenatal care, unexpected haemorrhage occurs. The first step in management is stabilization of haemodynamic status, which involves securing large bore i.v. access, invasive monitoring, and aggressive fluid management and transfusion therapy. Care for the patient with maternal bleeding should follow an algorithm that goes through a rapid and successive sequence of medical and surgical approaches to stem bleeding and decrease morbidity and mortality. With the addition of potent uterotonic agents and the advent of minimally invasive interventional radiological techniques such as angiographic embolization and arterial ligation, definitive yet conservative management is now possible in an attempt to avoid hysterectomy in patients with severe peripartum bleeding. If these interventions are inadequate to control the bleeding, the decision to proceed to hysterectomy must be made expeditiously. Recombinant factor VIIa is a relatively new treatment that could prove useful for severe coagulopathy and intractable bleeding.

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Keywords: anaesthesia, obstetric; anaesthetic techniques; complications, haemorrhage; uterus, blood flow

Obstetric haemorrhage is the single most significant cause of maternal mortality worldwide accounting for 25–30% of all maternal deaths. From 1991 to 1999, 17% of pregnancy-related deaths in the USA were due to haemorrhage. The most recent report of the Confidential Enquiries into Maternal Deaths in the UK showed that 17 of 132 direct deaths were due to haemorrhage. Life-threatening postpartum haemorrhage (PPH) occurs in ~1:1000 deliveries in the developed world. Serious morbidity resulting from haemorrhage includes adult respiratory distress syndrome, coagulopathy, shock, loss of fertility, and pituitary necrosis. Although the risk of dying from pregnancy decreased dramatically during the last century, 60–90% of deaths from PPH are potentially preventable with better medical care.

This review covers the aetiology, evaluation, and management of maternal haemorrhage.

Definition

A variety of definitions for PPH have been proposed, yet no single satisfactory definition exists. The World Health Organization defines PPH as blood loss ≥500 ml in the first 24 h after delivery. However, there is evidence that 500 ml is actually normal blood loss after vaginal delivery and 1000 ml after Caesarean section with little clinical relevance. Practitioners are poorly trained in estimating blood loss leading to an inaccurate and often underestimated value. Another popular definition of haemorrhage is a 10% decrease in either the haemoglobin or the haematocrit, but determinations of these values are often delayed and might not reflect the patient’s current haemodynamic status. Commonly, PPH is diagnosed when the amount of bleeding exceeds the practitioner’s estimates of ‘normal’. Clinical signs and symptoms of blood loss including weakness, sweating, and tachycardia might not
occur until 15–25% of total blood volume is lost with haemodynamic collapse occurring only at losses between 35% and 45%. Given the wide range of definitions applied to maternal haemorrhage and their limitations, it is important to combine the clinical presentation and objective data, while keeping in mind the probability of concealed bleeding within the uterus, peritoneal cavity, and retroperitoneal space, and the relative masking of haemodynamic signs of haemorrhagic shock (Table 1) due to the physiological adaptations of pregnancy.9

### Antepartum haemorrhage

Antepartum haemorrhage is defined as bleeding from the genital tract after 24 weeks of gestation and has an incidence of 2–5% of all pregnancies beyond 24 weeks.11 The causes of antepartum haemorrhage range from cervicitis to placental abnormalities, most commonly placental praevia or placental abruption.35 A proactive approach should be used for patients at high risk for haemorrhage since preoperative preparedness can improve outcome.17 Complications of antepartum haemorrhage include maternal shock, a greater risk of premature delivery, fetal hypoxia, and sudden fetal death, making antepartum haemorrhage an even greater risk to the fetus than to the mother.11

### Placental abruption

Haemorrhage arising from premature separation of a normally situated placenta is known as placental abruption (Table 2).11 Abruption complicates about 1% of pregnancies and is the leading cause of vaginal bleeding in the latter half of pregnancy. The classic presentation consists of vaginal bleeding, uterine tenderness, and increased uterine activity.43 Known risk factors include: hypertension, pre-eclampsia, advanced maternal age, multiparity, maternal/paternal tobacco use, cocaine use, trauma, premature rupture of membranes, chorioamnionitis, and prior abortion.35 The diagnosis of abruption is made clinically with ultrasound confirmation in certain cases.11 The maternal effect of abruption depends primarily on its severity, but its effect on the fetus is determined by both its severity and the gestational age at which it occurs.43 In cases of concealed abruption, vaginal bleeding can be absent, and an underestimation of maternal hypovolaemia can occur.35 The management of placental abruption, including the timing and route of delivery, depends on the degree of maternal and fetal compromise, presentation, and gestational age.43 The major complications of abruption include haemorrhagic shock, acute renal failure, coagulopathy, and fetal demise.35 Abruption is the most common cause of disseminated intravascular coagulation in pregnancy.43 Epidural analgesia can be offered to a patient with partial abruption as long as coagulation and volume status are considered.35 Most urgent cases of placental abruption, with a non-reassuring fetal heart rate, are performed under general anaesthesia. After delivery, the patient should be monitored closely due to the risk of persistent haemorrhage from uterine atony or coagulopathy.43
Uterine rupture

Uterine rupture is tearing of the uterine wall during pregnancy or delivery.\textsuperscript{51} It remains one of the most life-threatening emergencies in obstetrics, as it is associated with high maternal and perinatal morbidity and mortality.\textsuperscript{36} Although most cases result from rupture of a previous Caesarean scar and are diagnosed within a hospital setting, the primigravid uterus is not immune to spontaneous rupture and it must be considered in all women, regardless of parity.\textsuperscript{52} Other risk factors include prior uterine surgery, trauma, uterine anomalies, dystocia, use of uterotonic drugs, and abnormal placentation. Clinical presentation can vary from subtle findings, such as uterine tenderness and non-reassuring fetal heart patterns, to severe localized abdominal pain and a rapid onset of maternal hypovolaemic shock.\textsuperscript{36} Prompt recognition of uterine rupture leading to surgical intervention is critical in influencing perinatal and maternal morbidity and mortality.\textsuperscript{51}

Placenta praevia

Placenta praevia occurs when the placenta is totally or partially inserted in the lower uterine segment (Fig. 1). Conditions associated with placenta praevia have a common element of prior uterine trauma and include multiparity, advanced maternal age, previous C-section or other uterine surgery, and prior praevia.\textsuperscript{35} The classic sign of placenta praevia is painless vaginal bleeding during the second or third trimester. The initial bleed is often mild. Diagnosis is usually made by ultrasound, followed by planned management by the obstetric service based on severity and fetal maturity.\textsuperscript{11} Ultimately, Caesarean section is the recommended mode of delivery. Patients with placenta praevia are at a significantly increased risk for high intraoperative blood loss due to the possibility of the obstetrician incising through the placenta and the increased risk for placenta accreta.\textsuperscript{49} In addition, the uterine site of abnormal implantation does not contract as effectively as a normal uterine segment.\textsuperscript{4} Although there is no consensus on the use of general vs regional anaesthesia in patients with placenta praevia, and each case can be decided only after a complete evaluation, Bonner and colleagues\textsuperscript{7} determined that most obstetric anesthesiologists preferred neuraxial anaesthesia over general anaesthesia in both elective and emergency situations.

Postpartum haemorrhage

PPH can be categorized as an abnormality of one or more of the following: uterine tone (uterine atony), retained tissue (placental tissue or blood clots), trauma (genital tract lacerations), or coagulation (coagulopathy).\textsuperscript{17} Primary PPH occurs during the first 24 h and is more likely to result in maternal morbidity and mortality while secondary haemorrhage refers to haemorrhage 24 h to 6 weeks after delivery.\textsuperscript{35} Prevention of PPH relies on active management, at the time of delivery, including administration of prophylactic uterotonic agents, early cord clamping, and controlled cord traction of the umbilical cord at placental delivery.\textsuperscript{18} Although many risk factors have been associated with PPH, it often occurs without warning. A management protocol for the diagnosis and treatment of PPH should be available at every delivery unit.\textsuperscript{10} A notable example of this implementation is the interdisciplinary protocol developed by New York State Department of Health in conjunction with American College of Obstetricians and Gynecologists (ACOG).\textsuperscript{39}

Risk factors for PPH

Uterine atony

Uterine atony, defined as the lack of efficient uterine contractility after placental separation, is the most common cause of PPH and complicates \( \sim 1 \) in 20 deliveries.\textsuperscript{10} Risk factors for uterine atony include conditions in which the uterus is overdistended (polyhydramnios, multiple gestation, and macrosomia), fatigued (prolonged labour, chorioamnionitis), or unable to contract due to tocolytics or general anaesthesia.\textsuperscript{18} Risk factors for the development of uterine atony were quantitated in a study by Rouse and colleagues\textsuperscript{46} who examined more than 23 000 deliveries in 13 university centres. The overall incidence of atony was 6\%. Multiple gestation [odds ratio (OR) 2.40], Hispanic ethnicity (OR 2.21), labour induction \( \geq 18 \text{ h} \) (OR 2.23), and birth weight \( \geq 4500 \text{ g} \) (OR 2.05) were all significant predictors of atony. However, two-thirds of the women studied had no risk factors, yet they accounted for more than half of the cases of uterine atony. In the author’s words: ‘vigilance and preparation for this potential emergency are necessary in all women undergoing Cesarean delivery’.\textsuperscript{46} The most common physical findings in patients with uterine atony are a soft, boggy uterus and vaginal bleeding. An engorged atomic uterus can contain more than 1 litre of blood and unrecognized intrauterine bleeding can manifest as haemodynamic instability secondary to hypovolaemia.\textsuperscript{35} Initial treatment of uterine atony entails discontinuation of those agents, for example, inhalation anaesthetics, which often contribute to atony, emptying of the bladder, bimanual compression, uterine massage, and uterotonic agents. These procedures can diminish bleeding, expel blood and clots, and allow time for further pharmacological or surgical measures to be implemented.\textsuperscript{27}

Abnormal placentation

Abnormal placentation refers to abnormal attachment of the placenta to the uterine wall and includes accreta, increta, and percreta, depending on the extent of uterine invasion.\textsuperscript{4} Abnormal placentation can result in massive haemorrhage and along with uterine atony is the most common cause of postpartum hysterectomy.\textsuperscript{24} Important risk factors are the presence of placenta praevia and a
history of prior Caesarean deliveries. A markedly increased risk of placenta accreta is associated with an increasing number of prior Caesarean deliveries (Table 3), with and without placenta praevia. Antenatal diagnosis of placenta accreta can be made by ultrasound or MRI and facilitates effective planning. If the diagnosis or a strong suspicion is formed before delivery, ACOG suggests a number of preparatory measures including patient counselling regarding the likelihood of hysterectomy and

Table 3 Risk of placenta accreta after previous Caesarean section

<table>
<thead>
<tr>
<th>Number of previous Caesareans</th>
<th>Risk of placenta accreta (%)</th>
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<tr>
<td>0</td>
<td>1.9</td>
</tr>
<tr>
<td>1</td>
<td>15.6</td>
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<td>5</td>
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</table>
transfusion, availability of adequate personnel, blood products, cell salvage, and prior multidisciplinary assessment. Although the extent of surgical management depends on the extent of the abnormal attachment, attempts to separate the placenta can result in massive haemorrhage, and a prompt decision to proceed to hysterectomy without delay enhances the likelihood of an optimal outcome.

**Obstetric trauma**

The most common injuries at delivery are lacerations and haematomas of the perineum, vagina, and cervix. A majority of the cases are minor, but some injuries are associated with significant, immediate, or delayed haemorrhage. Risk factors for injuring the pelvic vasculature and haematoma formation include null parity, episiotomy, advanced maternal age, operative delivery, breech presentation, multiple gestation, and high birth weight. Although common presenting signs and symptoms are based on the location of the injury and include a rectal or vaginal mass, discolouration, and perineal pain or pressure, the clinical manifestations of the injuries are primarily the result of blood loss which can be significant before the development of signs and symptoms of hypovolaemia. This can be especially true with respect to retroperitoneal bleeding. Most retroperitoneal haematoma present within 24 h of delivery and can be accompanied by fever, ileus, thigh pain, and leg oedema. Conservative management involving observation, ice, pressure, and analgesics should be limited to those patients with small pelvic haematoma that are stable in size, with no evidence of haemodynamic compromise. Otherwise, surgical exploration, evacuation, and ligation of vessels should be performed in a controlled setting in order to avoid the known severe complications of infection, sepsis, pressure necrosis, profuse haemorrhage, and death. The choice of anaesthetic technique depends on the affected area, surgical requirements, physical status of the patient, and urgency of the procedure.

**Coagulopathy**

Although the majority of cases of PPH are related to the primary obstetric pathology and the cause can be easily identified, the possibility of an inherited bleeding disorder should be considered in situations of unexplained and recurrent haemorrhage. PPH can be the first indication of von Willebrand’s disease (VWD) and should be considered. VWD is a result of a deficiency in, or a dysfunction of, a multimeric protein named von Willebrand factor (VWF). VWF plays an important role in haemostasis by facilitating platelet adhesion and acting as a carrier protein for coagulation factor VIII, thereby increasing the procoagulant activity of factor VIII. Other, less common, bleeding disorders associated with PPH include deficiencies in prothrombin, fibrinogen, and factors V, VII, X, and XI. Screening women with a history of menorrhagia is an important strategy in identifying patients with these disorders, thereby reducing PPH and maternal mortality.

