Maternal Collapse in Pregnancy and the Puerperium

Green–top Guideline No. 56

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This is the first edition of this guideline.

1.   Purpose and scope

Maternal collapse is a rare but life-threatening event with a wide-ranging aetiology. The outcome, primarily for the mother but also for the fetus, depends on prompt and effective resuscitation. The purpose of this guideline is to discuss the identification of women at increased risk of maternal collapse and the different causes of maternal collapse, to delineate the initial and continuing management of maternal collapse and to review maternal and neonatal outcomes. It covers both hospital and community settings, and includes all gestations and the postpartum period. The resuscitation team and equipment and training requirements will also be covered.

2.   Background and introduction

Maternal collapse is defined as an acute event involving the cardiorespiratory systems and/or brain, resulting in a reduced or absent conscious level (and potentially death), at any stage in pregnancy and up to six weeks after delivery. While there is a robust and effective system for maternal mortality audit in the UK in the form of the Confidential Enquiry into Maternal and Child Health (CEMACH), now the Centre for Maternal and Child Enquiries (CMACE), the incidence of maternal collapse or severe maternal morbidity is unknown as morbidity data are not routinely collected. There are drivers to improve this situation, but resources are limited.[1] The UK Obstetric Surveillance System (UKOSS), run by the National Perinatal Epidemiology Unit (NPEU), has made a significant contribution towards the study of rare events and maternal morbidity.[2] Severe maternal morbidity data was collected Scotland-wide for 5 years and published in 2007.[3] A woman was defined as having had a severe maternal morbidity event if there was a risk of maternal death without timely intervention. The data showed a severe maternal morbidity rate of 6/1000 (600/100 000) maternities, but not all cases of severe maternal morbidity involved maternal collapse (although all cases of collapse were included in the figures). A recent publication from Dublin showed a severe maternal morbidity rate of 3.2/1000 (320/100 000) births.[4] In the last triennium in the UK the maternal mortality rate was 14/100 000 births,[1] but again not all maternal deaths are preceded by maternal collapse. Thus, the true rate of maternal collapse lies somewhere between 0.14 and 6/1000 (14 and 600/100 000) births. As it is such a rare event, with potentially devastating consequences, it is essential that caregivers are skilled in initial effective resuscitation techniques and are able to investigate and diagnose the cause of the collapse to allow appropriate, directed continuing management. Unfortunately, in reports regarding morbidity[3,4] and in the CEMACH report,[1] areas of substandard care continue to be identified, including poor resuscitation skills, but it should also be remembered that death and disability may result despite excellent care.[1] It should be noted that vasovagal attacks and the postictal state following an epileptic seizure are the most common causes of 'maternal collapse' and are not covered by this guideline.

3.   Identification and assessment of evidence

This guideline was developed in accordance with standard methodology for producing RCOG Green-top Guidelines.[5] The Cochrane Library (including the Cochrane Database of Systematic Reviews, DARE and EMBASE), TRIP, Medline and PubMed were searched for relevant randomised controlled trials, systematic reviews and meta-analyses. The search was restricted to articles published between 1960 and 2010. The databases were searched using the relevant MeSH terms, including all subheadings, and this was combined with a keyword search. Search words included 'amniotic fluid embolism', 'cardiac arrest and pregnancy', 'DVT and pregnancy', 'hypovolaemia and pregnancy', 'hypoxia and pregnancy', 'massive haemorrhage', 'maternal collapse' and 'resuscitation and pregnancy'. The search was also limited to humans and the English language. The National Library for Health and the National Guidelines Clearing House were also searched for relevant guidelines and reviews.

4. Clinical issues

4.1 *Can women at risk of impending collapse be identified early?*

An obstetric early warning score chart should be used routinely for all women, to allow early recognition of the woman who is becoming critically ill.

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In some cases maternal collapse occurs with no prior warning, although there may be existing risk factors that make this more likely. Antenatal care for women with significant medical conditions at risk of maternal collapse should include multidisciplinary team input with a pregnancy and delivery management plan in place. Often there are clinical signs that precede collapse. In the latest CEMACH report,[1] substandard care was often identified where these signs and symptoms were not recognised and acted upon. The report recommended that a national obstetric early warning scoring system should be introduced and used for all obstetric women, including those being cared for outside the obstetric setting.[1]

Evidence level 4

The first early warning scoring (EWS) systems[6] were introduced on the basis that a deterioration in simple physiological vital signs will precede significant clinical deterioration, and that early intervention will reduce morbidity.[7-11] EWS systems are now extensively used in acute settings and critical care,[12-14] although it has not been possible to identify the optimal system.[15]

Evidence level 1+

Despite this, EWS systems have not been demonstrated to be highly effective, even when their use has triggered input from a specialised medical emergency team,[16] and although their use is recommended by the National Institute for Health and Clinical Excellence (NICE),[17] this is based on informal consensus rather than evidence.

The physiological changes of pregnancy may render the existing EWS systems inappropriate,[18] and no validated system for use in the pregnant woman currently exists. Because of this, many maternity hospitals have developed their own modified EWS system, and there is continuing work in the UK to try and develop a national obstetrics EWS system. However, this should be subjected to rigorous scrutiny to ensure that it is effective before it is universally implemented.

4.2 *What are the causes of maternal collapse?*

There are many causes of collapse, and these may be pregnancy-related or result from conditions not related to pregnancy and possibly existing before pregnancy. Systematic consideration of the causes of collapse can enable skilled rescuers to identify the cause of collapse in the hospital setting and, where the cause is reversible, survival can be improved.[19] The common reversible causes of collapse in any woman can be remembered using the well known 'aide memoire' employed by the Resuscitation Council (UK) of the 4 T's and the 4 H's.[19] In the pregnant woman, eclampsia and intracranial haemorrhage should be added to this list, and obstetric-specific causes are clearly more likely and must also be considered systematically (Figure 1). Owing to the lack of robust morbidity data regarding collapse, maternal deaths are often used as a reference point. The common causes of maternal collapse are discussed below, but this is not an exhaustive list, as this is beyond the scope of this guideline.

4.2.1 Haemorrhage

This is the most common cause of maternal collapse, and was responsible for 17 maternal deaths in the last triennium.[1] Major obstetric haemorrhage has an estimated incidence of 3.7/1000 maternities.[20] Causes of major obstetric haemorrhage include postpartum haemorrhage, major antepartum haemorrhage from placenta praevia/accreta, placental abruption, uterine rupture and ectopic pregnancy. In most cases of massive haemorrhage leading to collapse, the cause is obvious, but concealed haemorrhage should not be forgotten, including following caesarean section and ruptured ectopic pregnancy. Other rarer causes of concealed haemorrhage include splenic artery rupture[21] and hepatic rupture. Blood loss is often underestimated,[22,23] especially slow, steady bleeding, and fit healthy women can tolerate significant loss prior to showing signs of decompensation.

Figure 1. Causes of maternal collapse
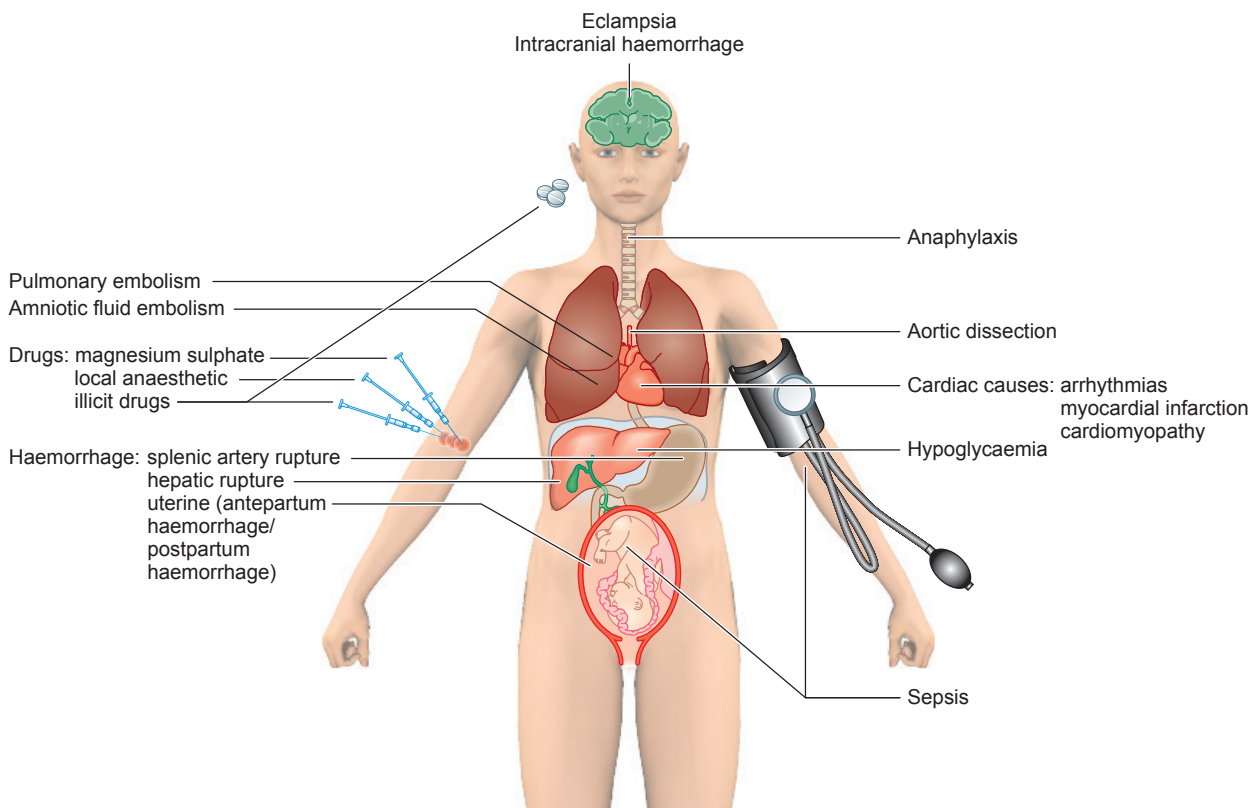
Reversible cause		Cause in pregnancy
4 H's	Hypovolaemia	Bleeding (may be concealed) (obstetric/other) or relative hypovolaemia of dense spinal block; septic or neurogenic shock
	Hypoxia	Pregnant patients can become hypoxic more quickly
		Cardiac events: peripartum cardiomyopathy, myocardial infarction, aortic dissection, large-vessel aneurysms
	Hypo/hyperkalaemia and other electrolyte disturbances	No more likely
	Hypothermia	No more likely
4 T's	Thromboembolism	Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction
	Toxicity	Local anaesthetic, magnesium, other
	Tension pneumothorax	Following trauma/suicide attempt
	Tamponade (cardiac)	Following trauma/suicide attempt
Eclampsia and pre-eclampsia		Includes intracranial haemorrhage

