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Appendix 1: Explanation of guidelines and evidence levels

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/green-top-development). 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

1 + +	High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias
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
2 + +	High-quality systematic reviews of case–control or cohort studies or high-qualitycase–control or cohort studies with a very low risk of confounding, bias or chance and ahigh probability that the relationship is causal
2+	Well-conducted case–control or cohort studies with a low risk of confounding, bias or chanceand a moderate probability that the relationship is causal
2–	Case–control or cohort studies with a high risk of confounding, bias or chance and asignificant risk that the relationship is not causal
3	Non-analytical studies, e.g. case reports, case series
4	Expert opinion

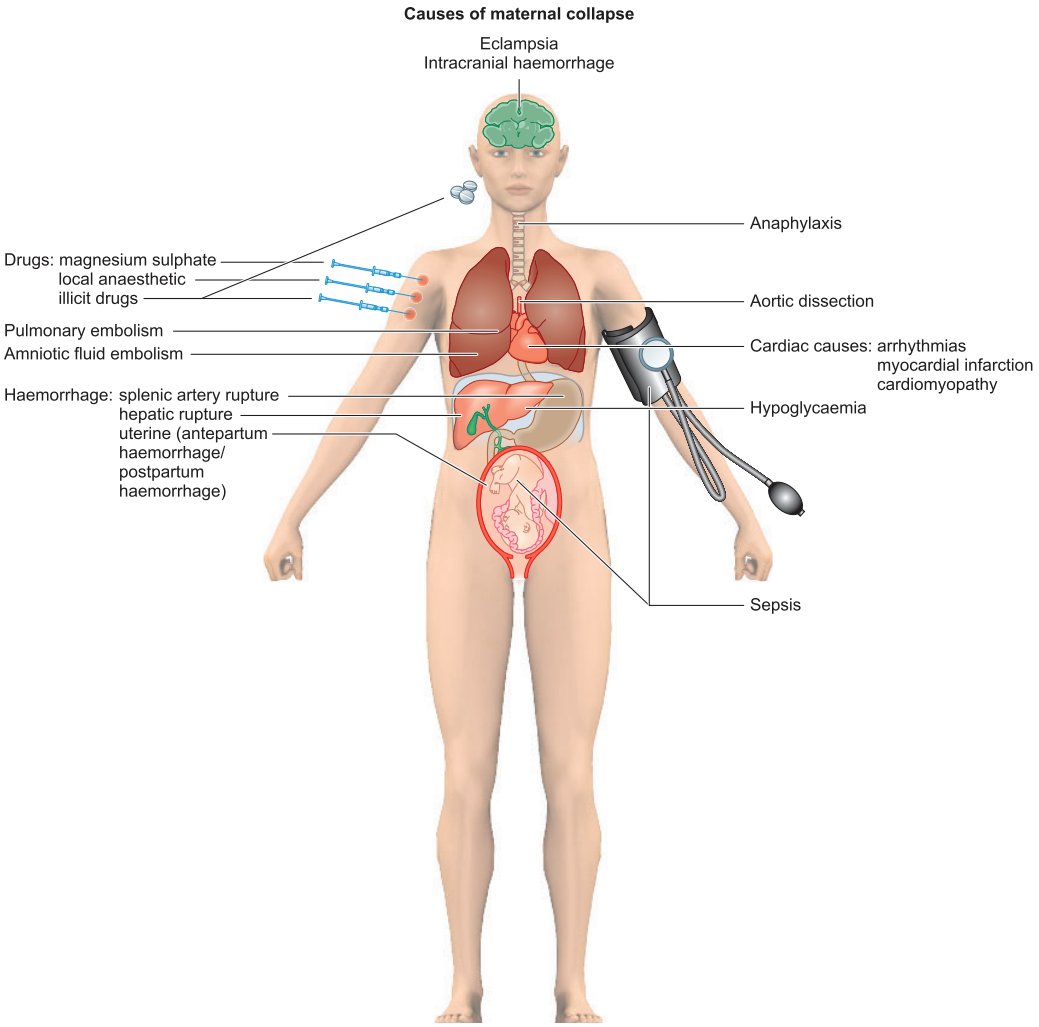
Grades of Recommendation

A	At least one meta-analysis, systematic reviews or RCT rated as 1 + +, and directly applicable to the target population; or a systematic review of RCTs or a body of evidence consisting principally of studies rated as 1 + , directly applicable to the target population and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2++ directly applicable to the target population, and demonstrating overall consistency of results; orExtrapolated evidence from studies rated as 1++ or 1 +
C	A body of evidence including studies rated as 2+ directly applicable to the target population, and demonstrating overall consistency of results; orExtrapolated evidence from studies rated as 2 + +
D	Evidence level 3 or 4; orExtrapolated evidence from studies rated as 2+

Good Practice Points

✓	Recommended best practice based on the clinical experience of the guideline development group

Appendix 2: Causes of maternal collapse



Reversible cause		Cause in pregnancy
4H's	Hypovolaemia	Bleeding (obstetric/other; may be concealed) or relative hypovolaemia of dense spinal block, septic or neurogenic block
	Hypoxia	Pregnant women can become hypoxic more quickly. Cardiac events – peripartum cardiomyopathy, myocardial infarction, aortic dissection, large vessel aneurysms
	Hypo/hyperkalaemia and Hyponatraemia	Hypo and hyperkalaemia are no more likely. Hyponatraemia may be caused by oxytocin use
	Hypothermia	No more likely
4T's	Thromboembolism	Amniotic fluid embolus, pulmonary embolus, air embolus, myocardial infarction
	Toxicity	Local anaesthetic, magnesium, other
	Tension pneumothorax	Following trauma/suicide attempts
	Tamponade	Following trauma/suicide attempts
Eclampsia and pre-eclampsia		Includes intracranial haemorrhage

Appendix 3: Physiological and physical changes in pregnancy

System	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%	Hypoxia develops more quickly when apnoeic
Arterial pCO_2	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 venous cava.