**Management**

**General principles**

Although specific interventions will be required depending on the underlying cause of the haemorrhage, several basic steps are essential in the initial and continued management of any patient with obstetric haemorrhage. Bonnar describes a five-step management plan for massive obstetric haemorrhage: (i) organization of the multidisciplinary team; (ii) restoration of blood volume via large bore access using fluid and blood; (iii) correction of defective coagulation with blood products and factors; (iv) evaluation of response to treatment by haemodynamic and laboratory assessment; and (v) remediating of the underlying cause of the bleeding. Another therapeutic goal in the treatment of severe PPH is avoidance of myocardial ischaemia by increasing the myocardial supply-demand ratio. A significant percentage of parturients with haemorrhagic shock experienced electrocardiographic signs of ischaemia and decreased contractility that correlated with the severity of haemorrhage. Given that most maternal morbidity and mortality is due to underestimation of blood loss, inadequate volume replacement, and delay in intervention, a management protocol for treatment of obstetric haemorrhage should be available at every delivery unit that initiates a sequence of conservative and operative interventions. Success of each treatment should be rapidly assessed with the swift institution of the next intervention if it has failed. Prompt communication between anaesthesiology, obstetric–gynaecology, nursing, laboratory, and blood bank is essential for effective evaluation and management of excessive blood loss.
Conservative treatment of maternal haemorrhage

Oxytocin is routinely used to maintain uterine tone during Caesarean section (Table 4). Typical concentrations are 20–40 units litre⁻¹, administered at 500–1000 ml h⁻¹. At least one study suggested that a concentration of 160 units litre⁻¹ administered at 1000 ml h⁻¹ significantly decreased the need for further uterotonics, with no increase in the incidence of hypotension. The use of bolus doses of oxytocin is controversial; Svanström and colleagues demonstrated that a 10 unit bolus during Caesarean section led to significant hypotension and electrocardiographic evidence of myocardial ischaemia. However, Carvalho and colleagues calculated the ED₉₀ for bolus oxytocin to be 0.35 units, with a calculated response rate of 100% when 1 unit was administered as a bolus, with no hypotension at this dose.

Ergot alkaloids such as ergometrine or its derivative methylergonovine (methylergometrine, Methergine) are commonly used when the response to oxytocin is insufficient. The action of these agents is mediated through alpha-adrenergic receptors, and as such, their use is associated with hypertension, especially when phenylephrine has been administered previously. Coronary artery vasospasm leading to myocardial ischaemia or even myocardial infarction has been reported. Nausea and vomiting are common.

There is increasing recognition of the usefulness of prostaglandins for the treatment of uterine atony. 15-Methyl PGF₂α (Hemabate) can be administered i.m. or directly into the uterus, in 250 µg doses every 15 min, up to a maximum of 2 mg. Hypertension and diarrhoea are not uncommon. Increases in bronchial tone are also common, and can be life-threatening, especially in patients with reactive airway disease.

Misoprostol (Cytotec), a PGE₁ analogue originally marketed as a gastric cytoprotective agent for patients receiving non-steroidal anti-inflammatory drug therapy, and more recently as an abortifacient, has also shown great promise as a uterotonic agent. In 14 women with uncontrollable obstetric haemorrhage, rectal administration of 1000 µg led to sustained uterine contraction and control of haemorrhage within 3 min. The buccal administration of misoprostol before elective Caesarean section has been described; while blood loss was unchanged, a 200 µg dose inserted in the buccal space immediately before Caesarean section reduced the need for additional uterotonic agents by almost 40%.

Invasive treatment of maternal haemorrhage

Regardless of the cause of obstetric haemorrhage, conservative measures might fail to control bleeding. In these cases, invasive procedures must be performed promptly to avoid severe morbidity and mortality. Options with their respective success rates include intravascular balloon tamponade (84%), uterine compression sutures (92%), angiographic arterial embolization (91%), arterial ligation (85%), and hysterectomy. It is recommended that obstetricians consider all available interventions in order to stop the haemorrhage and that all hospitals with delivery units should aim to provide an emergency interventional radiology service, as these procedures have the potential to save the lives of patients with catastrophic PPH. There are no randomized controlled studies on the various treatment methods and the success rate of one method has not been shown to be better than another in the management of severe PPH.

Balloon tamponade

Uterine packing procedures have been used for many years in order to control PPH. Roller gauze packs were originally used, but due to their potential traumatic placement and concern for concealed ongoing haemorrhage, the use of balloon tamponade has been favoured more recently. Although a variety of balloon devices have proven successful, the Sengstaken-Blakemore oesophageal catheter is the most frequently reported. The device can be deployed quickly, requires minimal analgesia for both insertion and removal, and it preserves fertility. It can be used to perform a ‘tamponade test’ which arrests the bleeding in most women and allows the obstetrician to identify which patients will require surgical intervention.

The catheter is inserted in the uterine cavity and the balloon is filled with warm sterile water or saline until the uterus is firm on abdominal palpation and until the bleeding is arrested. If little or no bleeding is observed via the cervix or through the lumen of the catheter, the tamponade test is considered positive and laparotomy is avoided. Ongoing bleeding would require surgical exploration.

Although intrauterine balloon tamponade has not been proven superior to other methods of controlling postpartum bleeding, it is the least invasive, most rapid, and lacks significant complications, thus making it a logical first step in PPH management.

Uterine compression sutures

Uterine compression sutures function in a manner similar to manual compression. They have been used as an
adjunctive intervention aimed at maintaining uterine contractility through tamponade. They are most useful in cases of refractory uterine atony but have also been used in cases of retained placenta and coagulopathy. Advantages include a high success rate, ease of placement at the time of laparotomy, and fertility preservation. Disadvantages include the need for laparotomy and usually hysterotomy and reported complications including uterine wall erosion, uterine necrosis, and pyometra. Modifications of uterine compression sutures such as the U-sutures have evolved leading to a reduction in these complications. Uterine compression sutures are more likely to successfully arrest bleeding, if bimanual compression of the uterus achieves homeostasis. The specific placement of compression sutures and their location requires operator judgement. If profuse bleeding is noted from the placental attachment site, placental bed sutures can be used to ligate vessels.

**Angiographic arterial embolization**

The uterine arteries, which are branches of the anterior trunk of the internal iliac arteries, provide the primary blood supply of the uterus. The ovarian arteries also contribute significantly to uterine blood flow during pregnancy. Arterial embolization of these vessels is of significant value in treating obstetric haemorrhage. Embolization requires fluoroscopic guidance and the expertise of an interventional radiologist. Using angiography, the radiologist can identify the vessels responsible for the bleeding and embolize them. The patient must be stable enough for transfer to a radiological suite and should be monitored at all times with the ability to proceed to surgical intervention should the patient become unstable. Angiographic occlusion balloon catheters can be placed to occlude the hypogastric or common iliac arteries as a temporizing measure. Prophylactic placement of arterial catheters for cases with high risk of haemorrhage such as placenta praevia and accreta can allow rapid occlusion of these vessels if necessary. Several studies have noted that clotting disorders improve rapidly after embolization possibly due to the facilitation of uterine contraction that leads to a secondary liberation of procoagulant factors into circulation. Success rates for embolization are reported as high as 85–95%. Reported complications of this procedure include fever, vascular perforation, lower extremity ischaemia, bladder, and rectal wall necrosis. Flow through the vessels return over time, preserving both the uterus and fertility. Embolization therapy is unlikely to be successful after arterial ligation.

**Arterial ligation**

Surgical ligation of the uterine, ovarian, and internal iliac arteries can be useful when other methods to control maternal haemorrhage have failed. Ligation results in a decrease in pulse pressure distal to the ligature, thereby more readily achieving haemostasis through clot formation. Bilateral ligation of the uterine vessels is a more attractive option than internal iliac artery ligation because the uterine arteries are easily accessible, the procedure is more successful, and the field of dissection generally is not near the ureters and the iliac veins. Stepwise uterine devascularization has been described whereby uterine, tubal branches of the ovarian, and finally internal iliac arteries are ligated. In a study of 103 patients, uterine devascularization was 100% effective and hysterectomy was not needed in any case. But ligation techniques are technically challenging and time-consuming requiring both surgical expertise and a haemodynamically stable patient. Risks include lower extremity ischaemia, neuropathy, intestinal occlusion, and peripheral nerve ischaemia. When it is successful, surgical ligation permits preservation of fertility.

**Hysterectomy**

Hysterectomy is often the definitive treatment for PPH with the most common indications being uterine atony and placenta accreta. Peripartum hysterectomy is estimated to occur ~0.8 per 1000 deliveries. If alternative interventions fail, hysterectomy should not be delayed in patients who continue to bleed. Prompt control of uterine haemorrhage is vital to decrease morbidity and prevent death, as continued blood loss can lead to disseminated intravascular coagulation. The operative technique and preparation for hysterectomy depend on the timing and indication of the procedure. Peripartum hysterectomy can be a technically challenging operation due to the enlarged uterus, engorged vessels, and oedematous tissues. Preoperative preparation, including standardized protocols, availability of trained staff, and immediate access to equipment, is essential to minimizing morbidity. Complications of hysterectomy include operative site infection, bladder and ureteral injuries, further intra-abdominal haemorrhage, and injury to other organs.

**Transfusion therapy**

In healthy young women, adequate oxygen delivery can be maintained at haemoglobin levels as low as 6–7 g dl⁻¹. Nevertheless, in the setting of haemodynamic instability, coexisting cardiopulmonary disease, or coagulopathy, administration of blood products is usually necessary. Optimal therapy will be guided by laboratory determinations of haemoglobin concentration, platelet count, and coagulation function. In life-threatening situations, however, empiric treatment with packed red blood cells (RBCs), platelets, fresh-frozen plasma, or cryoprecipitate might be necessary. There is no current uniform consensus on the optimal ratio of blood products to transfuse into severely bleeding patients and prior recommendations for fresh-frozen plasma:RBC and platelets:RBC transfusion ratios range from 1:10 to 2:3 and 6:10 to 12:10, respectively. Studies in patients with severe
haemorrhage from combat-related trauma requiring massive transfusion have found that a high 1:1:4 plasma:RBC ratio is independently associated with significantly improved survival by decreasing death from haemorrhage. On the basis of these studies, it is now recommended by the US Army Surgeon General to transfuse plasma and RBCs in a 1:1 ratio in patients with significant trauma. Considering the similarities between maternal haemorrhage and major haemorrhage due to trauma with regard to the accompanying hypothermia, acidosis, and coagulopathy, it might be appropriate to extend these recommendations to massive PPH, although definitive data are not available.44

There is considerable interest in the use of intraoperative blood salvage during significant maternal haemorrhage. One obstacle to the use of cell salvage has been the concern that reinfusion of the as yet undetermined causative agent of amniotic fluid embolism syndrome might precipitate that life-threatening complication. Although at least one study of 139 women undergoing cell salvage and reinfusion during Caesarean section suggests that this modality can be used with minimal risk, others have suggested that the number of patients studied to date is insufficient to provide a blanket endorsement of the safety of this practice.45 If cell salvage is to be utilized, it is recommended that collection should be initiated only after the surgical field has been irrigated and gross contamination with amniotic fluid has been eliminated. The use of a leucocyte depletion filter during reinfusion of processed blood might enhance the margin of safety.

Recombinant activated factor VII
Recombinant activated factor VII (rFVIIa) (Novoseven) is a synthetic vitamin K-dependent glycoprotein that has been administered for haemorrhage unresponsive to conventional blood product resuscitation. FVIIa works via activation of the extrinsic pathway of the coagulation cascade leading to an enhanced generation of thrombin and a stable fibrin plug at the site of injury. It was originally developed for the treatment of bleeding in patients with haemophilia A or B with inhibitors to factor VIII or IX.53 rFVII has also been successfully used to prevent or control bleeding in several other conditions including thrombocytopenia, platelet function disorders, impaired liver function, and extensive surgery and severe trauma with massive bleeding.10 Although no randomized controlled studies have been published on the use of FVIIa in PPH, case reports have suggested great efficacy in helping to control massive obstetric bleeding.23 Consideration for the use of rFVII in PPH must take into account efficacy, side-effects including increased risk of thromboembolism, and costs of rFVII vs other treatment.53 The American Society of Anesthesiologists recommends consideration of rFVIIa when ‘traditional well tested options for treating microvascular bleeding have been exhausted’.3 After failure of all medical and surgical treatments and after multiple transfusions of RBCs and fresh-frozen plasma but before hysterectomy, Welsh and colleagues53 suggest an rFVIIa dose of 90 μg kg⁻¹ followed by a second identical dose if no response is seen after 20 min.