4.2.2 Thromboembolism

In the last CEMACH report[1] there were 41 deaths from thromboembolism (33 pulmonary embolism and eight cerebral vein thrombosis), making it the most common cause of direct maternal death. Appropriate use of thromboprophylaxis has improved maternal morbidity and mortality, but improvements in clinical risk assessment and prophylaxis are still required.[1,24]

4.2.3 Amniotic fluid embolism

The estimated frequency of amniotic fluid embolism (AFE) lies somewhere between 1.25/100 000 and 12.5/100 000 maternities, with the most recent UK data giving an incidence of 2/100 000 maternities.[25] Survival rates seem to have improved significantly over time, from 14% in 1979[26] to around 30% in 2005[27] and 80% in 2010,[25] although neurological morbidity in survivors is well recognised.[28] The perinatal mortality

rate in cases of AFE is 135/1000 total births.[25] AFE presents as collapse during labour or delivery or within 30 minutes of delivery in the form of acute hypotension, respiratory distress and acute hypoxia.[29] Seizures and cardiac arrest may occur. There are different phases to disease progression,[28,30] which clearly impacts on maternal survival. Initially, pulmonary hypertension may develop secondary to vascular occlusion either by debris or by vasoconstriction. This often resolves and left ventricular dysfunction or failure develops. Coagulopathy often develops if the mother survives long enough, often giving rise to massive postpartum haemorrhage. If AFE occurs prior to delivery, profound fetal distress develops acutely.[29] The underlying patho-physiological process has been compared to anaphylaxis or severe sepsis.[28] Diagnosis in nonfatal cases is clinical, as there is no established accurate diagnostic test premortem.[31]

4.2.4 Cardiac disease

Cardiac disease was the most common overall cause of maternal death in the CEMACH report,[1] being responsible for 48 maternal deaths. The majority of deaths secondary to cardiac causes occur in women with no previous history.[32] The main cardiac causes of death are myocardial infarction, aortic dissection and cardiomyopathy.[1] The incidence of primary cardiac arrest in pregnancy is much rarer at around 1/30000 maternities, and most cardiac events have preceding signs and symptoms. Aortic root dissection can present in otherwise healthy women, and signs and symptoms such as central chest or interscapular pain, a wide pulse pressure, mainly secondary to systolic hypertension, and a new cardiac murmur must prompt referral to a cardiologist and appropriate imaging. The incidence of congenital and rheumatic heart disease in pregnancy is increasing secondary to increased survival rates owing to improved management of congenital heart disease and increased immigration.[1] These cases should be managed by an appropriately skilled and experienced multidisciplinary team, usually in regional centres. Other cardiac causes include dissection of the coronary artery, acute left ventricular failure, infective endocarditis and pulmonary oedema.

4.2.5 Sepsis

Sepsis has been recognised for centuries as a significant cause of maternal morbidity and mortality, and substandard care continues to feature in the cases that result in death.[1] Bacteraemia, which can be present in the absence of pyrexia or a raised white cell count, can progress rapidly to severe sepsis and septic shock leading to collapse;[33] the most common organisms implicated in obstetrics are the streptococcal groups A, B and D, pneumococcus and *Escherichia coli*.

4.2.6 Drug toxicity/overdose

Drug toxicity/overdose should be considered in all cases of collapse, and illicit drug overdose should be remembered as a potential cause of collapse outside of hospital. In terms of therapeutic drug toxicity, the common sources in obstetric practice are magnesium sulphate in the presence of renal impairment and local anaesthetic agents injected intravenously by accident.

Toxic effects associated with local anaesthetics usually result from excessively high plasma concentrations. Effects initially include a feeling of inebriation and lightheadedness followed by sedation, circumoral paraesthesia and twitching; convulsions can occur in severe toxicity. On intravenous injection, convulsions and cardiovascular collapse may occur very rapidly. Local anaesthetic toxicity resulting from systemic absorption of the local anaesthetic may occur some time after the initial injection. Signs of severe toxicity include sudden loss of consciousness, with or without tonic–clonic convulsions, and cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias can all occur.[34]

In terms of local anaesthetics, total spinal block or high spinal/epidural block are rarer and usually easily recognised causes of collapse.

4.2.7 Eclampsia

Eclampsia as the cause of maternal collapse is usually obvious in the inpatient setting, as often the diagnosis of pre-eclampsia has already been made and the seizure witnessed. Epilepsy should always be considered in cases of maternal collapse associated with seizure activity.

4.2.8 Intracranial haemorrhage

Intracranial haemorrhage is a significant complication of uncontrolled, particularly systolic, hypertension, but can also result from ruptured aneurysms and arteriovenous malformations. The initial presentation may be maternal collapse, but often severe headache precedes this.

4.2.9 Anaphylaxis

Anaphylaxis is a severe, life-threatening generalised or systemic hypersensitivity reaction[35] resulting in respiratory, cutaneous and circulatory changes and, possibly gastrointestinal disturbance and collapse. There is significant intravascular volume redistribution, which can lead to decreased cardiac output. Acute ventricular failure and myocardial ischaemia may occur. Upper airway occlusion secondary to angioedema, bronchospasm and mucous plugging of smaller airways all contribute to significant hypoxia and difficulties with ventilation. Common triggers are a variety of drugs, latex, animal allergens and foods. The incidence is between 3 and 10/1000, with a mortality rate of around 1%.[36] Anaphylaxis is likely when all of the following three criteria are met:

- sudden onset and rapid progression of symptoms
- life-threatening airway and/or breathing and/or circulation problems
- skin and/or mucosal changes (flushing, urticaria, angioedema).

Exposure to a known allergen for the woman supports the diagnosis, but many cases occur with no previous history. Mast cell tryptase levels can be useful.

4.2.10 Other causes

Other causes of maternal collapse include hypoglycaemia and other metabolic/electrolyte disturbances, other causes of hypoxia such as airway obstruction secondary to aspiration/foreign body, air embolism, tension pneumothorax, cardiac tamponade secondary to trauma and hypothermia. There will be other very unusual and rare causes of maternal collapse, but detailed discussion of all causes is beyond the scope of this guideline.

4.3   What are the physiological and anatomical changes in pregnancy that affect resuscitation?

It is essential that anyone involved in the resuscitation of pregnant women is aware of the physiological differences. This includes paramedics and emergency room staff. ✓

The pregnant woman undergoes a variety of physiological changes that accelerate the development of hypoxia and acidosis and make ventilation more difficult.[37] The cardiovascular changes also promote rapid blood loss and reduced oxygen-carrying capacity. These changes are listed in Table 1[38] and, combined with other physical changes, make resuscitation during pregnancy more challenging. It is essential that anyone involved in the resuscitation of a pregnant woman is aware of these differences. This includes paramedics and emergency room staff.

4.3.1 Aortocaval compression

Aortocaval compression significantly reduces cardiac output from 20 weeks of gestation onwards.

From around 20 weeks of gestation onwards, in the supine position the gravid uterus can compress the inferior vena cava and aorta (to a much lesser extent), thus reducing venous return and, as a consequence, cardiac output by up to 30–40%, causing what is known as supine hypotension.[39] Supine hypotension itself can precipitate maternal collapse, which is usually reversed by turning the woman into the left lateral position.

Evidence level 2+

Aortocaval compression significantly reduces the efficacy of chest compressions during resuscitation.

When cardiopulmonary arrest occurs, chest compressions are needed to produce a cardiac output. In nonpregnant women, chest compressions achieve around 30% of the normal cardiac output.[40–42] Aortocaval compression further reduces cardiac output to around 10% that achieved in nonpregnant women.[43] Cardiopulmonary resuscitation (CPR) is less likely to be effective in a woman who is 20 weeks pregnant or more.

