

Review Article

Management of pre-eclampsia: issues for anaesthetists

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Summary

Pre-eclampsia is a leading cause of maternal morbidity and mortality. Substandard care is often present and many deaths are preventable. The aim of this review is to summarise the key management issues for anaesthetists in the light of the current literature. A systematic literature search of electronic databases was undertaken including MEDLINE, EMBASE and the Cochrane Library using the key words obstetrics, pregnancy, pregnancy complications, maternal, pre-eclampsia, preeclampsia, cardiac function, haemodynamics, haemolysis, elevated liver enzymes, low platelets (HELLP), eclampsia, anaesthesia, anesthesia, neuraxial. Relevant Colleges and Societies websites were examined for pertinent guidelines. The disease is defined within the context of hypertensive diseases, and early recognition of pre-eclampsia and its complications, as well as multidisciplinary expert team management is highlighted. Accurate monitoring and recording of observations including the use of transthoracic echocardiography is discussed. The importance of the treatment of systolic blood pressure > 180 mmHg and the use of intravenous antihypertensive medication as well as the use of parenteral magnesium sulphate for the treatment and prevention of eclampsia is emphasised. Restricted intravenous fluid therapy and avoidance of ergometrine is discussed. Neuraxial analgesia and anaesthesia, and general anaesthesia for birth is summarised as well as postpartum management including analgesia, thromboprophylaxis, management of acute pulmonary oedema and the use of pharmacological agents in the setting of breastfeeding.

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Pre-eclampsia is a multisystem disease unique to human pregnancy characterised by hypertension and organ system involvement. The disease is responsible for considerable morbidity and mortality, complicating 5–8% of pregnancies. Deaths are due to intracranial haemorrhage and cerebral infarction, acute pulmonary oedema, respiratory failure and hepatic failure or rupture [1, 2]. Severe maternal complications include antepartum haemorrhage due to placental abruption, eclampsia, cerebrovascular accidents, organ failure and disseminated intravascular coagulation [1–4]. Pre-eclampsia is the leading cause of fetal growth

restriction, intrauterine fetal demise and preterm birth [2, 5].

Women who experience pre-eclampsia are at increased risk of hypertension, cerebrovascular disease and ischaemic heart disease, in later life [6–11]. Anaesthetists are frequently involved in the multi-disciplinary management of critically ill women with pre-eclampsia, and clinical practice should be based on current scientific evidence.

This article was developed as there was a need to provide a current succinct summary of the anaesthetic issues relating to the care of women with pre-eclampsia

relevant for anaesthetists. It was originally produced as part of a PhD investigating cardiac function in women with pre-eclampsia at the University of Melbourne. In 2008, the first version of this article was produced as part of the Obstetric Anaesthesia: Scientific Evidence project within the Australian Society of Anaesthetists, the New Zealand Society of Anaesthetists and the Australian and New Zealand College of Anaesthetists, and appeared on the latter's website. This review covers the management of women with pre-eclampsia, including eclampsia and the syndrome of haemolysis with elevated liver enzymes and low platelets (HELLP) in the peripartum period, which is specifically relevant to anaesthetists. It does not cover prevention, screening, risk factors, pathophysiology, and prognosis or long-term management of the disease.

Methods

Electronic search strategies included searching the databases Ovid MEDLINE, Ovid EMBASE (both until December 2011) and the Cochrane Library using the key words obstetrics, pregnancy, pregnancy complications, maternal pre-eclampsia, pre-eclampsia, cardiac function, haemodynamics, haemolysis, elevated liver enzymes, low platelets (HELLP), eclampsia, anaesthesia, and anaesthesia, and neuraxial. Literature in languages other than English was included in the searches. Relevant college and society web-based resources were examined including the Royal College of Obstetricians and Gynaecologists, the National Institute for Health and Clinical Excellence college and society (NICE), the American College of Obstetricians and Gynecologists, the Canadian Society of Obstetricians and Gynecologists, the Society of Obstetric Medicine of Australia and New Zealand, the International Society for the Study of Hypertension in Pregnancy, the International Society of Obstetric Medicine, the World Health Organization, the International Federation of Gynecologists and Obstetricians, the Obstetric Anaesthetists' Association, the Society of Obstetric Anesthesiologists and Perinatologists, the American Heart Association and the European Society of Cardiology, to search for any relevant publications, position statements or pre-existing guidelines. Where appropriate, levels of evidence are given in the article according to the NICE 2005 levels of evidence for intervention studies scale (Table 1).

Table 1 National Institute for Health and Clinical Excellence 2005 levels of evidence for intervention studies.

Level	Intervention
1++	High-quality meta-analyses, systematic reviews of RCTs or RCTs with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews of RCTs or RCTs with a low risk of bias
1-	Meta-analyses, systematic reviews of RCTs or RCTs with a high risk of bias
2++	High-quality systematic reviews of case-control or cohort studies; 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 casual
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 casual
2-	Case-control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not casual
3	Non-analytical studies (e.g. case reports, case series)
4	Expert opinion, formal consensus

RCT, randomised controlled trial.

Current guidelines for the overall management of women with hypertension in pregnancy have been developed by Royal College of Obstetricians and Gynaecologists United Kingdom, NICE, the American College of Obstetricians and Gynecologists, the Canadian Society of Obstetricians and Gynecologists, the Society of Obstetric Medicine of Australia and New Zealand, the International Society for the Study of Hypertension in Pregnancy, and the European Society of Cardiology.