Table 5 Managing maternal haemorrhage algorithm.39 *Most commonly used dose for haemorrhage

<table>
<thead>
<tr>
<th>Vital signs</th>
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<tbody>
<tr>
<td>Normal vitals do not always assure patient stability</td>
</tr>
<tr>
<td>Control the airway</td>
</tr>
<tr>
<td>Maintain oxygenation and ventilation</td>
</tr>
<tr>
<td>Support the circulation</td>
</tr>
<tr>
<td>Pallor, delayed capillary refill, and decreased urine output can indicate compromised blood volume without change in arterial pressure or heart rate</td>
</tr>
<tr>
<td>Decreased urine output, hypotension, and tachycardia may be late signs of compromise</td>
</tr>
</tbody>
</table>

Infusions
Start second large bore (16 G or larger)
Salt solutions replace blood loss at 3:1
Volume expanders replace blood loss at 1:1 (albumin, hetastarch, dextran)
Packed RBCs
Coagulation factors
Warm blood products and infusions to prevent hypothermia, coagulopathy, and arrhythmias
Medications for uterine atony
Oxytocin
20 mg per rectum every 2 h (avoid with hypotension)
Methylergometrine (Methergine)
0.2 mg i.m. every 2–4 h up to 5 doses (avoid with hypertension)
Prostaglandin F₂ Alpha (Hemabate)
10–40 units in 1 litre saline i.v. rapid infusion
*M30–40 units litre⁻¹ is the most commonly used dose for haemorrhage
Prostaglandin E₂ suppositories (Dinoprostone, Prostin E₂)
0.2 mg i.m. or intramyometrial, repeat every 20–90 min maximum
Misooprostol (Cytotec)
20 mg per rectum every 2 h (avoid with hypotension)
Misoprostol (Cytotec)
1000 μg per rectum or sublingual (10 100 μg tabs or 5 200 μg tabs)
Surgical interventions
May be life-saving measure and should not be delayed

Conclusion
Anaesthesiologists play a vital role in the care and management of patients with peripartum haemorrhage. As members of the multidisciplinary team, anaesthesiologists should utilize their expertise in fluid management, transfusion therapy, and critical care to prevent and treat the catastrophic events that accompany severe bleeding. Table 5 is an example of an algorithm that can serve as a guide to the management and treatment of peripartum haemorrhage. Although guides and protocols such as this one are useful, they cannot take the place of experienced and trained physicians in assessing and managing maternal haemorrhage. With the recent advent of minimally invasive treatments and drugs to temporize bleeding, physicians now have more options at their disposal. Therefore, vigilance and an aggressive approach (Table 5) are keys in reducing morbidity and mortality and ensuring the best possible outcome for patients with maternal haemorrhage.
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Expanded Executive Summary

By
NCCEMD
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List of abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
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<tr>
<td><strong>Provinces</strong></td>
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<tr>
<td>EC</td>
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</tr>
<tr>
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<td>Free State</td>
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<td>Acute collapse and embolism</td>
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<tr>
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<td>Acquired Immune Deficiency Syndrome</td>
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<td>Antepartum haemorrhage</td>
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<tr>
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<tr>
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<tr>
<td>MD</td>
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</tr>
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<td>Non-pregnancy related infections</td>
</tr>
<tr>
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<td>Postpartum haemorrhage</td>
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<tr>
<td>PRS</td>
<td>Pregnancy related sepsis</td>
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<tr>
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<td>Tuberculosis</td>
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<td>Unknown</td>
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<td>Urinary tract infection</td>
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<td>Community Health Centre</td>
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<td>NCCEMD</td>
<td>National Committee on Confidential Enquiries into Maternal Deaths</td>
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<td>TOP</td>
<td>Termination of pregnancy</td>
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</table>
1. Summary of findings and recommendations

- Maternal deaths are defined as “deaths of women while pregnant or within 42 days of termination of pregnancy from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes”\(^1\).

- Confidential enquiries into maternal deaths are a “systematic multidisciplinary anonymous investigation of all or a representative sample of maternal deaths occurring at an area, region (state) or national level, which identifies the numbers, causes and avoidable or remediable factors, associated with them. Through the lessons learnt from each woman’s death, and through aggregating the data, confidential enquiries provide evidence of where the main problems in overcoming maternal mortality lie and an analysis of what can be done in practical terms, and highlight the key areas requiring recommendations for health sector and community action as well as guidelines for improving clinical outcomes”\(^1\).

- In this triennium (2005-2007) there has been a 20.1% increase in the number of deaths reported compared with the previous triennium (2002-2004).

- The “big five” causes of maternal death have remained the same, namely non-pregnancy related infections – mainly AIDS (43.7%), complications of hypertension (15.7%), obstetric haemorrhage (antepartum and postpartum haemorrhage; 12.4%), pregnancy-related sepsis (9.0%) and pre-existing maternal disease (6.0%).

- There has been a significant decrease (14%) in the institutional Maternal Mortality Ratio (MMR) for complications of hypertension. There was a significant increase (21%) in deaths due to non-pregnancy related infections. There were no other significant changes in the disease pattern.

- Women less than 20 years of age were at greater risk of dying due to complications of hypertension whereas women 35 years and older were at greater risk of dying of obstetric haemorrhage, ectopic pregnancies, embolism, acute collapse and pre-existing medical disease.

- Non-attendance and delayed attendance at the health institutions were the most common patient orientated problems.

- Poor transport facilities, lack of health care facilities and lack of appropriately trained staff were the major administrative problems.

- The most frequent health care provider avoidable factors were failure to follow standard protocols and poor problem recognition and initial assessment.

- Assessors thought 38.4% of the deaths were clearly avoidable within the health care system (patient orientated factors being excluded). Complications of hypertension, obstetric haemorrhage, pregnancy related sepsis and non-pregnancy related infections were responsible for 4 out of 5 of avoidable deaths.

- Recommendations concern four main areas (knowledge development, quality of care and coverage of reproductive health services, establishing norms and standards and community involvements):
  - Improving health care provider knowledge and skills in providing emergency care and ensuring adequate screening and treatment of the major causes of maternal death.
  - Improving quality and coverage of reproductive health services, namely contraceptive and termination of pregnancy services.
  - Management provision of staffing and equipment norms, transport and availability of blood for transfusion.
  - Community involvement and empowerment regarding maternal, neonatal and reproductive health in general.
2. Introduction

Confidential enquiries into maternal deaths (CEMD) can be defined as a systematic multidisciplinary anonymous investigation of all or a representative sample of maternal deaths occurring at an area, region (state) or national level which identifies the numbers, causes and avoidable or remediable factors associated with them. Through the lessons learnt from each woman’s death, and through aggregating the data, confidential enquiries provide evidence of where the main problems in overcoming maternal mortality lie and an analysis of what can be done in practical terms, and highlight the key areas requiring recommendations for health sector and community action as well as guidelines for improving clinical outcomes.¹

The Confidential Enquiries system of recording and analysing maternal deaths has been in operation in South Africa since 1 October 1997. The first comprehensive report into maternal deaths was published in October 1999, and dealt in detail with maternal deaths occurring during 1998². The second comprehensive report covered the triennium 1999-2001³, and the third comprehensive report covered 2002-2004⁴. All have described the magnitude of the problem of maternal deaths, the pattern of disease causing maternal deaths, the avoidable factors, missed opportunities and substandard care related to these deaths and made recommendations concerning ways of decreasing the number of maternal deaths. This is the fourth comprehensive report in the series and deals with the triennium 2005-2007.

The definitions used in this report are the same as those used in all the “Saving Mothers” reports. Data used for this report consist of the maternal deaths that occurred from 1st January 2005 to 31st December 2007 and were reported to the National Committee on Confidential Enquiries into Maternal Deaths (NCCEMD) secretariat before 30th April 2008. This cut-off date was selected to enable the report to be written and published in 2008. The data is compared with the data in the previous report.

During 2005-2007, a total of 4077 maternal deaths were reported (including coincidental deaths), up from the 3406 reported in the 2002-2004 triennium. Figure
1 illustrates the number of cases reported per province from 1998-2007. As expected the most populous provinces have the most maternal deaths.

In most provinces there has been an increase in the number of maternal deaths reported. The increase in deaths reported is probably due to a combination of better reporting and an actual increase in deaths.

Figure 1. Number of maternal deaths reported per province 1998-2007

Estimations of the population based MMR for South Africa vary between 150/100000 live births\(^5\); 181-382/100000 live births (Graham and Newell\(^7\)); 240-400/100000 live births (UN estimates\(^8\)); and 578/100000 live births (2001 Census estimates\(^9\)). CEMD systems are not carefully designed epidemiological surveys like the Demographic and Health Surveys\(^5\) or Burden of Disease Estimates\(^6\) and cannot report an accurate maternal deaths ratio (MMR) for the country or province. Most maternal deaths occurring outside of health institutions are not reported to the NCCEMD. In rural areas it is estimated only between 20% and 66% of maternal deaths occur in health institutions\(^7\). This lack of reporting and therefore information on deaths outside of the health institutions can bias this report. However, the report does give accurate information on the causes of deaths and quality of care within the health institutions.

The National Health Information System collects data on births in institutions, via the District Health Information System (DHIS), but does not record births outside of the
health institutions. An institutional MMR can be calculated using the DHIS data for the number of births and the CEMD data for the number of deaths. This institutional MMR must always be treated with caution as increasing indices might be due to improved reporting or an actual increase in deaths, conversely reducing indices might be due to poor reporting or and an actual reduction in deaths. However, it is useful to compare trends over the years.

3. Demographic data

Table 1 illustrate the differences in the ages at which maternal deaths in various disease categories occur and compare it with the general pregnant population. The data for the general pregnant population was obtained from Statistics South Africa’s Recorded Live Births reports.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>&lt; 20 y</th>
<th>20 – 24 y</th>
<th>25 – 29 y</th>
<th>30 – 34 y</th>
<th>35 – 39 y</th>
<th>40 – 44 y</th>
<th>45+ y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gen. Pop</td>
<td>11.3</td>
<td>29</td>
<td>25.2</td>
<td>19.5</td>
<td>10.7</td>
<td>3.6</td>
<td>0.6</td>
</tr>
<tr>
<td>HT</td>
<td>17.4</td>
<td>23.3</td>
<td>19.3</td>
<td>20.4</td>
<td>13.3</td>
<td>5.3</td>
<td>0.8</td>
</tr>
<tr>
<td>PPH</td>
<td>7.0</td>
<td>11.7</td>
<td>19.6</td>
<td>30.0</td>
<td>21.1</td>
<td>8.1</td>
<td>1.8</td>
</tr>
<tr>
<td>APH</td>
<td>12.0</td>
<td>14.8</td>
<td>15.7</td>
<td>24.1</td>
<td>23.1</td>
<td>7.4</td>
<td>2.8</td>
</tr>
<tr>
<td>EC</td>
<td>3.6</td>
<td>10.9</td>
<td>23.6</td>
<td>27.3</td>
<td>23.6</td>
<td>9.1</td>
<td>1.8</td>
</tr>
<tr>
<td>Ab</td>
<td>5.1</td>
<td>18.4</td>
<td>31.6</td>
<td>21.3</td>
<td>17.6</td>
<td>5.1</td>
<td>0.7</td>
</tr>
<tr>
<td>PRS</td>
<td>12.6</td>
<td>22.9</td>
<td>30.5</td>
<td>17.5</td>
<td>11.2</td>
<td>4.5</td>
<td>0.0</td>
</tr>
<tr>
<td>AA</td>
<td>14.0</td>
<td>30.8</td>
<td>18.7</td>
<td>22.4</td>
<td>8.4</td>
<td>4.7</td>
<td>0.9</td>
</tr>
<tr>
<td>Em</td>
<td>3.5</td>
<td>22.8</td>
<td>15.8</td>
<td>26.3</td>
<td>17.5</td>
<td>14.0</td>
<td>0.0</td>
</tr>
<tr>
<td>AC</td>
<td>10.2</td>
<td>24.2</td>
<td>19.5</td>
<td>18.0</td>
<td>19.5</td>
<td>7.0</td>
<td>0.8</td>
</tr>
<tr>
<td>NPRI</td>
<td>4.3</td>
<td>21.3</td>
<td>35.0</td>
<td>25.7</td>
<td>10.9</td>
<td>2.5</td>
<td>0.1</td>
</tr>
<tr>
<td>AIDS</td>
<td>3.0</td>
<td>19.9</td>
<td>36.2</td>
<td>25.6</td>
<td>12.5</td>
<td>2.5</td>
<td>0.2</td>
</tr>
<tr>
<td>MD</td>
<td>8.9</td>
<td>18.6</td>
<td>29.5</td>
<td>24.1</td>
<td>14.8</td>
<td>3.0</td>
<td>0.8</td>
</tr>
<tr>
<td>Unk</td>
<td>12.1</td>
<td>16.1</td>
<td>28.2</td>
<td>19.5</td>
<td>13.8</td>
<td>7.5</td>
<td>0.0</td>
</tr>
</tbody>
</table>

(AIDS is a subset of NPRI)

There is an excess of maternal deaths due to complications of hypertension occurring in women less than 20 years when compared with the general pregnant population. The percentage of maternal deaths due to antepartum haemorrhage, postpartum haemorrhage, ectopic pregnancies, embolism, acute collapse and pre-existing medical conditions are higher than the general pregnant population in women older than 34 years. Deaths due to non-pregnancy related infections peak at 25-29 years and this peak is mirrored in deaths due to complications of abortion and pregnancy related sepsis following a viable pregnancy.
Level of Care

Figure 2 illustrates the number of maternal deaths occurring at the various levels of care. The deaths occurring outside health facilities are reflected as home deaths are only those that were reported to health institutions or in some cases to state mortuaries.