Table 1. Physiological and physical changes in pregnancy

	Changes in pregnancy	Impact on resuscitation
Cardiovascular system		
Plasma volume	Increased by up to 50%	Dilutional anaemia Reduced oxygen-carrying capacity
Heart rate	Increased by 15–20 bpm	Increased CPR circulation demands
Cardiac output	Increased by 40% Significantly reduced by pressure of gravid uterus on IVC	Increased CPR circulation demands
Uterine blood flow	10% of cardiac output at term	Potential for rapid massive haemorrhage
Systemic vascular resistance	Decreased	Sequesters blood during CPR
Arterial blood pressure	Decreased by 10–15 mmHg	Decreased reserve
Venous return	Decreased by pressure of gravid uterus on IVC	Increased CPR circulation demands Decreased reserve
Respiratory system		
Respiratory rate	Increased	Decreased buffering capacity, acidosis more likely
Oxygen consumption	Increased by 20%	Hypoxia develops more quickly
Residual capacity	Decreased by 25%	Decreased buffering capacity, acidosis more likely
Arterial PCO2	Decreased	Decreased buffering capacity, acidosis more likely
Laryngeal oedema	Increased	Difficult intubation
Other changes		
Gastric motility	Decreased	Increased risk of aspiration
Lower oesophageal sphincter	Relaxed	Increased risk of aspiration
Uterus	Enlarged	Diaphragmatic splinting reduces residual capacity and makes ventilation more difficult
		Aortocaval compression causes supine hypotension, reduces venous return and significantly impairs CPR
Weight	Increases	Large breasts may interfere with intubation
		Makes ventilation more difficult

CPR = cardiopulmonary resuscitation; IVC = inferior vena cava; PCO2 = partial pressure of carbon dioxide

4.3.2 Respiratory changes

Changes in lung function, diaphragmatic splinting and increased oxygen consumption make the pregnant woman become hypoxic more readily and make ventilation more difficult.

The increased progesterone level in pregnancy increases the respiratory drive,[44,45] leading to an increase in tidal volume and minute ventilation. Splinting of the diaphragm by the enlarged uterus reduces the functional residual capacity and also makes ventilation more difficult. These factors, along with the markedly increased oxygen consumption of the fetoplacental unit, mean that the pregnant woman becomes hypoxic much more rapidly during periods of hypoventilation.

<div style="text-align: right">Evidence level 2+</div>

4.3.3 Intubation

Difficult intubation is more likely in pregnancy.

Weight gain in pregnancy, large breasts inhibiting the working space and laryngeal oedema can all contribute to make intubation more difficult.

<div style="text-align: right">Evidence level 3</div>

4.3.4 Aspiration

Pregnant women are at an increased risk of aspiration.

The pregnant woman is at a significantly higher risk of regurgitation and aspiration secondary to the progesterone effect relaxing the lower oesophageal sphincter and delayed gastric emptying, along with the raised intra-abdominal pressure secondary to the gravid uterus. Aspiration pneumonitis in the pregnant woman, known as Mendelson syndrome,[46] can be severe, particularly as the gastric pH is lower than in the nonpregnant population. Early intubation with effective cricoid pressure and the use of H_2 antagonists and antacids prophylactically in all women considered to be at high risk of obstetric intervention during labour is advised.

Evidence level 2+

4.3.5 Circulation

The increased cardiac output and hyperdynamic circulation of pregnancy mean that large volumes of blood can be lost rapidly, especially from the uterus, which receives 10% of the cardiac output at term. Otherwise healthy women tolerate blood loss remarkably well, and can lose up to 35% of their circulation before becoming symptomatic. Blood loss is tolerated less well if there is a pre-existing maternal anaemia, and clotting is less efficient if there is a significant anaemia. Concealed bleeding and underestimation of loss mean that intervention is often delayed. Where signs of hypovolaemia have been subtle, hypovolaemia as the cause of maternal cardiopulmonary arrest may go unrecognised, particularly where blood loss has been concealed.

Evidence level 3

4.4 What is the optimal initial management of maternal collapse?

Maternal resuscitation should follow the Resuscitation Council (UK) guidelines using the standard A, B, C approach, with some modification.

✓

In the UK, resuscitation is conducted according to the UK Resuscitation Council Guidelines: basic life support (BLS), adult advanced life support (ALS) and automated external defibrillation (AED) algorithms and recommendations.[47,48] These guidelines were updated in 2010 by international experts under the auspices of the International Liaison Committee on Resuscitation[49] and are used in the resuscitation of the pregnant woman.

Evidence level 1++

It is recognised that the divisions into basic and advanced life support are somewhat arbitrary in the hospital setting.[50–52]

In the community setting, basic life support should be administered and rapid transfer arranged, unless appropriate personnel and equipment are available.

✓

In the pregnant woman of 20 weeks or more gestation, adaptations are made to the resuscitation process. There are also algorithms for special patient groups. While algorithms for generic, paediatric and neonatal life support are available in standardised posters, adaptations for maternal resuscitation are addressed but are not available in algorithmic and poster form. For this reason, the Resuscitation Council (UK) algorithm for advanced life support has been modified by the authors (Appendix 2).

There are essential adaptations to the management of the collapsed pregnant woman because of the physiological and anatomical changes of pregnancy.

4.4.1 Tilt

From 20 weeks of gestation onwards, the pressure of the gravid uterus must be relieved from the inferior vena cava and aorta.

C

A left lateral tilt of 15° on a firm surface will relieve aortocaval compression in the majority of pregnant women and still allow effective chest compressions to be performed.[53]

<div style="float:right">**C**</div>

A left lateral tilt of 15° can be achieved on an operating table using a Cardiff wedge[54] by having someone kneel on the right side of the woman with their knees under the woman's thorax, although this has the disadvantage of the tilt being removed for defibrillation;[54,55] alternatives are using an upturned chairback or using manual displacement of the uterus to the left.

<div style="float:right">*Evidence level 2+*</div>

In cases of major trauma, the wedge should be placed under the spinal board. In the absence of a spinal board, manual displacement of the uterus should be used. Using soft surfaces such as a bed or objects such as pillows or blankets is not nearly as effective and compromises effective chest compressions, but is better than leaving the woman supine.

4.4.2 Airway

The airway should be protected as soon as possible by intubation with a cuffed endotracheal tube.

<div style="float:right">✓</div>

In pregnancy, the airway is more vulnerable because of the increased risk of regurgitation and aspiration. For this reason it is important to clear and protect the airway as early as possible. Intubation should then be performed as soon as possible. This will protect the airway, ensure good oxygen delivery and facilitate more efficient ventilation. Intubation can be more difficult in pregnancy, so this should be undertaken by someone with the appropriate skills. During cardiac arrest in the nonpregnant patient it is acceptable to use a supraglottic device such as the laryngeal mask airway as an alternative to the tracheal tube.[49] However, it should be emphasised that the pregnant woman is more likely to regurgitate and aspirate in the absence of a secured airway (tracheal tube) than the nonpregnant patient, and that the early involvement of an appropriately skilled anaesthetist remains best practice. Capnography is recurrent in the intubated patient.[49]

Box 1. Suggested equipment for airway management[56]

Recommended equipment for routine airway management:
- Facemasks
- Oropharyngeal airways: three sizes
- Nasopharyngeal airways: three sizes
- Laryngeal mask airways
- Tracheal tubes in a range of sizes
- Two working laryngoscope handles
- Macintosh blades: sizes 3 and 4
- Tracheal tube introducer ('gum-elastic' bougie)
- Malleable stylet
- Magill forceps

Recommended equipment for management of unanticipated difficult intubation
- Difficult Airway Society guidelines algorithm flowcharts (or modified local version)
- Equipment list for restocking
- At least one alternative blade (e.g. straight, McCoy)
- Intubating laryngeal mask airway (ILMA™) set (sizes 3, 4 and 5 with dedicated tubes and pusher)
- Tracheal tubes – reinforced and microlaryngeal, sizes 5 mm and 6mm
- Flexible fibreoptic laryngoscope (with portable/battery light source)
- Proseal laryngeal mask airway (ProSeal LMA™)
- Cricothyroid cannula (e.g. Ravussin) with a high-pressure jet ventilation system (e.g. Manujet) or large-bore cricothyroid cannula (e.g. Quicktrac)
- Surgical cricothyroidotomy kit
- (Scalpel with no. 20 blade, tracheal hook, 6/7 mm tracheal and tracheostomy tubes)

Alternative specialised techniques of proven value[57]
- Bullard-type laryngoscope
- Trachlight
- Aintree intubation catheter
- Combitub

Box 1 lists suggested equipment that should be available for cases where airway management may be difficult.[56,57]

4.4.3 Breathing

Supplemental oxygen should be administered as soon as possible. ☑

Because of the increased oxygen requirements and rapid onset of hypoxia in pregnancy, it is important to ensure optimal oxygen delivery by adding high-flow 100% oxygen to whatever method of ventilation is being employed.

Bag and mask ventilation should be undertaken until intubation can be achieved. ☑

Ventilation, by face mask, by a supraglottic airway device and self-inflating bag or by a cuffed endotracheal tube, may be more difficult because of the physiological changes of pregnancy described above. It can also be difficult to see the chest rise.

4.4.4 Circulation

In the absence of breathing despite a clear airway, chest compressions should be commenced immediately. | B

Chest compressions should not be delayed by palpating for a pulse, but should be commenced immediately in the absence of breathing and continued until the cardiac rhythm can be checked and cardiac output confirmed. Compressions may be made difficult because of obesity and the tilted position. Hand position should be over the centre of the chest, and it is important to ensure that the direction of compression is perpendicular to the chest wall, thus the angle of tilt must be taken into account. Compressions should be performed at a ratio of 30:2 ventilations unless the woman is intubated, in which case chest compressions and ventilations should be desynchronised, with compressions being performed at a rate of 100/minute and ventilations at a rate of 10/minute.[19] Because chest compressions are not as effective after 20 weeks of gestation, there should be early recourse to delivery of the fetus and placenta if CPR is not effective. | Evidence level 2++

Two wide-bore cannulae should be inserted as soon as possible. ☑

There should be an aggressive approach to volume. ☑

Haemorrhage is the most common cause of maternal collapse and a consequence of other causes of collapse. There must be a high index of suspicion for bleeding and awareness of the limitations of clinical signs. Caution must be exercised in the presence of severe pre-eclampsia and eclampsia, where fluid overload can contribute to poor outcome. In the case where both significant haemorrhage and pre-eclampsia/eclampsia exist, careful fluid management is essential.