There is an increase in the number of deaths at all level 1 and 2 institutions, with level 3 institutions declining over time.

Figure 3 gives the distribution of births in the Community Health Centres (CHCs), District, Regional and Provincial Tertiary (Prov. Tert) and National Central (Nat. Cent.) Hospitals. CHCs and district hospitals are classified as level 1 institutions, regional hospitals level 2 institutions and provincial tertiary and national central hospitals as level 3 institutions. From the data below, 59% of all births occur in level 1 institutions, 30% in level 2 institutions and 11% at level 3 institutions.
Figure 3. Distribution of deliveries in South African public institutions (DHIS data)

Figure 4 gives the *institutional* MMR for the levels of care. Even though the majority of deaths occur at level 1 institutions, the Institutional MMR is highest for the Level 3 institutions and the pattern is as expected, with the lowest mortality in the CHCs.

Figure 4. MMR per level of care

There is however considerable variation of the *institutional* MMRs for levels of care per province. (See Figures 5).
The Free State province stands out with its Level 1 Institutional MMR being 198.6, level 2 being 626.1 and level 3 being 2498.4/100000 live births. The Free State province only has 24 level 3 maternity beds, all in one institution. Other provinces like North West, Northern Cape, Eastern Cape and Mpumalanga provinces do not have official level 3 beds. The organisation and distribution of tertiary level beds and hence care needs to be carefully examined.

**HIV testing**

Table 2 shows the HIV status of women who died and Figure 6 shows the percentage of HIV testing of maternal deaths per province. Fifty-nine percent of maternal deaths were tested for HIV infection from 2005-2007, up from 46.3% in the last triennium. Seventy-nine percent of those tested in 2005-2007 were HIV infected. This figure is very similar to that of the last triennium namely 78%. There has been a steady increase in testing, probably reflecting the expansion of the Prevention of Mother to Child Transmission Programme. Although the testing was variable, the total testing shows an improvement. This is likely to improve with further extension of the implementation of the strategic plan.
Table 2. HIV status of maternal deaths 2005-2007 compare with 2002-2004

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>Positive</td>
<td>1884</td>
<td>46.2</td>
</tr>
<tr>
<td>Negative</td>
<td>511</td>
<td>12.5</td>
</tr>
<tr>
<td>Unknown</td>
<td>1682</td>
<td>41.3</td>
</tr>
</tbody>
</table>

Figure 6. Percent HIV testing of maternal deaths per Province: 1999-2007

Antenatal Care
In this report, where the antenatal care status was known, 76.1% of women who died during or within 6 weeks of pregnancy attended antenatal clinics (Table 3). This is considerably lower than the 95% antenatal care attendance given for the general population\(^5\). Health messages promoting early and regular attendance at antenatal care should continue and be strengthened. Antenatal care must be able to identify risk factors and manage them accordingly to prevent maternal deaths. Table 4 shows the antenatal usage in the conditions where antenatal care can potentially make a difference. The majority of women who subsequently died attended antenatal care, giving the health care system a good opportunity to intervene and prevent the death.

Table 3. Antenatal usage 2005-2007

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>Percentage of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Received antenatal care</td>
<td>2601</td>
<td>63.8</td>
</tr>
<tr>
<td>No antenatal care</td>
<td>818</td>
<td>20.1</td>
</tr>
<tr>
<td>Unknown</td>
<td>658</td>
<td>16.1</td>
</tr>
</tbody>
</table>
### Table 4. Antenatal usage in those conditions where antenatal care can make a difference

<table>
<thead>
<tr>
<th>Condition</th>
<th>Attended</th>
<th>Did not attend</th>
<th>% attended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-existing maternal disease</td>
<td>146</td>
<td>46</td>
<td>76.0</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>64</td>
<td>19</td>
<td>77.1</td>
</tr>
<tr>
<td>Non pregnancy related infections</td>
<td>1094</td>
<td>372</td>
<td>74.6</td>
</tr>
<tr>
<td>AIDS</td>
<td>624</td>
<td>174</td>
<td>78.2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>441</td>
<td>105</td>
<td>80.8</td>
</tr>
</tbody>
</table>

### Timing of emergency event leading to maternal death

The postpartum period was the most common period for the problem resulting in death to occur, occurring in 45.4% of cases compared with 9% in early pregnancy, 36.7 antenatally and 8.7% intrapartum. The timing was unknown in 0.2% of cases.
4. Primary obstetric causes of death

Table 5 gives the primary obstetric causes of death and compares it with the 1999-2001 and 2002-2004 trienniums. The top five conditions have remained the same, namely non-pregnancy related infections (43.7%), complications of hypertension (15.7%), obstetric haemorrhage (antepartum and postpartum haemorrhage; 12.4%), pregnancy-related sepsis (9.0%) and pre-existing maternal disease (6.0%).


<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
<td>%</td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>507</td>
<td>20.7</td>
<td>628</td>
<td>19.1</td>
<td>622</td>
<td>15.7</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>240</td>
<td>9.8</td>
<td>313</td>
<td>9.5</td>
<td>383</td>
<td>9.7</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>100</td>
<td>4.1</td>
<td>129</td>
<td>3.9</td>
<td>108</td>
<td>2.7</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>27</td>
<td>1.1</td>
<td>47</td>
<td>1.4</td>
<td>55</td>
<td>1.4</td>
</tr>
<tr>
<td>Abortion</td>
<td>120</td>
<td>4.9</td>
<td>114</td>
<td>3.5</td>
<td>136</td>
<td>3.4</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>210</td>
<td>8.6</td>
<td>274</td>
<td>8.3</td>
<td>223</td>
<td>5.6</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>76</td>
<td>3.1</td>
<td>91</td>
<td>2.8</td>
<td>107</td>
<td>2.7</td>
</tr>
<tr>
<td>Embolism</td>
<td>48</td>
<td>2</td>
<td>64</td>
<td>1.9</td>
<td>57</td>
<td>1.4</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>134</td>
<td>5.5</td>
<td>107</td>
<td>3.2</td>
<td>128</td>
<td>3.2</td>
</tr>
<tr>
<td>Indirect</td>
<td>939</td>
<td>38.4</td>
<td>1430</td>
<td>43.4</td>
<td>1966</td>
<td>49.7</td>
</tr>
<tr>
<td>Non pregnancy related Infections</td>
<td>768</td>
<td>31.4</td>
<td>1246</td>
<td>37.8</td>
<td>1729</td>
<td>43.7</td>
</tr>
<tr>
<td>AIDS</td>
<td>416</td>
<td>17</td>
<td>662</td>
<td>20.1</td>
<td>915</td>
<td>23.1</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>171</td>
<td>7</td>
<td>184</td>
<td>5.6</td>
<td>237</td>
<td>6.0</td>
</tr>
<tr>
<td>Unknown</td>
<td>44</td>
<td>1.8</td>
<td>99</td>
<td>3</td>
<td>174</td>
<td>4.4</td>
</tr>
<tr>
<td>Total</td>
<td>2445</td>
<td>100</td>
<td>3296</td>
<td>100</td>
<td>3959</td>
<td>100</td>
</tr>
<tr>
<td>Coincidental</td>
<td>45</td>
<td>110</td>
<td>118</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 6 gives the details of the indirect deaths for 2005-2007.
### Table 6. Indirect causes of death 2005-2007

<table>
<thead>
<tr>
<th>Indirect causes of death</th>
<th>Total</th>
<th>%</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Sub-category</td>
<td>All deaths</td>
</tr>
<tr>
<td><strong>Non-pregnancy related infections</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>393</td>
<td>22.7</td>
<td>9.6</td>
</tr>
<tr>
<td>AIDS</td>
<td>915</td>
<td>52.9</td>
<td>22.4</td>
</tr>
<tr>
<td>TB</td>
<td>229</td>
<td>13.2</td>
<td>5.6</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>1</td>
<td>0.1</td>
<td>0</td>
</tr>
<tr>
<td>UTI</td>
<td>3</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td>Malaria</td>
<td>16</td>
<td>0.9</td>
<td>0.4</td>
</tr>
<tr>
<td>Meningitis</td>
<td>106</td>
<td>6.1</td>
<td>2.6</td>
</tr>
<tr>
<td>Other</td>
<td>66</td>
<td>3.8</td>
<td>1.6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1729</td>
<td><strong>100.0</strong></td>
<td><strong>43.7</strong></td>
</tr>
<tr>
<td><strong>Pre-existing medical disease</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>97</td>
<td>40.9</td>
<td>2.4</td>
</tr>
<tr>
<td>Endocrine</td>
<td>13</td>
<td>5.5</td>
<td>0.3</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>31</td>
<td>13.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Central Nervous System</td>
<td>21</td>
<td>8.9</td>
<td>0.5</td>
</tr>
<tr>
<td>Respiratory</td>
<td>40</td>
<td>16.9</td>
<td>1</td>
</tr>
<tr>
<td>Haematological</td>
<td>22</td>
<td>9.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Genito-urinary</td>
<td>4</td>
<td>1.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Auto-immune</td>
<td>7</td>
<td>3.0</td>
<td>0.2</td>
</tr>
<tr>
<td>Skeletal</td>
<td>2</td>
<td>0.8</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>237</td>
<td><strong>100.0</strong></td>
<td><strong>5.8</strong></td>
</tr>
<tr>
<td><strong>Grande Total</strong></td>
<td>1966</td>
<td></td>
<td><strong>49.7</strong></td>
</tr>
</tbody>
</table>

Table 7 gives the primary obstetric causes expressed as maternal deaths per 100,000 live births. There has been a significant decrease in the rate of maternal deaths/100,000 live births in complications of hypertension (p<0.01, OR 0.86). A significant decrease was also recorded in deaths due to pregnancy related sepsis (p<0.001, OR 0.80) but this is probably due to cases being misclassified as dying due to AIDS rather than pregnancy related sepsis (see below). Where the calculations were repeated using the same proportion of cases misclassified in 2005-2007 for 2002-2004, there was no difference deaths due to pregnancy related sepsis. There was a 20.9% increase in deaths due to non-pregnancy related infections. The MMR is used here only for comparison and to evaluate trends. The confidential enquiry is not an epidemiological study rather its value is in evaluating the quality of care.
### Table 7. Comparison of institutional Maternal Mortality Ratios per disease category

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>2002-2004</th>
<th>2005-2007</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>27.72</td>
<td>23.92</td>
<td>&lt;0.01; OR 0.86</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>13.82</td>
<td>14.73</td>
<td>NS</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>5.69</td>
<td>4.15</td>
<td>NS</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>2.07</td>
<td>2.12</td>
<td>NS</td>
</tr>
<tr>
<td>Abortion</td>
<td>5.03</td>
<td>5.23</td>
<td>NS</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>12.09</td>
<td>8.58</td>
<td>&lt;0.0001; OR 0.80</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>4.02</td>
<td>4.11</td>
<td>NS</td>
</tr>
<tr>
<td>Embolism</td>
<td>2.82</td>
<td>2.19</td>
<td>NS</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>4.72</td>
<td>4.92</td>
<td>NS</td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pregnancy related Infections</td>
<td>55.00</td>
<td>66.49</td>
<td>&lt;0.0001; OR 1.21</td>
</tr>
<tr>
<td>AIDS</td>
<td>29.22</td>
<td>35.19</td>
<td></td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>8.12</td>
<td>9.11</td>
<td>NS</td>
</tr>
<tr>
<td>Unknown</td>
<td>4.37</td>
<td>6.69</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>145.48</td>
<td>152.25</td>
<td>NS</td>
</tr>
</tbody>
</table>

### The impact of HIV infection on maternal deaths

In the last triennium, the number of women being tested for HIV infection has risen sharply and the true impact of the disease on the pregnant population is now becoming apparent. The disease profile of pregnant women who die and are HIV infected is shown in Tables 8 and 9.