Abdominal ultrasound by a skilled operator can assist in the diagnosis of concealed haemorrhage. | C

Very occasionally, ultrasound by a skilled operator can aid diagnosis in cases of massive abruption and intra-abdominal bleeding, although laparotomy should not be delayed if the findings are negative or the index of suspicion is high.[58,59] However, this should not interfere with the resuscitation process. | Evidence level 2+

The same defibrillation energy levels should be used as in the nonpregnant patient. | B

If defibrillation is required, the same settings should be used as in the nonpregnant patient as there is no change in thoracic impedance.[60] Adhesive defibrillator pads are preferable to defibrillator | Evidence level 2++

paddles, and the left defibrillation pad should be applied lateral to the left breast. The energy from the defibrillation shock is directed across the heart and there is no evidence that shocks from a direct current defibrillator have an adverse effect on the fetus. Uterine monitors should be removed before shock delivery.

<div style="text-align: right;">Evidence level 2++</div>

4.4.5 Drugs

There should normally be no alteration in algorithm drugs or doses.

✓

Common, reversible causes of maternal cardiopulmonary arrest should be considered throughout the resuscitation process.

D

Throughout the resuscitation process, consideration should be given to the cause of the collapse, so that continuing therapy can be directed towards the specific cause to optimise outcome.[19]

<div style="text-align: right;">Evidence level 4</div>

Resuscitation efforts should be continued until a decision is taken by the consultant obstetrician, and consultant anaesthetist in consensus with the cardiac arrest team.

✓

4.5 When, where and how should perimortem caesarean section be performed?

If there is no response to correctly performed CPR within 4 minutes of maternal collapse or if resuscitation is continued beyond this in women beyond 20 weeks of gestation, delivery should be undertaken to assist maternal resuscitation. This should be achieved within 5 minutes of the collapse.

D

The concept of perimortem caesarean section was introduced in 1986,[61] along with the recommendation that it be initiated after 4 minutes of maternal cardiopulmonary arrest if resuscitation is ineffective, and be achieved within 5 minutes of collapse. The rationale for this timescale is that the pregnant woman becomes hypoxic more quickly than the nonpregnant woman, and irreversible brain damage can ensue within 4–6 minutes. The gravid uterus impairs venous return and reduces cardiac output secondary to aortocaval compression. Delivery of the fetus and placenta reduces oxygen consumption, improves venous return and cardiac output, facilitates chest compressions and makes ventilation easier. It also allows the heart to be compressed easily through the diaphragm against the chest wall by placing the hand behind the heart (with the diaphragm closed) and compressing it against the posterior aspect of the anterior chest wall. This improves cardiac output beyond that achieved with closed chest compressions.[62] Before 20 weeks of gestation there is no proven benefit from delivery of the fetus and placenta. Perimortem caesarean section should be considered a resuscitative procedure to be performed primarily in the interests of maternal, not fetal, survival.

<div style="text-align: right;">Evidence level 4</div>

Delivery within 5 minutes of maternal collapse improves the chances of survival for the baby, but this is not the reason for delivery. If maternal resuscitation continues beyond 4 minutes of the collapse, delivery of the fetus and placenta should be performed as soon as possible to aid this, even if the fetus is already dead. There is, of course, the possibility that the outcome could be that of a severely damaged surviving child, but the interests of the mother must come first.

Perimortem caesarean section should not be delayed by moving the woman – it should be performed where resuscitation is taking place.

✓

Time should not be wasted by moving the woman to an operating theatre; a perimortem caesarean section can be performed anywhere, with a scalpel being the only essential equipment required. With no circulation, blood loss is minimal and no anaesthetic is required. If resuscitation is successful following delivery, there should be prompt transfer to an appropriate environment at that point, as well as anaesthesia and sedation, to control ensuing haemorrhage and complete the operation. The doctrine of 'the best interests of the patient' would apply to conduct of this procedure being carried out without consent.

The operator should use the incision that will facilitate the most rapid access. ☑

In terms of the best incision to use, a midline abdominal incision and a classic uterine incision will give the most rapid access, but many will be unfamiliar with this approach and, as delivery can be achieved rapidly with a transverse approach, the operator should use the approach they are most comfortable with. If resuscitation is successful, the uterus and abdomen should be closed in the usual way to control blood loss and minimise the risk of infection. Where the outcome is not successful, the case should be discussed with the coroner/procurator fiscal to determine whether a postmortem is required before any medical devices such as lines and endotracheal tubes are removed, as per the Royal College of Pathologists recommendations.[63,64]

A perimortem caesarean section tray should be available on the resuscitation trolley in all areas where maternal collapse may occur, including the accident and emergency department. ☑

To ensure there are no delays in executing a perimortem caesarean section when indicated, the equipment necessary should be immediately available on the resuscitation trolley. All that is required is a fixed blade scalpel and two clamps for the cord. In the absence of a specific tray, a scalpel alone will enable delivery of the fetus and placenta and cutting the cord, which can then be manually compressed until a clamp is found if the baby is alive.

4.6 What does the continuing management consist of?

Senior staff with appropriate experience should be involved at an early stage. ☑

Transfer should be supervised by an adequately skilled team with appropriate equipment. ☑

Continuing management depends very much on the underlying cause of the collapse, and appropriate senior staff must be involved early. It is essential the woman is transferred to an appropriate environment to ensure optimal contiuing care. This would usually mean transfer to a high-dependency/critical care area with appropriate staff and monitoring facilities.[65]

4.6.1 Haemorrhage
The continuing management of major postpartum haemorrhage is comprehensively covered in the RCOG Green-top Guideline No. 52: *Prevention and Management of Postpartum Haemorrhage*.[66]

In the case of maternal collapse secondary to antepartum haemorrhage, the fetus and placenta should be delivered promptly to allow control of the haemorrhage. ☑

In the case of massive placental abruption, caesarean section may occasionally be indicated even if the fetus is dead to allow rapid control of the haemorrhage. ☑

Management of collapse secondary to massive haemorrhage as a result of placenta praevia should be managed in accordance with the RCOG Green-top Guideline No. 27: *Placenta Praevia, Placenta Praevia Accreta and Vasa Praevia: Diagnosis and Management*.[67]

4.6.2 Venous thromboembolism
The specific management of massive pulmonary embolism is covered in the RCOG Green-top Guideline No. 28: *Thromboembolic Disease in Pregnancy and the Puerperium: Acute Management*.[68]

4.6.3 Amniotic fluid embolism

The management of AFE is supportive rather than specific, as there is no proven effective therapy.[2] ☑

Early involvement of senior experienced staff, including obstetrician, anaesthetist, haematologist and intensivist, is essential to optimise outcome. ☑

On top of resuscitation and supportive measures, arrhythmias may develop and will require standard treatment. Inotropic support is likely to be needed and measurement of cardiac output may help direct therapy and avoid fluid overload, as this will exacerbate pulmonary oedema and increases the risk of acute respiratory distress syndrome. High filling pressures are indicative of a failing left ventricle.

Coagulopathy needs early, aggressive treatment, including aggressive use of fresh frozen plasma. ☑

If undelivered, delivery of the fetus and placenta should be performed as soon as possible. The incidence of uterine atony is increased in this condition and contributes to the postpartum haemorrhage. This should be managed as stated in the RCOG Green-top Guideline No. 52: *Prevention and Management of Postpartum Haemorrhage.*[66]

Various other therapies have been tried, including steroids, heparin, plasmapheresis and haemofiltration, usually in single cases. As such, there is no robust evidence to support their use.[27]

4.6.4 Cardiac disease

After successful resuscitation, cardiac cases should be managed by an expert cardiology team. ☑

After initial resuscitation, the continuing management of cardiac disease is similar to that in the nonpregnant state, although in many cases delivery will be necessary to facilitate this.

Although thrombolysis can be associated with significant bleeding from the placental site, it should be given to women with acute coronary insufficiency, although caution should be exercised in the perioperative period.[69] If available, percutaneous angioplasty allows accurate diagnosis and definitive therapy.

Evidence level 4

4.6.5 Sepsis

Septic shock should be managed in accordance with the Surviving Sepsis Campaign guidelines. **A**

The Surviving Sepsis Campaign has updated the management of sepsis and septic shock.[70] The speed and appropriateness of therapy administered in the initial hours after severe sepsis develops are likely to influence outcome with early resuscitation improving survival rates. A multidisciplinary team approach is required including midwives, consultant obstetricians, consultant anaesthetists, consultant haematologists, consultant intensivists and consultant microbiologists. The following 'care bundle' should be applied immediately or within 6 hours, and has been shown to significantly improve survival rates:[71-73]

1. Measure serum lactate.
2. Obtain blood cultures/culture swabs prior to antibiotic administration.
3. Administer broad-spectrum antibiotic(s) within the first hour of recognition of severe sepsis and septic shock according to local protocol
4. In the event of hypotension and/or lactate >4 mmol/l:
   a) deliver an initial minimum of 20 ml/kg of crystalloid/ colloid
   b) once adequate volume replacement has been achieved, a vasopressor (norepinephrine, epinephrine) and/or an inotrope (e.g. dobutamine) may be used to maintain mean arterial pressure over 65 mmHg.

Further management consists of:

5. In the event of hypotension despite fluid resuscitation (septic shock) and/or lactate over 4 mmol/l:
   a) achieve a central venous pressure of at least 8 mmHg (or over 12 mmHg if the woman is mechanically ventilated) with aggressive fluid replacement
   b) consider steroids.
6. Maintain oxygen saturation with facial oxygen. Consider transfusion if haemoglobin is below 7g/dl.

Evidence level 1+

Continuing management involves continued supportive therapy, removing the septic focus, administration of blood products if required and thromboprophylaxis.[71]

4.6.6 Drug overdose/toxicity

Many drug overdoses have specific therapy dependent on the drug in question, and appropriate help should be sought in the management of such cases. In obstetric practice, the two main drugs that can give rise to overdose or toxic problems are magnesium sulphate and local anaesthetic agents.

4.6.6.1 Magnesium sulphate

The antidote to magnesium toxicity is 10 ml 10% calcium gluconate given by slow intravenous injection.

4.6.6.2 Local anaesthetic agents

If local anaesthetic toxicity is suspected, stop injecting immediately.   ✓

Lipid rescue should be used in cases of collapse secondary to local anaesthetic toxicity.   C

Intralipid 20% should be available in all maternity units.   ✓

Treatment of cardiac arrest with lipid emulsion[34,73] consists of an intravenous bolus injection of Intralipid 20% 1.5 ml.kg^{-1} over 1 minute (100 ml for a 70 kg woman) followed by an intravenous infusion of Intralipid 20% at 0.25 ml.kg^{-1} min^{-1} (400 ml over 20 minutes for a 70 kg woman). The bolus injection can be repeated twice at 5-minute intervals if an adequate circulation has not been restored (a further two boluses of 100 ml at 5-minute intervals for a 70 kg woman). After another 5 minutes, the infusion rate should be increased to 0.5 ml.kg^{-1} min^{-1} if adequate circulation has not been restored. CPR should be continued throughout this process until an adequate circulation has been restored, and this may take over an hour.

Evidence level 2+

Manage arrhythmias as usual, recognising that they may be very refractory to treatment.   ✓

Prolonged resuscitation may be necessary, and it may be appropriate to consider other options. The first-line treatment should be lipid emulsion, but if the facilities are available, some may consider the use of cardio-pulmonary bypass.

A copy of the guidance that can be put on the wall locally can be found at http://www.aagbi.org/publications/guidelines/docs/latoxicity07.pdf.

All cases of lipid rescue should be reported to the National Patient Safety Agency (www.npsa.nhs.uk) and to the Lipid Rescue site (www.lipidrescue.org).   ✓

4.6.7 Eclampsia

Eclampsia should be managed in accordance with the RCOG Green-top Guideline No. 10(A): *The Management of Severe Pre-eclampsia/Eclampsia*.[74]

4.6.8 Intracranial haemorrhage

Expert neuroradiology is required to establish an accurate diagnosis. Management is the same as in the nonpregnant state, although delivery may be necessary to facilitate this. The appropriate experts should be involved at the earliest opportunity.

4.6.9 Anaphylaxis

In cases of anaphylaxis, all potential causative agents should be removed, and the A, B, C, D, E approach followed.[36,75]   A

If the anaphylactic reaction occurs in the community, the woman should receive basic life support and be transferred to a hospital setting as quickly as possible, unless a suitably trained healthcare professional is present with appropriate equipment/drugs, in which case definitive resuscitation and treatment should be commenced.

✓

The definitive treatment for anaphylaxis is 500 micrograms (0.5 ml) of 1:1000 adrenaline intramuscularly. PLEASE NOTE THIS DOSE IS FOR INTRAMUSCULAR USE ONLY.

A

Adrenaline treatment can be repeated after 5 minutes if there is no effect.[36,75] In experienced hands it can be given intravenously as a 50 microgram bolus (0.5 ml of 1:10 000 solution). Adjuvant therapy consists of chlorpheniramine 10 mg and hydrocortisone 200 mg. Both are given intramuscularly or by slow intravenous injection.[36,75]

Evidence level 1+

4.7   What are the outcomes for mother and baby?

Owing to the lack of robust population data, it is not possible to be accurate regarding outcomes. It is widely accepted that there is significant selection bias in publications relating to the topic. The Confidential Enquiries into Maternal Death have given robust data when resuscitation was not successful, and the Scottish Maternal Morbidity data[3] and the Dublin study[4] report maternal survival figures for severe maternal morbidity, but not for collapse per se. These data do give 'near miss' to death ratios of 56:1[3] and 79:1.[4] For some conditions such as AFE, the maternal survival figures are more robust, but accurate data collection is required for maternal collapse as a whole.

In 2005, Katz et al. reviewed maternal and fetal outcomes for perimortem caesarean section over an 18-year period from 1986 to 2004.[76] There were 38 procedures, 30 of which resulted in surviving babies between 25 and 42 weeks of gestation, with intact survival most likely with a collapse to delivery interval of 5 minutes or less. In 18 cases, the cause of the collapse was felt to be irreversible. Of the 20 cases in which the cause of collapse was known and felt to be reversible, 13 women survived, giving a survival rate of 65%. The paper also demonstrated the positive effect of the delivery on the maternal circulation, supporting their original advice of achieving delivery within 5 minutes of collapse if CPR is ineffective,[61] which was based on theory and a single case report.

The latest CEMACH report[1] details the neonatal outcomes of the 52 perimortem or postmortem sections that were performed in which the mothers did not survive. Fifty-four percent were liveborn, although eight out of these 28 babies died in the early neonatal period. Neonatal survival is associated with advanced gestation and delivery within a delivery suite or critical care setting, and not the emergency department.

There have been successful cases of somatic support after maternal brain death to facilitate neonatal outcome,[77] the longest being from 15 weeks to delivery at 32 weeks.[78] This process is not without difficulties, both in medical terms and ethically,[79] and what is not known is how many such cases have not been successful. In view of the complex nature of such cases, a multidisciplinary discussion, including the family, should be conducted in each case.

4.8   Who should be on the team?

In addition to the general arrest team, there should be a senior midwife, an obstetrician and an obstetric anaesthetist included in the team in cases of maternal collapse.

✓

If the maternity unit is an integral part of a general hospital, the maternal cardiopulmonary resuscitation team should be the hospital cardiopulmonary arrest team with the addition of:
- a senior midwife
- the most senior resident obstetrician – usually ST 3–7
- a resident anaesthetist who has recognised skills in obstetric anaesthesia – usually ST 3–7.

This will mean that the request needs to be specific with common terminology, so that the switchboard operators know exactly who to call. While managing the arrest, there must be dialogue between the team leader, the obstetrician and the obstetric anaesthetist as to how best to manage the pregnant woman.

In stand-alone consultant-led maternity units, or those that are geographically distant from the main general hospital, the entire arrest team is often made up of staff from within the maternity unit. In this case, the team is usually made up of senior midwifery staff, operating department practitioners, resident obstetric staff and the resident obstetric anaesthetist.

The consultant obstetrician and consultant obstetric anaesthetist should be summoned at the time of the cardiopulmonary arrest call. ✓

The neonatal team should be called early if delivery is likely (antepartum collapse over 22 weeks of gestation). ✓

Where the woman survives, a consultant intensivist should be involved as soon as possible. ✓

In a stand-alone midwifery unit or a homebirth environment, the midwifery staff should provide life support and call a 999 ambulance to transfer the woman to the nearest appropriate environment. Maternity services that include a stand-alone midwifery unit should ensure that there is a written agreement with the ambulance service confirming the emergency status of a 999 call from the midwifery unit, which must not be considered a place of safety as an NHS facility

5. Clinical governance

5.1 Documentation

Accurate documentation in all cases of maternal collapse, whether or not resuscitation is successful, is essential. ✓

Poor documentation remains a problem in all aspects of medicine, and can have potential medico-legal consequences.[80] Contemporaneous note-keeping is difficult in a resuscitation situation, unless someone is scribing. Those involved should then write full notes as soon as possible after the event.

5.2 Incident reporting

All cases of maternal collapse should generate a clinical incident form and the care should be reviewed through the clinical governance process. ✓

Maternal collapse is a rare and potentially devastating event, and substandard care continues to be high-lighted.[1-4] In all cases of maternal collapse, care should be reviewed to ensure individual and organisational learning, and also to reassure staff and the family when care has been optimal.

In view of the significant reduction in maternal mortality over the years, robust population-based data regarding maternal collapse through a national reporting system would render valuable information about management and outcomes.

All cases of maternal death should be reported to CMACE. D

National reporting and scrutiny of maternal deaths continues to provide valuable information and learning.

5.3 Training

All generic life support training should make mention of the adaptation of CPR in the pregnant woman. ✓

All front-line staff must be aware of the adaptations for CPR in pregnancy. This includes paramedics who will deal with collapse in the community setting and accident and emergency department personnel as well as staff within a maternity unit.

All maternity staff should have annual formal training in generic life support and the management of maternal collapse. ✓

The RCOG, the Royal College of Midwives and CEMACH[1] recommend that all staff undergo annual training in obstetric emergencies, and this is now included in the NHS Litigation Authority,[81] Clinical Negligence and Other Risks (Non-clinical) Indemnity Scheme[82] and Welsh Pool Risk[83] maternity standards.