### Table 8. Distribution of HIV positive, negative and unknown in relation to primary causes of death (numbers)

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>HIV +</th>
<th>HIV -</th>
<th>Unk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>111</td>
<td>136</td>
<td>375</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>79</td>
<td>83</td>
<td>221</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>15</td>
<td>21</td>
<td>72</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>6</td>
<td>5</td>
<td>44</td>
</tr>
<tr>
<td>Abortion</td>
<td>40</td>
<td>5</td>
<td>91</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>91</td>
<td>38</td>
<td>94</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>18</td>
<td>19</td>
<td>70</td>
</tr>
<tr>
<td>Embolism</td>
<td>10</td>
<td>21</td>
<td>26</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>31</td>
<td>31</td>
<td>66</td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pregnancy related Infections</td>
<td>1347</td>
<td>55</td>
<td>327</td>
</tr>
<tr>
<td>AIDS</td>
<td>891</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>67</td>
<td>59</td>
<td>111</td>
</tr>
<tr>
<td>Unknown</td>
<td>57</td>
<td>29</td>
<td>88</td>
</tr>
<tr>
<td>Total</td>
<td>1872</td>
<td>502</td>
<td>1585</td>
</tr>
<tr>
<td>Coincidental</td>
<td>12</td>
<td>9</td>
<td>97</td>
</tr>
</tbody>
</table>
Table 9. Distribution of HIV positive, negative and unknown in relation to primary causes of death (percentage)

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>HIV + (%)</th>
<th>HIV – (%)</th>
<th>Unk (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>5.9</td>
<td>27.1</td>
<td>23.7</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>4.2</td>
<td>16.5</td>
<td>13.9</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>0.8</td>
<td>4.2</td>
<td>4.5</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>0.3</td>
<td>1.0</td>
<td>2.8</td>
</tr>
<tr>
<td>Abortion</td>
<td>2.1</td>
<td>1.0</td>
<td>5.7</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>4.9</td>
<td>7.6</td>
<td>5.9</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>1.0</td>
<td>3.8</td>
<td>4.4</td>
</tr>
<tr>
<td>Embolism</td>
<td>0.5</td>
<td>4.2</td>
<td>1.6</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>1.7</td>
<td>6.2</td>
<td>4.2</td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pregnancy related Infections</td>
<td>72.0</td>
<td>11.0</td>
<td>20.6</td>
</tr>
<tr>
<td>AIDS</td>
<td>47.6</td>
<td>0.2</td>
<td>1.5</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>3.6</td>
<td>11.8</td>
<td>7.0</td>
</tr>
<tr>
<td>Unknown</td>
<td>3.0</td>
<td>5.8</td>
<td>5.6</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Most HIV infected women die of non-pregnancy related infections; the only other diseases which are relatively common are hypertension (5.9%), postpartum haemorrhage (4.2%) and pregnancy related sepsis (4.9%). Conversely, most of the HIV negative women die due to direct causes of death, namely hypertension (27.1%), obstetric haemorrhage (20.7%), and sepsis (8.6%).

Table 10 shows the distribution of the sub-categories of non-pregnancy related infection in relation to their HIV status.

Table 10. Distribution of causes of non pregnancy related infections in relation to their HIV status

<table>
<thead>
<tr>
<th>Sub categories</th>
<th>HIV +</th>
<th>HIV -</th>
<th>Unk</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Pneumonia</td>
<td>224</td>
<td>21</td>
<td>148</td>
<td>393</td>
</tr>
<tr>
<td>- AIDS</td>
<td>891</td>
<td>1</td>
<td>23</td>
<td>915</td>
</tr>
<tr>
<td>- TB</td>
<td>138</td>
<td>20</td>
<td>71</td>
<td>229</td>
</tr>
<tr>
<td>- Endocarditis</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>- UTI</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>- Appendicitis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>- Malaria</td>
<td>3</td>
<td>2</td>
<td>11</td>
<td>16</td>
</tr>
<tr>
<td>- Meningitis</td>
<td>56</td>
<td>6</td>
<td>44</td>
<td>106</td>
</tr>
<tr>
<td>- Other</td>
<td>34</td>
<td>5</td>
<td>27</td>
<td>66</td>
</tr>
<tr>
<td>Total</td>
<td>1347</td>
<td>55</td>
<td>327</td>
<td>1729</td>
</tr>
</tbody>
</table>
Note: 24 patients wrongly classified as having AIDS (HIV-/unknown). A further 138 should have been classified as having AIDS as there were diagnosed as having TB.

In examining the group classified has having died due to AIDS the following were found:

- 58 women with AIDS also had had an abortion and 558 died postpartum
- 181 women with AIDS died postpartum and were classified as having septic shock as the final cause of death.
- 16 women with AIDS and dying postpartum had the final cause of death as hypovolaemic shock and had postpartum haemorrhage
- If these cases are re-classified under the direct causes of death, then 255 maternal deaths would be reclassified as direct deaths.

The net effect of this on all the maternal deaths is illustrated in Table 11.

**Table 11. New distribution of primary causes of death following the re-classification of the cases with AIDS having concomitant direct obstetric diseases**

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>HIV + (n)</th>
<th>HIV – (n)</th>
<th>Unk (n)</th>
<th>Total (n)</th>
<th>New %</th>
<th>Current %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>111</td>
<td>136</td>
<td>375</td>
<td>622</td>
<td>15.7</td>
<td>15.7</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>95</td>
<td>83</td>
<td>221</td>
<td>399</td>
<td>10.1</td>
<td>9.7</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>15</td>
<td>21</td>
<td>72</td>
<td>108</td>
<td>2.7</td>
<td>2.7</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>6</td>
<td>5</td>
<td>44</td>
<td>55</td>
<td>1.4</td>
<td>1.4</td>
</tr>
<tr>
<td>Abortion</td>
<td>98</td>
<td>5</td>
<td>91</td>
<td>194</td>
<td>4.9</td>
<td>3.4</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>272</td>
<td>38</td>
<td>94</td>
<td>404</td>
<td>10.2</td>
<td>5.6</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>18</td>
<td>19</td>
<td>70</td>
<td>107</td>
<td>2.7</td>
<td>2.7</td>
</tr>
<tr>
<td>Embolism</td>
<td>10</td>
<td>21</td>
<td>26</td>
<td>57</td>
<td>1.4</td>
<td>1.4</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>31</td>
<td>31</td>
<td>66</td>
<td>128</td>
<td>3.2</td>
<td>3.2</td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non pregnancy related Infections</td>
<td>1122</td>
<td>55</td>
<td>327</td>
<td>1504</td>
<td>38.0</td>
<td>43.7</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>67</td>
<td>59</td>
<td>111</td>
<td>237</td>
<td>6.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Unknown</td>
<td>57</td>
<td>29</td>
<td>88</td>
<td>174</td>
<td>4.4</td>
<td>4.4</td>
</tr>
<tr>
<td>Total</td>
<td>1872</td>
<td>502</td>
<td>1585</td>
<td>3959</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Using the new redistribution of primary causes of death (Table 11) there is a slight increase in deaths due to postpartum haemorrhage (6%), an increase of 44% in deaths due to abortion, an 82% increase in death due to pregnancy related sepsis and a decline in non-pregnancy related infections (14%). There were 598 deaths due to pregnancy related sepsis (abortion plus pregnancy related sepsis following viable pregnancies), 622 due to hypertension and 507 due to obstetric haemorrhage.
Direct causes of death are often regarded as areas where active treatment will save a life. The importance of this finding is that the role of sepsis (something we can manage using antibiotics and where necessary surgery) seems to have been underestimated previously. More attention must be placed on diagnosing and treating sepsis especially post-abortion and postnatally.

By extrapolating the proportion of women who attended antenatal care and tested, or declined testing and the proportion testing HIV positive or negative from the DHIS data for 2007, denominators for the number of women delivering who were HIV infected, negative or not known can be estimated. The institutional MMR for HIV negative women was 34/100000 live births, 328/100000 live births for those women who were HIV infected, and 275/100000 live births for those not testing.

**Obstructed labour/prolonged labour**

Obstructed labour/prolonged labour is not recorded as a specific primary cause of death in the South African system. In the next report obstructed/prolonged labour will be recorded for maternal death as an underlying cause. This will give us a better idea of the impact of obstructed/prolonged labour on maternal deaths. However, in this triennium there were 163 maternal deaths that could be directly ascribed to obstructed labour (Table 12).

<table>
<thead>
<tr>
<th>Sub-category</th>
<th>Obstructed labour deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRS following CPD</td>
<td>51</td>
</tr>
<tr>
<td>PPH due to atony following prolonged labour</td>
<td>32</td>
</tr>
<tr>
<td>Ruptured uterus</td>
<td>80</td>
</tr>
<tr>
<td><strong>Total (%)</strong></td>
<td><strong>163 (4.1%)</strong></td>
</tr>
</tbody>
</table>

**Levels of Care**

The proportion of the various causes of maternal deaths varied between the levels of care (see Figure 7), however non-pregnancy related infections was the most common cause at all levels of care. Postpartum haemorrhage and anaesthetic related deaths occurred most commonly at level 1 hospitals whereas complications due to hypertension and pregnancy related sepsis occurred at the same frequency at level 2 and 3 hospitals. Only in deaths due to pre-existing maternal disease did level 3
hospitals have more deaths than level 2 hospitals. Ideally, most deaths should occur in level 3 institutions, and the apparent excess of deaths in level 1 and 2 institutions is a cause for concern.

When the institutional MMR is calculated per disease category per level of care, an appropriate pattern appears.

Figure 7. Primary obstetric cause of reported maternal deaths: Numbers at Level 1, 2 and 3 Hospitals

Figure 8. Primary obstetric cause of reported maternal deaths: Institutional MMR at Level 1, 2 and 3 Hospitals
Caesarean sections

In 2005-2007 there were 477210 caesarean sections performed in South Africa, giving a caesarean section rate of 18.4%. The *institutional* MMR for caesarean sections was 198.2/100000 live births, whereas that for vaginal delivery was 77.8/100000 live births, a 2.5 times increase in risk of dying.
5. Avoidable factors, missed opportunities and substandard care

Tables 13-17 give a summary of the avoidable factors, missed opportunities and substandard care for the two triennia.

**Table 13. Avoidable factors, missed opportunities and substandard care for all cases**

<table>
<thead>
<tr>
<th>Category</th>
<th>% of avoidable factors in assessable cases</th>
<th>2005-2007</th>
<th>2002-2004</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient orientated</td>
<td>45.9</td>
<td>43.9</td>
<td></td>
</tr>
<tr>
<td>Administrative factors</td>
<td>29.9</td>
<td>32.1</td>
<td></td>
</tr>
<tr>
<td>Health worker related emergency management problems</td>
<td>Primary level</td>
<td>58</td>
<td>53.8</td>
</tr>
<tr>
<td></td>
<td>Secondary level</td>
<td>49</td>
<td>48.3</td>
</tr>
<tr>
<td></td>
<td>Tertiary level</td>
<td>30.1</td>
<td>36.5</td>
</tr>
<tr>
<td>Resuscitation</td>
<td>22.7</td>
<td>22.3</td>
<td></td>
</tr>
</tbody>
</table>

There were 1519 (38.4%) clearly avoidable deaths within the health system reported by the assessors. This is approximately the same as reported in 2002-2004 where the clearly avoidable deaths 36.7%.

**Table 14. Avoidable factors, missed opportunities and substandard care with respect to patient orientated problems for all cases**

<table>
<thead>
<tr>
<th>Major Problems</th>
<th>% of assessable deaths with avoidable factors</th>
<th>2005-2007 (n=3419)</th>
<th>2002-2004 (n=2836)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No antenatal care</td>
<td>17.7</td>
<td>18.1</td>
<td></td>
</tr>
<tr>
<td>Infrequent antenatal care</td>
<td>6.0</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>Delay in seeking medical help</td>
<td>26.7</td>
<td>26.8</td>
<td></td>
</tr>
<tr>
<td>Unsafe abortion*</td>
<td>25.7</td>
<td>21.1</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>6.9</td>
<td>6.0</td>
<td></td>
</tr>
</tbody>
</table>

* - Denominator is women who died due to abortions (136), not all maternal deaths

The lack of blood for transfusion has increased dramatically over the last triennium, from 9.2% to 19% of cases requiring blood.
Table 15. Avoidable factors, missed opportunities and substandard care with respect to administrative problems for all cases

<table>
<thead>
<tr>
<th>Major Problems</th>
<th>% of assessable deaths with avoidable factors</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2005-2007 (n=3664)</td>
</tr>
<tr>
<td></td>
<td>2002-2004 (n=3079)</td>
</tr>
<tr>
<td>Transport problem home to institution</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
</tr>
<tr>
<td>Transport problem between institutions*</td>
<td>8.4</td>
</tr>
<tr>
<td></td>
<td>9.7</td>
</tr>
<tr>
<td>Barriers to entry</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td>0.5</td>
</tr>
<tr>
<td>Lack of accessibility</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>1.0</td>
</tr>
<tr>
<td>Lack of specific health care facilities</td>
<td>8.6</td>
</tr>
<tr>
<td></td>
<td>11.2</td>
</tr>
<tr>
<td>Lack of ICU facilities*</td>
<td>9.2</td>
</tr>
<tr>
<td>Lack of blood for transfusion***</td>
<td>19.0</td>
</tr>
<tr>
<td></td>
<td>9.2</td>
</tr>
<tr>
<td>Lack of personnel</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>0.6</td>
</tr>
<tr>
<td>Lack of appropriately trained staff</td>
<td>8.9</td>
</tr>
<tr>
<td></td>
<td>12.8</td>
</tr>
<tr>
<td>Communication problems</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td>3.4</td>
</tr>
<tr>
<td>Other</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td>5.0</td>
</tr>
</tbody>
</table>

* - Denominator is the number of cases that were referred between institutions (1649).
** - Denominator is the number of women managed in tertiary institutions (850).
*** - Denominator was the number of cases which require urgent blood transfusions namely ectopic pregnancies, abortions due to trauma, ante and postpartum haemorrhage (559).