Life support training reduces morbidity and mortality. **A**

Small-group interactive practical training is recommended. **C**

A systematic review of life support training showed a significant reduction in morbidity and mortality,[84] and there is now a wealth of evidence emerging to show that multidisciplinary training in obstetric emergencies improves outcomes.[85-88]

Evidence level 1+

The best method of training is not clear,[89] although there is evidence to support small-group interactive training.[90] Various courses exist,[50-52] and have been evaluated well by those undertaking them.[91] When asked, those who participated felt that such courses significantly increased their confidence in managing obstetric emergencies.[92] Where outcomes have been seen to improve after the introduction of training, it appears that the elements of multiprofessional training of all staff and integrating teamwork training with clinical teaching are important.[87,88,93,94] The ideal frequency of training is not clear, but this should occur at least annually for all staff.[95,96] Despite all this evidence in support of training, it cannot be assumed that the presence of training equates to the receipt of training,[97] and this remains a challenge.

5.4 Debriefing

Debriefing is recommended for the woman, her family and the staff involved in the event. ✓

Maternal collapse can be associated with post-traumatic stress disorder, postnatal depression and tocophobia. Family and staff members should not be forgotten. Debriefing is an important part of holistic maternity care and should be offered by a competent professional.[98]

6. Auditable standards

- Proportion of staff undergoing annual training in life support.
- Proportion of staff undergoing annual training in maternal collapse.
- Audit of the management of maternal collapse.
- Compliance with incident reporting.
- Achievement of perimortem caesarean section within 5 minutes of collapse on hospital premises where there is no response to resuscitation.
- Presence of a perimortem caesarean section tray on resuscitation trolleys.
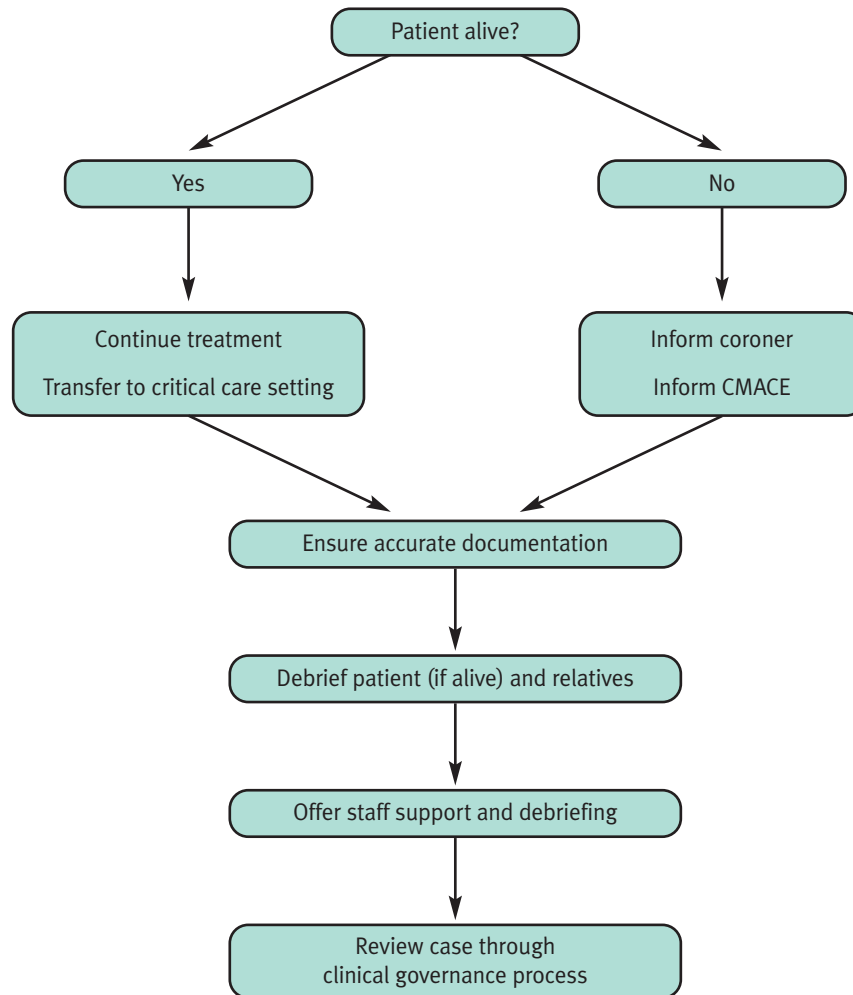
References

1. Confidential Enquiry into Maternal and Child Health. *Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer – 2003–2005. The Seventh Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom*. London: CEMACH; 2007 [http://www.cmace.org.uk/getattachment/927cf18a-735a-47a0-9200-cdea103781c7/Saving-Mothers–Lives-2003-2005_full.aspx].

2. Knight M, Kurinczuk JJ, Tufnell D, Brocklehurst P. The UK Obstetric Surveillance System for rare disorders of pregnancy. *BJOG* 2005:112:263–5.

3. NHS Quality Improvement Scotland. *Scottish Confidential Audit of Severe Maternal Morbidity. 5th Annual Report*. Edinburgh: NHS Quality Improvement Scotland; 2007 [http://www.nhshealthquality.org/nhsqis/files/SCASMM_ANNREP2007_MAY09.pdf].

4. Murphy CM, Murad K, Deane R, Byrne B, Geary M, McAuliffe FM. Severe maternal morbidity for 2004–2005 in the three Dublin maternity hospitals. *Eur J Obstet Gynecol Reprod Biol* 2009;143:34–7.

5. Royal College of Obstetricians and Gynaecologists. Clinical Governance Advice No. 1c: *Development of RCOG Green-top Guidelines: Producing a Clinical Practice Guideline*. London: RCOG; 2006 [http://www.rcog.org.uk/files/rcog-corp/Clinical%20governance%20Advice%201c.pdf].

6. Knaus WA, Zimmerman JE, Wagner DP, Draper EA, Lawrence DE. APACHE – acute physiology and chronic health evaluation: a physiologically based classification system. *Crit Care Med* 1981;9:591–7.

7. Lee A, Bishop G, Hillman KM, Daffurn K. The Medical Emergency Team. *Anaesth Intensive Care* 1995;23:183–6.

8. Morgan RJM, Williams F, Wright MM. An early warning scoring system for detecting developing critical illness. *Clin Intensive Care* 1997;8:100–1.

9. Kause J, Smith G, Prytherch D, Parr M, Flabouris A, Hillmak K; Australian and New Zealand Intensive Care Society Clinical Trials Group. A comparison of antecedents to cardiac arrests, deaths and emergency intensive care admissions in Australia and New Zealand, and the United Kingdom – the ACADEMIA study. *Resuscitation* 2004;62:275–82.

10. Watkinson PJ, Barber VS, Price JD, Hann A, Taressenko L, Young JD. A randomised controlled trial of the effect of continuous electronic physiological monitoring on the adverse event rate in high risk medical and surgical patients. *Anaesthesia* 2006;61:1031–9.

11. Jacques T, Harrison GA, McLaws ML, Kilborn G. Signs of critical conditions and emergency responses (SOCCER): a model for predicting adverse events in the inpatient setting. *Resuscitation* 2006;69:175–83.

12. Stenhouse C, Coates S, Tivey M, Allsop P, Parker T. Prospective evaluation of a modified Early Warning Score to aid earlier detection of patients developing critical illness on a general surgical ward. *Br J Anaesth* 2000;84:663.

13. Goldhill DR, McNarry AF, Mandersloot G, McGinley A. A physiologically-based early warning score for ward patients: the association between score and outcome. *Anaesthesia* 2005;60:547–53.

14. Subbe CP, Slater A, Menon D, Gemmell L. ASSIST: a screening tool for critically ill patients on general medical wards. *Intensive Care Med* 2002;28:S21.

15. Gao H, McDonnell A, Harrison DA, Moore T, Adam S, Daly K, et al. Systematic review and evaluation of physiological track and trigger warning systems for identifying at-risk patients on the ward. *Intensive Care Med* 2007;33:667–9.

16. Hillman K, Chen J, Creitkos M, Bellomo R, Brown D, Doig G, et al.; Merit Study Investigators. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. *Lancet* 2005;365:2091–7.

17. National Institute for Health and Clinical Excellence. NICE clinical guideline 50: *Acutely ill patients in hospital. Recognition of and response to acute illness in adults in hospital*. London: NICE; 2007 [http://www.nice.org.uk/nicemedia/live/11810/35950/35950.pdf].

18. Gopalan PD, Muckhart DJ. The critically ill obstetric patient: what's the score? *Int J Obstet Anesth* 2004;13:144–5.

19. Resuscitation Council (UK). Resuscitation Guidelines 2005 [http://www.resus.org.uk/pages/guide.htm].

20. Brace V, Kernaghan D, Penney G. Learning from adverse clinical outcomes: major obstetric haemorrhage in Scotland, 2003–05. *BJOG* 2007:114;1388–96.

21. Selo-Ojeme DO, Welch CC. Review: spontaneous rupture of splenic artery aneurysm in pregnancy. *Eur J Obstet Gynecol Reprod Biol* 2003;109:124–7.

22. Glover P. Blood losses at delivery: how accurate is your estimation? *Aust J Midwifery* 2003;16;21–4.

23. Toledo P, McCarthy RJ, Hewlett BJ, Fitzgerald PC, Wong CA. The accuracy of blood loss estimation after simulated vaginal delivery. *Anaesth Analg* 2007;105:1736–40.