Table 16 gives the health worker orientated avoidable factors per level of care. The rankings of the avoidable factors is the same in all levels with level 1 institutions being consistently higher than level 2 institutions which are again consistently higher than level 3 institutions.

Table 16. Health worker orientated problems per level of care

<table>
<thead>
<tr>
<th>Assessable deaths (n)</th>
<th>2005-2007</th>
<th>Level 1</th>
<th>Level 2</th>
<th>Level 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2090</td>
<td>%</td>
<td>1668</td>
<td>%</td>
</tr>
<tr>
<td>Initial assessment</td>
<td>252</td>
<td>12.1</td>
<td>135</td>
<td>8.1</td>
</tr>
<tr>
<td>Problem with recognition / diagnosis</td>
<td>459</td>
<td>22.0</td>
<td>298</td>
<td>17.9</td>
</tr>
<tr>
<td>Delay in referring patient</td>
<td>325</td>
<td>15.6</td>
<td>64</td>
<td>3.8</td>
</tr>
<tr>
<td>Managed at inappropriate level</td>
<td>287</td>
<td>13.7</td>
<td>52</td>
<td>3.1</td>
</tr>
<tr>
<td>Incorrect management (Incorrect diagnosis)</td>
<td>152</td>
<td>7.3</td>
<td>70</td>
<td>4.2</td>
</tr>
<tr>
<td>Substandard management (Correct diagnosis)</td>
<td>619</td>
<td>29.6</td>
<td>506</td>
<td>30.3</td>
</tr>
<tr>
<td>Not monitored / Infrequently monitored</td>
<td>153</td>
<td>7.3</td>
<td>93</td>
<td>5.6</td>
</tr>
<tr>
<td>Prolonged abnormal monitoring without action</td>
<td>126</td>
<td>6.0</td>
<td>102</td>
<td>6.1</td>
</tr>
</tbody>
</table>

Table 17 compares the health worker orientated problems in 2005-2007 with 2002-2004 per level of care. In level 1 institutions there has generally been little change except for a significant increase in substandard management in 2005-2007 (p=0.000, OR 1.42 95% CI 1.2-1.6). Level 2 institutions there has been a general improvement.
except again for a significant increase in substandard management (p<0.001 OR 1.33 95% CI 1.13-1.56). In Level 3 institutions there has been an overall improvement.

**Table 17a. Comparison health worker orientated problems at Level 1 institutions between 2005-2007 and 2002-2004**

<table>
<thead>
<tr>
<th>2005-2007 Level 1</th>
<th>2005-2007 Level 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessable deaths (n)</td>
<td>2090</td>
</tr>
<tr>
<td>Initial assessment</td>
<td>252</td>
</tr>
<tr>
<td>Problem with recognition / diagnosis</td>
<td>459</td>
</tr>
<tr>
<td>Delay in referring patient</td>
<td>325</td>
</tr>
<tr>
<td>Managed at inappropriate level</td>
<td>287</td>
</tr>
<tr>
<td>Incorrect management (Incorrect diagnosis)</td>
<td>152</td>
</tr>
<tr>
<td>Substandard management (Correct diagnosis)</td>
<td>619</td>
</tr>
<tr>
<td>Not monitored / Infrequently monitored</td>
<td>153</td>
</tr>
<tr>
<td>Prolonged abnormal monitoring without action</td>
<td>126</td>
</tr>
</tbody>
</table>

**Table 17b. Comparison health worker orientated problems at Level 2 institutions between 2005-2007 and 2002-2004**

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessable deaths (n)</td>
<td>1668</td>
</tr>
<tr>
<td>Initial assessment</td>
<td>135</td>
</tr>
<tr>
<td>Problem with recognition / diagnosis</td>
<td>298</td>
</tr>
<tr>
<td>Delay in referring patient</td>
<td>64</td>
</tr>
<tr>
<td>Managed at inappropriate level</td>
<td>52</td>
</tr>
<tr>
<td>Incorrect management (Incorrect diagnosis)</td>
<td>70</td>
</tr>
<tr>
<td>Substandard management (Correct diagnosis)</td>
<td>506</td>
</tr>
<tr>
<td>Not monitored / Infrequently monitored</td>
<td>93</td>
</tr>
<tr>
<td>Prolonged abnormal monitoring without action</td>
<td>102</td>
</tr>
</tbody>
</table>

**Table 17c. Comparison health worker orientated problems at Level 3 institutions between 2005-2007 and 2002-2004**

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessable deaths (n)</td>
<td>959</td>
</tr>
<tr>
<td>Initial assessment</td>
<td>52</td>
</tr>
<tr>
<td>Problem with recognition / diagnosis</td>
<td>90</td>
</tr>
<tr>
<td>Delay in referring patient</td>
<td>6</td>
</tr>
<tr>
<td>Managed at inappropriate level</td>
<td>0.0</td>
</tr>
<tr>
<td>Incorrect management (Incorrect diagnosis)</td>
<td>22</td>
</tr>
<tr>
<td>Substandard management (Correct diagnosis)</td>
<td>190</td>
</tr>
<tr>
<td>Not monitored / Infrequently monitored</td>
<td>43</td>
</tr>
<tr>
<td>Prolonged abnormal monitoring without action</td>
<td>39</td>
</tr>
</tbody>
</table>
Table 18 gives the problems recorded with resuscitation of the women. This is slightly up from 2002-2004 which recorded problems in 17.9% of cases compared with 20.0% in 2005-2007. The biggest problem recorded was in not supporting the circulation adequately.

**Table 18. Health Worker related resuscitation problems in all cases**

<table>
<thead>
<tr>
<th>Description</th>
<th>% of assessable deaths with avoidable factors</th>
<th>Distribution of avoidable factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resuscitation</td>
<td>20.0</td>
<td>2005-2007 (n=699)</td>
</tr>
<tr>
<td>- Airway not secured</td>
<td>16.0</td>
<td></td>
</tr>
<tr>
<td>- Circulation not corrected</td>
<td>56.5</td>
<td></td>
</tr>
<tr>
<td>- Inappropriate drugs given</td>
<td>1.6</td>
<td></td>
</tr>
<tr>
<td>- Incompletely investigated</td>
<td>8.2</td>
<td></td>
</tr>
<tr>
<td>- Not appropriately monitored</td>
<td>1.7</td>
<td></td>
</tr>
</tbody>
</table>

In the 2002-2004 triennium and again in 2005-2007 assessors were asked to classify each maternal death as to whether the death was clearly preventable within the health system, i.e. all preventable deaths excluding the patient orientated factors (Table 19). This is clearly a subjective measure, but was aimed at getting a measure of the potential for improvement and establishing areas for focusing attention. Over eighty percent of maternal deaths due to anaesthetic complications and postpartum haemorrhage were thought to be avoidable. Hypertension, obstetric haemorrhage, pregnancy related sepsis and non-pregnancy related infections were responsible for 4 out of 5 avoidable deaths.
Table 19. Avoidable deaths per disease category

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>Total deaths</th>
<th>Number avoidable</th>
<th>% Avoidable</th>
<th>All avoidable deaths (n=1519)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td>1819</td>
<td>1065</td>
<td>58.5</td>
<td>70.1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>622</td>
<td>304</td>
<td>48.9</td>
<td>20.0</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>383</td>
<td>308</td>
<td>80.4</td>
<td>20.3</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>108</td>
<td>74</td>
<td>68.5</td>
<td>4.9</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>55</td>
<td>37</td>
<td>67.3</td>
<td>2.4</td>
</tr>
<tr>
<td>Abortion</td>
<td>136</td>
<td>74</td>
<td>54.4</td>
<td>4.9</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>223</td>
<td>128</td>
<td>57.4</td>
<td>8.4</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>107</td>
<td>91</td>
<td>85.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Embolism</td>
<td>57</td>
<td>15</td>
<td>26.3</td>
<td>1.0</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>128</td>
<td>34</td>
<td>26.6</td>
<td>2.2</td>
</tr>
<tr>
<td>Indirect</td>
<td>1966</td>
<td>421</td>
<td>21.4</td>
<td>27.7</td>
</tr>
<tr>
<td>Non Pregnancy Related Infections (AIDS)</td>
<td>1729</td>
<td>355</td>
<td>20.5</td>
<td>23.4</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>237</td>
<td>66</td>
<td>27.8</td>
<td>4.3</td>
</tr>
<tr>
<td>Unknown</td>
<td>174</td>
<td>33</td>
<td>19.0</td>
<td>2.2</td>
</tr>
<tr>
<td>Total</td>
<td>3959</td>
<td>1519</td>
<td>38.4</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Avoidable deaths are those classified by the assessors as being clearly avoidable within the health system (i.e. patient orientated factors are excluded).

% avoidable cause is the percentage of avoidable deaths that occurred in each primary cause category. For example assessors classified 304 of 622 women who died due to complications of hypertension in pregnancy as being avoidable, that is 48.9%.

% All avoidable deaths is the percentage of the deaths that were thought, by the assessors, to be avoidable of the total number of avoidable deaths. For example assessors thought 304 deaths due to hypertension were avoidable, that is 20.0% of all the avoidable deaths (n=1519).

Table 20 compares 2005-2007 with 2002-2004 with regard to whether there were any changes in the number of deaths thought to be avoidable per disease category. Table 21 compares the two trienniums with respect to the proportion which the deaths that were thought to be avoidable contributed to the overall number of avoidable deaths. There has been a significant shift in both deaths regarded as being avoidable and their contribution to the overall avoidable deaths by non-pregnancy related infection, mainly the AIDS category. This is expected as the strategic plan is scaled-up and HIV testing is performed on more pregnant women and antiretroviral drugs become more readily available. This is a reflection of the improved knowledge of the national guidelines on managing HIV infections.
### Table 20. Comparison of avoidable deaths per disease category with 2002-2004

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>2005-2007 % Avoidable cause</th>
<th>2002-2004 % Avoidable cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td>58.5</td>
<td>59.9</td>
</tr>
<tr>
<td>Hypertension</td>
<td>48.9</td>
<td>52.7</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>80.4</td>
<td>83.4</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>68.5</td>
<td>77.5</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>67.3</td>
<td>63.8</td>
</tr>
<tr>
<td>Abortion</td>
<td>54.4</td>
<td>50.0</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>57.4</td>
<td>57.7</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>85.0</td>
<td>90.1</td>
</tr>
<tr>
<td>Embolism</td>
<td>26.3</td>
<td>23.4</td>
</tr>
<tr>
<td>Acute collapse</td>
<td>26.6</td>
<td>23.4</td>
</tr>
<tr>
<td>Indirect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non Pregnancy Related Infections</td>
<td>20.5</td>
<td>9.1</td>
</tr>
<tr>
<td>(AIDS)</td>
<td>17.6</td>
<td>4.5</td>
</tr>
<tr>
<td>Pre-existing Maternal Disease</td>
<td>27.8</td>
<td>19.6</td>
</tr>
<tr>
<td>Unknown</td>
<td>19.0</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>38.4</td>
<td>36.7</td>
</tr>
</tbody>
</table>

(AIDS is a subset of non-pregnancy related infections)

### Table 21. Comparison of the contribution of each primary obstetric cause to avoidable deaths

<table>
<thead>
<tr>
<th>Primary Obstetric Cause</th>
<th>2005-2007 % All avoidable deaths (n=1519)</th>
<th>2002-2004 % All avoidable deaths (n=1208)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td>70.1</td>
<td>87.7</td>
</tr>
<tr>
<td>Hypertension</td>
<td>20.0</td>
<td>27.4</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>20.3</td>
<td>21.6</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>4.9</td>
<td>8.3</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>2.4</td>
<td>2.5</td>
</tr>
<tr>
<td>Abortion</td>
<td>4.9</td>
<td>4.7</td>
</tr>
<tr>
<td>Pregnancy Related Sepsis</td>
<td>8.4</td>
<td>13.1</td>
</tr>
<tr>
<td>Anaesthetic related</td>
<td>6.0</td>
<td>6.8</td>
</tr>
<tr>
<td>Embolism</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>Acute collapse</td>
<td>2.2</td>
<td>3.3</td>
</tr>
<tr>
<td>Indirect</td>
<td>27.7</td>
<td>12.3</td>
</tr>
<tr>
<td>Non-pregnancy related infections</td>
<td>23.4</td>
<td>9.4</td>
</tr>
<tr>
<td>(AIDS)</td>
<td>10.6</td>
<td>2.5</td>
</tr>
<tr>
<td>Pre-existing maternal disease</td>
<td>4.3</td>
<td>3.0</td>
</tr>
<tr>
<td>Unknown</td>
<td>2.2</td>
<td>0.0</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

(AIDS is a subset of non-pregnancy related infections)
6. Discussion

This fourth comprehensive report on confidential enquiries into maternal deaths continues to demonstrate the value of such a system. This report has once again clearly identified the numbers, causes and avoidable or remediable factors associated with maternal deaths. As stressed above, the CEMD system is not an epidemiological survey but can give an idea of the order of magnitude of the problems and potentially track any changes. In the triennium 2005-2007 the numbers of maternal deaths reported has increased, and the increase is mainly due to an increase in deaths due to non-pregnancy related infections and improved reporting.

The impact of the HIV epidemic is having on maternal deaths is clearly demonstrated in this report. Without the HIV epidemic the institutional MMR would be similar to other mid-income countries like Brazil, Argentina and Thailand. The institutional MMR of women who were HIV infected was almost ten times that of the HIV negative women. The institutional MMR of the group where the HIV status was unknown was also very high. The provider initiated counselling should reduce this number. If the HIV status is unknown, women who are HIV infected miss the opportunity of effective therapy that will save their lives. The increasing availability of antiretroviral therapy makes many of the deaths due to AIDS clearly avoidable and it is not surprising there has been an increase of clearly avoidable deaths from 9.1% of the non-pregnancy related infections and 4.5% of the AIDS deaths in 2002-2004 to 23.4% of non-pregnancy related infections and 17.6% of AIDS deaths. The scale-up of the antiretroviral therapy programme must continue expanding rapidly.

Complications of hypertension in pregnancy remain the most common direct cause of maternal deaths. There has been a 13.7% reduction in deaths due to complications of hypertension. This is unlikely to be due to problems with classification and may be the first real sign that the recommendations on managing hypertension in pregnancy are being adhered to. This is further supported by the small decrease in avoidable deaths due to hypertension. There is an apparent decrease in deaths due to pregnancy related sepsis (septic abortion and puerperal sepsis following viable pregnancies). However, this probably due more to problems with classification as discussed above. If the alternative classification is taken into account, pregnancy related sepsis would
have a MMR similar to that of complications of hypertension (22.93/100000 deliveries). Deaths due to obstetric haemorrhage (antepartum and postpartum haemorrhage) and ectopic pregnancies seem to have stabilised.

Four out of five of clearly avoidable maternal deaths were due to complications of hypertension, obstetric haemorrhage, pregnancy related sepsis and non-pregnancy related infections. The ways to prevent these deaths are known. Specific protocols have been developed and these have been included in the recommendations given in the previous report. Despite this, the most important avoidable factor is still substandard care (i.e. the lack of adherence to standard protocols). Renewed effort must be put in to ensuring the protocols are known and used. The most effective method of outreach has been shown to be face-to-face teaching on-site by a respected clinician. This ideal is difficult to attain but some systematic sustainable method of outreach must be devised. Further emphasis on the major causes of maternal deaths must take place in all medical schools and nursing colleges and all post-graduate degrees that involve pregnant women.

The NCCEMD believes the implementation of the recommendations will result in a reduction of maternal deaths in South Africa. This appears to be the case in complications in hypertension. Further it believes their implementation is feasible and can be achieved within the health resources of the country.

The relatively low number of deaths occurring in level 3 institutions compared with level 1 and 2 is a cause for concern. Are the level 1 and 2 hospitals not referring the patients because the problem is not recognised or there is no transport available or are the level 3 institutions not accepting the referrals’ as they have no space? Part of the answer is in lack of recognition of the problem as this is a common avoidable factor in both level 1 and 2 institutions. Delay in referral is a problem at level 1 institutions reflecting lack of recognition of the problem or lack of transport. Lack of transport between institutions was associated with 1 in 10 of maternal deaths where transport between institutions was required. There is no indication whether level 1 or 2 institutions tried to refer patients but the level 3 institutions were unable or unwilling to accept the patients, thus the magnitude of this problem is unknown at present. Alternatively are there just not enough tertiary beds. The Free State province is an
example of a province where there might be an acute shortage of tertiary beds. Other provinces have no tertiary beds and need to refer patients to other provinces. There are 9 national central hospitals and 6 provincial tertiary hospitals. Are these enough to manage all the tertiary patients seen in South Africa?

Lack of attendance at antenatal clinics continues to be a common patient related avoidable factor and health messages must continue to stress the need to attend antenatal clinic early in pregnancy. However, a large proportion of the women that subsequently died did attend clinics giving the health workers a chance to intervene and prevent the deaths. Attention will need to be paid to the quality of antenatal care so that these opportunities are not missed. This is particularly relevant to HIV infected women, women with hypertension or other pre-existing medical conditions.

Delay in seeking help was the most common patient related avoidable factor. The exact meaning of this is hard to establish as assessors can only use the data available in the case notes. If lack of transport or other factors inhibiting the woman seeking help is not recorded in the notes, the assessor will not be able to document them. Independent research has indicated most of the delays are due to the inability to access transport especially at night leading to delay, rather than lack of knowledge or concern by the patient.

**Conclusion**

The final comment of the 1999-2001 report was “Every woman who becomes pregnant and continues with her pregnancy does so in the expectation of delivering a healthy child and the joy and satisfaction of watching the child grow. Surely, it is the duty of society and the health care profession to do the utmost to fulfil this expectation? To this end, the deficiencies identified in this report must be urgently addressed. The committee are anxious to see clear signs of progress by the next triennial report”.

Unfortunately this, with the notable exception of women dying from complications of hypertension in pregnancy, has not come to pass. We will have to redouble our efforts.
7. References


Ten Key Recommendations

The National Committee on Confidential Enquiries into Maternal Deaths (NCCEMD) reviewed the recommendations as outlined in the *Saving Mothers 2002-2004* report, assessing whether the recommendations are still relevant. A large number of problems previously identified were still present in this 2005-2007 report. Thus the majority of recommendations have remained the same or slightly modified. The key strategies identified in the previous report to accelerate implementation of the recommendations are included in these recommendations. The *golden threads* running through the implementation strategies for the recommendations continue as:

- Introduction of recommendations into managers Key Performance Areas
- Outreach on-site, face to face teaching and training that is documented

Areas for implementation of the recommendations are classified into policy and management, administration and monitoring and clinical practice where applicable. Targets to be achieved have been specified and should be fully in place by December 2010.

1. **Protocols on the management of important conditions causing maternal deaths must be available and utilised appropriately in all institutions (including facilities which only provide antenatal and postnatal services) where women deliver. All midwives and doctors must be trained on the use of these protocols.**

**Motivation for recommendation**

The most frequent health worker related avoidable factor was substandard care, namely lack of adherence to standard protocols. This was present in 30% of cases managed in level 1 and 2 institutions and 20% in level 3 institutions. Four out of five clearly avoidable maternal deaths were due to complications of hypertension, obstetric haemorrhage, pregnancy related sepsis and non-pregnancy related infections. Problems in resuscitation occurred in 20% of cases.

The following are key conditions of which relevant protocols must be available:
• Hypertensive disorders in pregnancy
• Obstetric haemorrhage,
• Septic abortion,
• Puerperal Infections,
• HIV/AIDS (including community acquired pneumonias, PCP, meningitis and lactic acidosis), TB and Malaria
• Resuscitation: Maternal and Neonatal.
• Cardiac disease in pregnancy

Note: Protocols are derived from guidelines and are specific to each institution. For example the protocol for managing a woman with severe pre-eclampsia will be different in a primary health care clinic from a secondary level hospital, however, they will both comply with the guidelines for managing severe pre-eclampsia. Protocols will also differ between similar levels of institutions as referral routes; telephone numbers, types of emergency transport differ in different areas. Protocols are very detailed instructions for managing a condition and are specific to that institution. Guidelines contain the principles on how the woman should be managed. Protocols are derived from guidelines by the institution. (For more detail see Saving Mothers: Policy and management guidelines for common causes of maternal death. Government Printer, Pretoria, 2001, pp2-4)

Indicators

1. Availability of relevant written protocols derived from Guidelines for Maternity Care in South Africa 2007; Essential Steps in Managing the Common Causes of Maternal Death in South Africa; and Policy and Guidelines for the Implementation of the PMTCT Programme (11 February 2008) in the form of posters, individual booklets or tool kits in relevant sections of health facilities
2. Availability of a functioning training programme for all institutions at district level
3. Availability of the functioning program on quality assurance for proper use of guidelines by midwives and doctors at district level
Targets
1. All institutions must have relevant written protocols in the form of posters, individual booklet or tool kit in relevant sections of these facilities
2. All districts must have a written functioning training programme in all institutions
3. All districts must have a written functioning program on quality assurance for proper use of guidelines by all health professionals including midwives and doctors

Implementation strategy
• Policy and Management
  – Clinical guidelines on management of important conditions causing maternal deaths should be updated, strengthened and distributed to all health institutions
  – The clinical guidelines should be included in undergraduate and postgraduate curricula for doctors and nurses. Motivations to the appropriate health professional bodies must be performed
  – Recommend to policy makers that implementation of the guidelines be incorporated into the Key Performance Areas of the appropriate managers.
• Administration and monitoring
  – Systems must be developed to facilitate training by institutional managers
  – All guidelines to be made available to the health care workers involved in maternity care either in hard copy or electronically
  – Have a strategy in place for monitoring availability of protocols and use of the protocols
• Clinical practice
  – On-site face-to face training should involve academic institutions and referral centres. Academic institutions and referral centres, including medical schools and nursing colleges, should be allocated areas of responsibilities. In addition medical schools and nursing colleges should improve the teaching of relevant clinical competencies and skills.
All undergraduates must be trained on the Guidelines for Maternity Care in South Africa 2007; Essential Steps in Managing the Common Causes of Maternal Death in South Africa; and Policy and Guidelines for the Implementation of the PMTCT Programme (11 February 2008).

2. Training should be provided for all health professional working in maternity units in practical obstetrical and surgical skills. Skills should be provided in anaesthesia, especially in level 1 institutions.

Motivation for recommendation
Lack of appropriately trained staff was present in almost one in ten cases of maternal death. This varied from 2.2% to 21.2% in the provinces. It was present in 22.4% of anaesthetic related deaths, 17.5% of obstetric haemorrhages cases and approximately 10% in hypertension and pregnancy related sepsis. A special comment has been made at the apparent lack of surgical skills for caesarean section in the chapter on obstetric haemorrhage. Lack of resuscitation skills was also recorded in 20% of cases.

A training programme developed in South Africa for improving obstetric and surgical skills is available and has been tested on over a hundred interns throughout the country. The training programme (Essential Steps in Managing Obstetric Emergencies – ESMOE) improved the interns’ skills between 31% and 65%. ESMOE Fire-Drills will be available in March 2009. The fire-drills are intended to train all the staff in the maternity units.

Indicators
1. Availability of ESMOE fire-drills and skills training in health care facilities
2. Availability of a ESMOE skills training programme for all institutions at district level
3. Availability of the functioning program on quality assurance for assessing skills of midwives and doctors at district level
4. Anaesthetic indicator
Target
1. All hospitals must have implemented ESMOE fire-drills and skills training
2. All sub-districts must have access to ESMOE skills training programme for all institutions in the sub-district
3. All districts must have a functioning program on quality assurance for assessing skills of all health professionals including midwives and doctors

Implementation strategy
• Policy and senior management
  – Develop a tool kit for regional anaesthesia to guide doctors at lower level institutions and this should be added to ESMOE training package
  – Recommend to policy makers that improving practical obstetric and surgical skills and skills in anaesthesia be incorporated into the Key Performance Areas of the appropriate managers.
  – Involve the HPCSA, the Colleges of Medicine (Obstetrics and Gynaecology, Anaesthesia, Paediatricians), Deans Committee, South African Society of Obstetrics and Gynaecology, South African Chapter of the RCOG, Society of Midwives of South Africa and Nursing Council in negotiations on this matter

• Administration and monitoring
  – ESMOE programme to be made available for doctors for improving skills
  – ESMOE programme to be made available for midwives for improving skills
  – Implement ESMOE Fire-drills for all maternity units

• Clinical practice
  – Outreach programmes by academic institutions and referral hospitals to all institutions performing caesarean sections
  – Strengthen supportive supervision of doctors and midwives
  – Outreach face-to-face, on-site training programmes (including District Hospitals and MOUs)
A quality assurance programme should be implemented, using an appropriate tool.

3. All pregnant women should be offered information on, screening for and appropriate management of non-pregnancy related infections and common medical disorders

The specific conditions are:

- HIV/AIDS
- Tuberculosis
- Malaria
- Hypertension
- Anaemia
- Cardiac disease

**Motivation for recommendation**

AIDS was the most common cause of maternal deaths in this report. Only 60% of the women who died were tested for HIV. In 2007, nationally approximately 22% of pregnant women were not counselled for or declined HIV testing at antenatal clinics. Without knowledge of the HIV status many opportunities to prevent maternal deaths are missed. Tuberculosis is commonly associated with AIDS.