24. Knight M; UKOSS. Antenatal pulmonary embolism: risk factors, management and outcomes. *BJOG* 2008;115:453–61.

25. Knight M, Tuffnell D, Brocklehurst P, Spark P, Kurinczuk JJ; UK Obstetric Surveillance System. Incidence and risk factors for amniotic-fluid embolism. *Obstet Gynecol* 2010;115:910–7.

26. Morgan M. Amniotic fluid embolism. *Anaesthesia* 1979;4:20–32.

27. Tufnell DJ. United Kingdom amniotic fluid embolism register. *BJOG* 2005;112:1625–9.

28. Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol* 1995;172:1158–67.

29. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. *Am J Obstet Gynecol* 2009;201:445.e1-13.

30. Gei AF, Vadhera RB, Hankins GD. Embolism during pregnancy: thrombus, air, and amniotic fluid. *Anesthesiol Clin North America* 2003;21:165–82.

31. Kramer MS, Rouleau J, Baskett TF, Joseph KS; Maternal Health Study Group of the Canadian Perinatal Surveillance System. Amnotic-fluid embolism and medical induction of labour: a retrospective, population-based cohort study. *Lancet* 2006;368:1444–8.

32. Malhotra S, Yentis SM. Reports on Confidential Enquiries into Maternal Deaths: management strategies based on trends in maternal cardiac deaths over 30 years. *Int J Obstet Anesth* 2006;15:223–6.

33. Levy MM, Fink MP, Marshall JC, Abraham E, Angus D, Cook D, et al.; International Sepsis Definitions Conference. 2001 SCCM/ESICM/ACCP/ATS/SIS International Sepsis Definitions Conference. *Intensive Care Med* 2003;29:530–8.

34. Foxall G, McCahon R, Lamb J, Hardman JG, Bedforth NM. Levobupivacaine-induced seizures and cardiovascular collapse treated with Intralipid. *Anaesthesia* 2007;62:516–8.

35. Johansson SG, Bieber T, Dahl R, Friedmjann PS, Lanier BQ, Lockey RF, et al. Revised nomenclature for allergy for global use: Report of the Nomenclature Review Committee of the World Allergy Organization. *J Allerg Clin Immunol* 2004;113:832–6.

36. Working Group of the Resuscitation Council (UK). *Emergency treatment of anaphylactic reactions. Guidelines for healthcare providers*. London: Resuscitation Council (UK); 2008 [http://www.resus.org.uk/pages/reaction.pdf].

37. Liberatore SM, Pistelli R, Patalano F, Moneta E, Incalzi RA, Ciappi G. Respiratory function during pregnancy. *Respiration* 1984;46:145–50.

38. Whitty JE. Maternal cardiac arrest in pregnancy. *Clin Obstet Gynecol* 2002;45:377–92.

39. Chesnutt AN. Physiology of normal pregnancy. *Crit Care Clin* 2004;20:609–15.

40. Sanders AB, Meislin HW, Ewy GA. The physiology of cardio-pulmonary resuscitation. An update. *JAMA* 1984;252:3283–6.

41. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 3: adult basic life support. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. *Circulation* 2000:102:22–59.

42. Donegan JH. *Cardiopulmonary resuscitation*. Springfield, IL: Charles C Thomas; 1982.

43. Kerr MG. The mechanical effects of the gravid uterus in late pregnancy. *J Obstet Gynaecol Br Commonw* 1965;2:513–29.

44. Prowse CM, Gaensler EA. Respiratory and acid-base changes during pregnancy. *Anesthesiology* 1965;26;381–92.

45. Skatrud JB, Dempsey JA, Kaiser DG. Ventilatory response to medroxyprogesterone acetate in normal subjects: time course and mechanism. *J Appl Physiol* 1978;44:393–44.

46. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anaesthesia. *Am J Obstet Gynecol* 1946;52:191–205.

47. Resuscitation Council (UK). Resuscitation Guidelines 2010 [http://www.resus.org.uk/pages/guide.htm].

48. Soar J, Deakin CD, Nolan JP, Abbas G, Alfonzo A, Handley AJ, et al.; European Resuscitation Council. European Resuscitation Council Guidelines for Resuscitation 2005. Section 7. Cardiac arrest in special circumstances. *Resuscitation* 2005;67 Suppl 1:S135–70 [https://www.erc.edu/index.php/guidelines_download_2005/en/].

49. Nolan JP, Soar J, Zideman DA, Biarent D, Bossaert LL, Deakin CD, et al; ERC Guidelines Writing Group. European Resuscitation Council Guidelines for Resuscitation 2010 Section 1. Executive Summary. *Resuscitation* 2010;81:1219–76 [http://www.ilcor.org/en/publications/publications/].

50. PRactical Obstetric MultiProfessional Training (PROMPT) [www.prompt-course.org].

51. Advanced Life Support in Obstetrics (ALSO). Provider Course [http://www.aafp.org/online/en/home/cme/aafpcourses/clinicalcourses/also.html].

52. Howell C, Grady K, Cox C, editors. *Managing Obstetric Emergencies and Trauma – the MOET Course Manual*. 2nd edition. London: RCOG Press; 2007.

53. Kinsella SM. Lateral tilt for pregnant women: why 15 degrees? *Anaesthesia* 2003;58:835–6.

54. Rees GA, Willis BA. Resuscitation in late pregnancy. *Anaesthesia* 1988;43:347–9.

55. Goodwin AP, Pearce AJ. The human wedge. A manoeuvre to relieve aortocaval compression during resuscitation in late pregnancy. *Anesthesia* 1992;47:433–4.

56. Difficult Airway Society. Recommended Equipment for Management of Unanticipated Difficult Intubation. Difficult Airway Society; 2005 [www.das.uk.com/files/dasequipmentjuly2005.doc].

57. Henderson JJ, Popat MT, Latto IP, Pearce AC. Difficult Airway Society guidelines for management of the unanticipated difficult intubation. *Anaesthesia* 2004;59:675–94.

58. Goodwin H, Holmes JF, Wisner DH. Abdominal ultrasound examination in pregnant blunt trauma patients. *J Trauma* 2001;50:689–93.

59. Lazebnik N, Lazebnik RS. The role of ultrasound in pregnancy-related emergencies. *Radiol Clin North Am* 2004;42:315–27.

60. Nanson J, Elcock D, Williams M, Deakin CD. Do physiological changes in pregnancy change defibrillation energy requirements? *Br J Anaesth* 2001;87:327–9.

61. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. *Obstet Gynecol* 1986;68:571–6.

62. Bozcar ME, Howard MA, Rivers EP, Martin GB, Horst HM, Lewandowski C, et al. A technique revisited: hemodynamic comparison of closed- and open-chest cardiac massage during human cardiopulmonary resuscitation. *Crit Care Med* 1995;23:498–503.

63. Royal College of Pathologists. *Guidelines on Autopsy Practice. Report of a working group of the Royal College of Pathologists*. London: Royal College of Pathologists; 2002 [http://www.rcpath.org.uk/resources/pdf/main_document.pdf].

64. Royal College of Pathologists. *Guidelines on Autopsy Practice. Scenario 5: Maternal death*. London: Royal College of Pathologists; 2005 [http://www.rcpath.org/resources/pdf/AutopsyScenario5Aug05.pdf].

65. Association of Anaesthetists of Great Britain and Ireland. *AAGBI Safety Guideline. Interhospital Transfer*. London: AAGBI; 2009. [http://www.aagbi.org/publications/guidelines.htm#i]

66. Royal College of Obstetricians and Gynaecologists. Green-top Guideline No. 52: *Prevention and Management of Postpartum Haemorrhage*. London: RCOG; 2009 [http://www.rcog.org.uk/files/rcog-corp/Green-top52PostpartumHaemorrhage.pdf].

67. Royal College of Obstetricians and Gynaecologists. Green-top Guideline No. 27: *Placenta Praevia, Placenta Praevia Accreta and Vasa Praevia: Diagnosis and Management*. London: RCOG; 2010.

68. Royal College of Obstetricians and Gynaecologists. Green-top Guideline No. 37b: *The acute management of thrombosis and embolism during pregnancy and the puerperium*. London: RCOG; 2007.

69. Steer PJ, Gatzoulis MA, Baker P. *Heart Disease and Pregnancy*. London: RCOG Press; 2006.

70. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al.; International Surviving Sepsis Campaign Guidelines Committee; American Association of Critical-Care Nurses; American College of Chest Physicians; American College of Emergency Physicians; Canadian Critical Care Society; European Society of Clinical Microbiology and Infectious Diseases; European Society of Intensive Care Medicine; European Respiratory Society; International Sepsis Forum; Japanese Association for Acute Medicine; Japanese Society of Intensive Care Medicine; Society of Critical Care Medicine; Society of Hospital Medicine; Surgical Infection Society; World Federation of Societies of Intensive and Critical Care Medicine. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2008;36:296–327.

71. Levy MM, Dellinger RP, Townsend SR, Lind-Zwirble WT, Marshall JC, Bion J, et al.; Surviving Sepsis Campaign. The Surviving Sepsis Campaign: results of an international guideline-based performance improvement program targeting severe sepsis. *Crit Care Med* 2010;38:364–74.

72. Gao F, Melody T, Daniels R, Giles S, Fox S. The impact of compliance with 6-hour and 24-hour sepsis bundles on hospital mortality in patients with severe sepsis: a prospective observational study. *Crit Care* 2005;9:764–70.

73. National Patient Safety Agency. Safer practice with epidural injections and infusions. Patient safety alert 21. London: National Patient Safety Agency; 2007 [http://www.nrls.npsa.nhs.uk/resources/type/alerts/?entryid45=59807&q=0%c2%acepidural+injections%c2%ac].

74. Royal College of Obstetricians and Gynaecologists. Green-top Guideline No. 10(A): *The Management of Severe Pre-eclampsia/Eclampsia*. London: RCOG; 2006 [http://www.rcog.org.uk/files/rcog-corp/uploaded-files/GT10aManagementPreeclampsia2006.pdf].

75. Harper NJ, Dixon T, Dugué P, Edgar DM, Fay A, Gooi HC, et al; Working Party of the Association of Anaesthetists of Great Britain and Ireland. Suspected anaphylactic reactions associated with anaesthesia. *Anaesthesia* 2009;64:199–211.

76. Katz V, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? *Am J Obstet Gynecol* 2005;192:1916–20.

77. Powner DJ, Bernstein IM. Extended somatic support for pregnant women after brain death. *Crit Care Med* 2003;31:1241–9.

78. Bernstein IM, Watson M, Simmons GM, Catalano PM, Davis G, Collins R. Maternal brain death and prolonged fetal survival. *Obstet Gynecol* 1989;74:434–7.

79. Mallampalli A, Guy E. Cardiac arrest in pregnancy and somatic support after brain death. *Crit Care Med* 2005;33 Suppl 10:S325–31.

80. Penney G, Brace V. Near miss audit in obstetrics. *Curr Opin Obstet Gynecol* 2007;19:145–50.

81. NHS Litigation Authority. Clinical Negligence Scheme for Trusts: Maternity Standards. London: NHSLA; 2009 [http://www.nhsla.com/].

82. Scottish Executive. Health Department. Directorate of Finance. Clinical Negligence and Other Risks (Non-clinical) Indemnity Scheme (CNORIS). Edinburgh: NHS MEL; 1999 [http://www.sehd.scot.nhs.uk/mels/1999_86.htm].

83. Welsh Risk Pool. Standard 15: Maternity services; 2005.

84. Jabbour M, Osmond MH, Klassen TP. Life support courses: are they effective? *Ann Emerg Med* 1996;28:690-8.

85. Draycott T, Sibanda T, Owen L, Akande V, Winter C, Reading S, et al. Does training in obstetric emergencies improve neonatal outcome? *BJOG* 2006;113:177-82.

86. Draycott TJ, Crofts JF, Ash JP, Wilson LV, Yard E, Sibanda T, et al. Improving neonatal outcome through practical shoulder dystocia training. *Obstet Gynecol* 2008;112:14-20.

87. Sibanda T, Crofts JF, Barnfield S, Siassakos D, Epee MJ, Winter C, et al. PROMPT education and development: saving mothers' and babies' lives in resource poor settings. *BJOG* 2009;4:868-9.

88. Siassakos D, Hasafa Z, Sibanda T, Fox R, Donald F, Winter C, et al. Retrospective cohort study of diagnosis-delivery interval with umbilical cord prolapse: the effect of team training. *BJOG* 2009;116:1089-96.

89. Black RS, Brocklehurst P. A systematic review of training in acute obstetric emergencies. *BJOG* 2003;110:837-41.

90. Baskett P. Progress of the advanced life support courses in Europe and beyond. *Resuscitation* 2004;62:311-3.

91. Johanson R, Cox C, O'Donnell E, Grady K, Howell C, Jones P. Managing obstetric emergencies and trauma (MOET). Structured skills training using models and reality-based scenarios. *TOG* 1999;1:46-52.

92. Taylor HA, Kiser WR. Reported comfort with obstetrical emergencies before and after participation in the advanced life support in obstetrics course. *Fam Med* 1998;30:103-7.

93. Ellis D, Crofts JF, Hunt LP, Read M, Fox R, James M. Hospital, simulation center, and teamwork training for eclampsia management: a randomized controlled trial. *Obstet Gynecol* 2008;111:723-31.

94. Siassakos D, Crofts J, Winter C, Weiner CP, Draycott TJ. The active components of effective training in obstetric emergencies. *BJOG* 2009;116:1028-32.

95. Crofts JF, Bartlett C, Ellis D, Hunt LP, Fox R, Draycott TJ. Management of shoulder dystocia: skill retention 6 and 12 months after training. *Obstet Gynecol* 2007;110:1069-74.

96. Crofts JF, Ellis D, Draycott TJ, Winter C, Hunt LP, Akande VA. Change in knowledge of midwives and obstetricians following obstetric emergency training: a randomised controlled trial of local hospital, simulation centre and teamwork training. *BJOG* 2007;114:1534-41.

97. Dijkman A, Huisman CM, Smit M, Schutte JM, Zwart JJ, Van Roosemalen, et al. Cardiac arrest in pregnancy: increasing use of perimortem caesarean section due to emergency skills training? *BJOG* 2010;117:282-7.

98. Weston R. When birth goes wrong. *Pract Midwife* 2001;4:10-2.

APPENDICES

APPENDIX 1 Post-collapse management algorithm

```
                        ┌─────────────────────┐
                        │    Patient alive?    │
                        └─────────────────────┘
                          /               \
                         /                 \
              ┌───────────────┐     ┌───────────────┐
              │      Yes       │     │      No        │
              └───────────────┘     └───────────────┘
                     │                      │
                     ▼                      ▼
          ┌─────────────────────┐  ┌─────────────────────┐
          │  Continue treatment  │  │   Inform coroner     │
          │                      │  │                      │
          │ Transfer to critical │  │    Inform CMACE      │
          │    care setting      │  │                      │
          └─────────────────────┘  └─────────────────────┘
                      \                    /
                       \                  /
                  ┌─────────────────────────────┐
                  │ Ensure accurate documentation │
                  └─────────────────────────────┘
                                │
                                ▼
                  ┌─────────────────────────────────┐
                  │ Debrief patient (if alive) and    │
                  │            relatives              │
                  └─────────────────────────────────┘
                                │
                                ▼
                  ┌─────────────────────────────────┐
                  │ Offer staff support and debriefing │
                  └─────────────────────────────────┘
                                │
                                ▼
                  ┌─────────────────────────────────┐
                  │        Review case through         │
                  │    clinical governance process     │
                  └─────────────────────────────────┘
```

APPENDIX 2   Maternal collapse algorithm

Unresponsive?

— No → Put into left lateral position / Call for help if appropriate / Check maternal obs / Assess fetal wellbeing / Call for obstetric review

— Yes ↓

Open airway / Look for signs of life

Wedge/tilt patient ← → Call obstetric resuscitation team ← Call consultant obstetrician and anaesthetist → Call neonatal team if >22 weeks of gestation

↓

CPR 30:2 / Until defibrillator/monitor attached → 100% supplemental O2 / Intubate early / Insert two IV cannulae (wide-bore)

← If no response to CPR after 4 minutes, proceed to delivery/perimortem caesarean section

↓

Assess rhythm

— **Shockable (VF/pulseless VT)** → 1 shock 150–360 J biphasic or 360 J monophasic → Immediately resume **CPR for 2 minutes** minimise interruptions

— Return of spontaneous circulation → **Immediate postcardiac arrest treatment**
- Use ABCDE approach
- Controlled oxygenation and ventilation
- 12-lead ECG
- Treat precipitating cause
- Temperature control/therapeutic hypothermia

— **Non-shockable (PEA/asystole)** → Immediately resume **CPR for 2 minutes** minimise interruptions

During CPR:
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseus)
- Give adrenaline every 3–5 minutes
- Correct reversible causes

Reversible causes:
- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis – coronary or pulmonary
- Tamponade – cardiac
- Toxins
- Tension pneumothorax

KEY
ABCDE = airway, breathing, circulation, disability, exposure; CPR = cardiopulmonary resuscitation;
RCG = electrocardiogram; PEA = pulseless electrical activity; VF = ventricular fibrillation; VT = ventricular tachycardia

Clinical guidelines are: 'systematically developed statements which assist clinicians and patients in making decisions about appropriate treatment for specific conditions'. Each guideline is systematically developed using a standardised methodology. Exact details of this process can be found in Clinical Governance Advice No.1: *Development of RCOG Green-top Guidelines* (available on the RCOG website at **http://www.rcog.org.uk/womens-health/clinical-guidance/development-rcog-green-top-guidelines-policies-and-processes**). These recommendations are not intended to dictate an exclusive course of management or treatment. They must be evaluated with reference to individual patient needs, resources and limitations unique to the institution and variations in local populations. It is hoped that this process of local ownership will help to incorporate these guidelines into routine practice. Attention is drawn to areas of clinical uncertainty where further research may be indicated.

The evidence used in this guideline was graded using the scheme below and the recommendations formulated in a similar fashion with a standardised grading scheme.

Classification of evidence levels	Grades of recommendations
1++ High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias	**A** At least one meta-analysis, systematic review or randomised controlled trial rated as 1++ and directly applicable to the target population; or A systematic review of randomised controlled trials or a body of evidence consisting principally of studies rated as 1+ directly applicable to the target population and demonstrating overall consistency of results
1+ Well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias	
1− Meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a high risk of bias	**B** A body of evidence including studies rated as 2++ directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
2++ High-quality systematic reviews of case–control or cohort studies or high-quality case–control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal	
2+ Well-conducted case–control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal	**C** A body of evidence including studies rated as 2+ directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
2− Case–control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal	**D** Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+
3 Non-analytical studies, e.g. case reports, case series	**Good practice point**
4 Expert opinion	✓ Recommended best practice based on the clinical experience of the guideline development group

The guidelines review process will commence in 2014 unless evidence requires earlier review.