Complications of hypertension were the most common direct causes of maternal deaths. Screening for hypertension is simple and inexpensive. Obstetric haemorrhage was the second most common direct cause of death. Pregnant women who are anaemic are at a major disadvantage if they develop an obstetric haemorrhage and are more likely to die. Screening for anaemia is simple and inexpensive. Pre-existing cardiac disease was the most common cause of maternal death in the pre-existing medical disorders. Although relatively uncommon women with cardiac lesions are at very high risk of death during pregnancy if the disease is not detected and managed appropriately. Screening is by taking an appropriate history and doing a clinical examination of the cardiovascular system.
**Indicator**

Percentage institutions providing appropriate maternity care offering information on screening for and appropriate management of non-pregnancy related infections and common medical disorders.

**Targets**

1. All institutions that perform maternity care should provide:
   a. Provider initiated counselling and testing for HIV in all districts and sub-districts. HIV staging (including CD4 counts) to be performed in HIV infected women at point of diagnosis. Dual therapy to be started for all immediately on diagnosis at 28 weeks or immediately if the diagnosis is made after 28 weeks. HAART treatment for women with AIDS should be referred for and started within 2 weeks.
   b. Appropriate history taking and examination for tuberculosis and where necessary for continued anti-tuberculosis therapy.
   c. Malaria screening in malaria areas and malaria prophylaxis
   d. Screening for anaemia, hypertensive disorders in pregnancy, and cardiac disease

**Implementation strategy**

- **Policy and Management**
  - Key information for all women must be developed and made available for everyone by the maternal health unit
  - Recommend to policy makers that the implementation of informing, screening and managing non-pregnancy related infections and common medical disorders be incorporated into the Key Performance Areas of the appropriate managers.

- **Administration and monitoring**
  - Screening tools and treatment facilities and schedules must be available
  - Specific training on antiretroviral therapy for HIV should be provided
Clinical practice

On-site face-to-face training should include screening and treating HIV, TB, malaria, hypertension, anaemia and cardiac disease. Preferably academic institutions, including medical schools and nursing colleges, should be allocated areas of responsibilities. In addition, medical schools and nursing colleges must improve the teaching of relevant clinical competencies and skills.

4. Criteria for referral and referral routes must be established and utilized appropriately in all provinces. Emergency transport facilities must be available for all pregnant women in need (at any site).

Motivation for recommendation

Problems with transport between institutions were reported in 8.4% of cases requiring transfer and that subsequently died. Delay in seeking medical help was reported in 26.7% of cases. The most common reason for the delay was the lack of transport between the woman’s home and a health care institution. Delay in referring cases occurred in 15.6% and managing them at an inappropriate level occurred in 13.7% of women managed at some point at a level 1 institution. As each category is mutually exclusive, almost 30% of maternal deaths managed at some point at a level 1 institution had problems with referral.

Indicators

1. Availability of referral routes and criteria for referral in the relevant areas in hospitals and emergency services
2. Time from call for and ambulance to arrival of the ambulance at site

Target

1. All facilities providing maternity services must have functional referral routes and referral criteria.
2. 70% of ambulances on red code calls must arrive at the emergency site within 1 hour of call.
Implementation strategy

• Policy and Management
  – Referral systems must be clearly stated
  – Recommend that patients clinical records/charts accompany the patient on transfer
  – Build into referral system, transfer directly to appropriate health care level if necessary rather than from level 1 to 2 them to 3
  – Make obstetric emergencies (including ectopic pregnancies and major gynaecological haemorrhages), red code (highest priority)
  – Consideration should be given to “waiting mothers areas” where required.
  – Telecommunication networks must be extended to cover rural areas so that contact can be made with clinics and ambulances
  – Recommend to policy makers that the implementation of transport policy and implementation of referral criteria and routes be incorporated into the KPA’s of the appropriate managers

• Administration and monitoring
  – Establish written agreements between different health districts/regions, and provinces, and include emergency services in negotiations
  – Develop risk classification guidelines
  – Monitor transport times and investigate cases with excessive delays
  – Inform all health professionals of policies at least twice a year because of staffing changes
  – Work with emergency medical services in each province to identify delays and solve problems

• Clinical practice
  – Training in criteria for referral for all health professionals (doctors, nurses and emergency services)
  – Emergency medical personnel to be trained on obstetric emergencies
  – All referred patients to be seen by an experienced practitioner at the referral site within one hour. Emergencies must be seen immediately.
5. Postnatal care must be strengthened

Motivation for recommendation
The postpartum period was the most common period for the problem resulting in death to occur, occurring in 45.4% of cases compared with 9% in early pregnancy, 36.7 antenatally and 8.7% intrapartum. Most maternal deaths occur after the woman has given birth.

Twenty-seven percent of the women who died due to pregnancy related sepsis following a viable pregnancy were reported to have delayed in seeking help. If these women were seen within 6 days of discharge their deaths might have been prevented.

Indicator
Recorded visits at DHIS as a percentage of births in the sub-district.

Targets
1. 60% of women and children attend postnatal care at 6 weeks
2. Establish a mechanism to monitor the number the of postpartum visits within 6 days of delivery

Implementation strategy
• Policy and Management
  – Enforce the policy that women and their babies in the postnatal period are examined at 6 hours, within 6 days of delivery and at 6 weeks
  – Recommend to policy makers that the implementation of postnatal policy be incorporated into the Key Performance Areas of the appropriate managers.

• Administration and monitoring
  – Ensure postnatal women are properly examined regularly until discharge from the birth institution. Women with risk factors must be appropriately managed and an individual postnatal follow-up plan developed
– Encourage the clinics to be integrated so that the woman and her baby can be seen at the same time.
– Investigate the possibility of community health workers visiting all women at their home within 6 days of delivery.
– High risk patients should be followed in the appropriate institutions
– Establish a mechanism of monitoring visits within 6 days of delivery

• Clinical practice
  – On-site face-to-face training should include training in managing the puerperium e.g. Essential postnatal care training and postnatal card
  – Training in contraception must be emphasised

6. Staffing and equipment norms must be established for each level and for every health institution concerned with the care of pregnant women.

Motivation for recommendation
Many of the health worker related avoidable factors may be explained by a lack of staff and lack of staff is the most frequent complaint made by health workers when considering quality of care issues. Lack of staff was very infrequently mentioned as an avoidable factor in this report. That does not mean the problem is uncommon, it may be under reported. Assessors can only record lack of staff as an avoidable factor if it is recorded in the case notes. The only way to resolve the question of staffing is to have norms against which each institution can measure itself. The size of the problem of lack of staff will then be apparent.

Indicator
1. Availability of guidelines on allocation of human resources for maternal and neonatal health services
2. Availability of guidelines on essential equipments for provision of maternal and neonatal health care at different levels.
Target
Written guidelines for human resource allocation and for essential equipment must be available at national, provincial, district and facility level.

Implementation strategy

• Policy and Management
  – Establish staffing norms
  – Recruit new health workers and institute processes to retain staff
  – Establish training sites for new recruits
  – Recommend to policy makers that establishing staffing norms and methods of retaining staff be incorporated into the Key Performance Areas of the appropriate managers
  – Restrict staff rotation in maternity units

• Administration and monitoring
  – Essential equipment must be available
  – Equipment lists for managing pregnant and post partum women and their babies be updated regularly
  – Measure staffing against norms

7. Blood for transfusion must be available at every institution where caesarean sections are performed

Motivation for recommendation
Lack of blood for transfusion is becoming an increasing problem. It occurred in 19% of maternal deaths that required blood urgently. This is up from 9.2% in the previous triennium.

Indicator
Percentage of applicable institutions having adequate emergency blood available

Target
All applicable institutions
Implementation strategy

• Policy and Management
  – Ensure that blood and all other blood products are available in all relevant facilities; Plasma expanders should also be available in the labour wards
  – Facilitate immediate replacement of depleted emergency blood supplies
  – Recommend to policy makers that availability of blood for transfusion be incorporated into the KPA’s of the appropriate managers

• Administration and monitoring
  – Ensure blood availability in facility
  – Audit use and availability of blood

• Clinical practice
  – Training health workers on proper use of blood and blood products
  – Training health workers on measures to prevent the need for blood transfusion, e.g. use of iron and folic acid prophylaxis for all pregnant and post partum women; advice on proper nutrition; active management of the third stage of labour and the antenatal transfer of women at risk of postpartum haemorrhage (PPH) to the appropriate level of health care.

8. Contraceptive use must be promoted through education and service provision.

Motivation for recommendation
The best way to prevent maternal deaths is to prevent pregnancy. Many women do not plan their pregnancies and some pregnancies are not wanted. These women are at higher risk than those who planned their pregnancies. Women 35 years or older had a greater risk of maternal deaths than younger women. Also teenagers also had a greater risk of maternal deaths, especially due to complications of hypertension.

Indicators
  1. Percentage of tubal ligations in women over 35 years of age
2. Number of intrauterine contraceptive devices (IUCD) being inserted
3. Number of health care workers trained in IUCD insertion
4. Percentage of regional and provincial tertiary and national central hospitals have contraceptive services
5. Number of vasectomies performed

Targets
1. Sustained increase in women using contraceptive services having tubal ligations, IUCD insertions and vasectomies performed
2. All districts must provide information on all forms of contraceptives and family planning
3. All regional and provincial tertiary and national central hospitals to have contraceptive services

Implementation strategy
• Policy and Management
  – Ensure availability of all contraceptive methods, especially emergency contraception
  – Ensure public is advised on rights and sites
  – All health facilities irrespective of level of care should offer contraceptives
  – Ensure availability of all contraceptive methods, especially emergency contraception and IUCDs.
  – Recommend to policy makers that the implementation of improving contraceptive services be incorporated into the Key Performance Areas of the appropriate managers.
  – All health facilities irrespective of level of care should offer contraceptives
• Administration and monitoring
  – Provision of facilities and staff for tubal ligation
  – Ensure availability of all contraceptives
    • Primary health care
    • High risk patients
  – Implement fast lanes for women requiring contraceptives
9. The number of deaths from unsafe abortion must be reduced

Motivation for recommendation
The number of deaths due to abortion increased slightly in the last triennium. This was disappointing especially after the significant decrease in deaths in the previous triennium. The number of terminations of pregnancy in public institutions has been falling in the last triennium and the number of public institutions performing TOPs is declining. There is concern the rise in maternal deaths might be related to this as women have less accessibility to TOP services.

Indicators
1. Percentage of functioning Termination of Pregnancy (TOP) services in relation to designated public sector units separately for first and second trimester pregnancies.
2. Availability of strategies for advertising TOP services within the district.

Targets
1. All sub-districts must be able to provide first trimester TOPs
2. 70% of sub-districts must be able to provide second trimester TOPs

Implementation strategy
• Policy and Management
  – Expand sites for second trimester TOP
  – Ensure availability of all contraceptive methods, especially emergency contraception
  – Ensure public is advised on rights and sites
  – Recommend to policy makers that reduction of numbers of deaths from unsafe abortion is incorporated in the KPA’s of the appropriate managers
– Incentives should be recommended for doctors, midwives and registered nurses doing TOPs. Occupation specific dispensation for TOPs should be considered and negotiated with the Department of Health
– Consider medical termination of pregnancy to reduce waiting times for first trimester TOPs and decrease the number of second trimester TOPs

• Administration and monitoring
  – Make institutions available for TOPs
  – Streamline administrative route from requesting TOP to performance of TOP to a **maximum** of 2 visits one for assessment and one for performance

• Clinical practice
  – Training in TOPs for doctors, midwives and registered nurses
  – Hold regular value clarification workshops

10. **Women, families and communities at large must be empowered, involved and participate actively in activities, projects and programmes aiming at improving maternal and neonatal health as well as reproductive health in general.**

**Motivation for recommendation**
Patient related avoidable factors were recorded in 46.5% of maternal deaths. Including communities in activities related to pregnancy and having consistent health messages will reduce the number of patient related avoidable factors and result in less women dying.

**Indicators**

1. Percent functioning community empowerment programmes at sub-district level
2. Availability of appropriate Information, Education and Communication Material (IEC) or Behaviour Change and Communication (BCC) material addressing major issues around maternal deaths for women and the general population.
**Targets**

1. 70% of sub-districts must be able to conduct at least quarterly activities targeting women and the general population to raise awareness and facilitate change of behaviour regarding maternal and neonatal health plus reproductive and sexual health in general; the activity should be documented and reported to the maternal health unit of the National Department of Health

2. 70% of sub-districts must be able to provide appropriate IEC or BCC material addressing major issues around maternal deaths for women and general population at all times.

**Implementation strategy**

- **Policy and Management**
  - Develop and disseminate relevant information, education and communication material
  - Encourage male participation in reproductive health
  - Encourage a healthy lifestyle (including proper nutrition) as preventative measure
  - Recommend to policy makers that empowering the community in maternal and child care be incorporated into the Key Performance Areas of the appropriate managers
  - Pregnant women should be encouraged to and supported in arranging transport when she has medical problems or when labour starts

- **Administration and monitoring**
  - Create opportunities for linkages (such as open days) with stakeholders within the community
  - Involve the communications dept/unit and health promotion unit in disseminating information and encouraging community participation

- **Clinical practice**
  - Train health professionals on culture and traditions of their community
  - Train all health workers to be empathic towards patients and their families