Defining the disease

Principles

Pre-eclampsia is classified within the broad category of hypertensive diseases of pregnancy. Pre-existing or chronic hypertension is present before and often during pregnancy, and gestational hypertension is defined as hypertension arising after 20 weeks' gestation, without any other organ system involvement. Pre-eclampsia is characterised by elevated blood pressure and the involvement of one or more organ systems. In addition, the definition includes a requirement for the condition to resolve in the postpartum period i.e. it is specifically a complication of pregnancy. From a management perspective, clinicians should always be alert to the possibility of alternative diagnoses. The importance of

defining acceptable levels of blood pressure readings, proteinuria, biochemical limits and seizures is to allow therapy to be guided and a decision to be made regarding the timing of the birth. Delivery of the baby and removing the placenta is currently the only definitive way of curing the condition. Terms that have been used in the past such as pregnancy induced hypertension (PIH) and pre-eclamptic toxemia (PET) must now be considered to be outdated.

Definition

Pre-eclampsia has many different definitions [1–5, 12–20]. Despite some differences in the detail there are features common to all definitions. Pre-eclampsia is defined as hypertension arising after 20 weeks' gestation, with one or more organ system involvement (Table 2). There is resolution of the disease by three months postpartum.

Blood pressure should be measured at rest, using Korotkoff V as the diastolic value, with an appropriately sized blood pressure cuff. Initial blood pressure should be measured with a calibrated manual auscultatory device by a trained operator, as automatic systems often underestimate systolic blood pressure [1, 21]. Repeated measurements of blood pressure confirming sustained hypertension should be made and these are usually done at four-hourly intervals.

Significant proteinuria is defined as > 0.3 g protein excreted in 24 h. The spot urinary protein:creatinine ratio is frequently measured and is based on the principle that the daily excretion rates of protein and creatinine are similar. Normal values are defined as ≤ 0.03 g.mmol⁻¹ [22–24].

Table 2 Definition of pre-eclampsia.

Hypertension
Systolic blood pressure
≥ 140 mmHg
And/or
Diastolic blood pressure
≥ 90 mmHg
And one or more abnormality of the:
Central nervous system
Cardiorespiratory system
Gastrointestinal system
Haematological system
Renal system
Uteroplacental/fetal circulation

Serum urate levels are often elevated in pre-eclampsia. Hyperuricemia is associated with perinatal complications, and although elevated levels have not predicted adverse maternal outcomes, urate is frequently measured in clinical practice [19, 25].

Severe pre-eclampsia can occur in the preterm, term and postpartum periods. It is a term applied to a condition with marked elevation of blood pressure (systolic ≥ 160 mmHg, diastolic ≥ 110 mmHg) and extreme derangements of organ function. These may include central nervous system problems including headache, seizures (eclampsia), impaired conscious state and visual disturbances, renal dysfunction (urinary protein ≥ 5 g protein excreted in 24 h), haematological complications and uteroplacental compromise. Women may be symptomatic. HELLP syndrome is considered a variant of severe pre-eclampsia. Haemolysis is diagnosed by observation of fragmented red blood cells and schistocytes on a blood film, elevated lactate dehydrogenase levels, elevated total bilirubin often in association with a decreasing haematocrit or the presence of a bleeding diathesis.

Pre-eclampsia with hepatic dysfunction, without haemolysis, may occur in severe pre-eclampsia. Other forms of severe hepatic dysfunction in pregnancy need to be differentiated from pre-eclampsia. Acute fatty liver of pregnancy is a rare condition of pregnancy, which is not associated with hypertension [26–28]. Similarly, haemolytic uraemic syndrome (HUS)/thrombotic thrombocytopenic purpura (TTP) should be differentiated from HELLP as treatment interventions differ significantly. HUS/TTP is a clinical diagnosis defined by the presence of a pentad of features; thrombocytopenia; microangiopathic haemolytic anaemia; neurological symptoms and signs; renal function abnormalities; and fever.

Preterm pre-eclampsia is often severe and associated with abnormalities of placentation and intrauterine growth restriction. Pre-eclampsia should be considered in any pregnant women with a severe headache or new onset epigastric pain [1]. Severe disease is summarised in Table 3, based on various sources [3, 12, 15, 17–20].

Diagnostic challenge

Hypertension in pregnancy may be caused by a variety of different pathologies and it is important to consider

Table 3 Characteristics of severe pre-eclampsia.

Severe pre-eclampsia threshold level	
Hypertension	Systolic blood pressure ≥ 140 mmHg ≥ 160 mmHg – severe hypertension ≥ 180 mmHg – hypertensive crisis And/or diastolic blood pressure ≥ 90 mmHg ≥ 110 mmHg – hypertensive crisis
And one or more of:	
Central nervous system	Seizures/eclampsia Headache Visual disturbances Papilloedema Clonus/hyperreflexia
Cardiorespiratory system	Pulmonary oedema
Gastrointestinal system	Elevated liver transaminase enzymes ≥ 70 IU.l ⁻¹ Liver tenderness Nausea and vomiting Epigastric pain
Haematological system	Haemolysis Thrombocytopenia $< 100 \times 10^9$.l ⁻¹ Disseminated intravascular coagulation
Renal system	Proteinuria > 5 g excreted in 24 h, 3 + protein dipstick Protein/creatinine > 0.5 g.mmol ⁻¹ Urine output < 500 ml in 24 h Renal failure
Uteroplacental/fetal circulation	Placental abruption Intrauterine growth restriction Umbilical artery/uterine artery blood flow abnormalities – absent or reversed end-diastolic flow

other aetiologies. These include renal disease, pheochromocytoma, drug usage such as cocaine and amphetamines and cardiovascular diseases such as coarctation, subclavian stenosis, aortic dissection and vasculitis.

Under extremely rare circumstances, pre-eclampsia may develop before 20 weeks' gestation in the setting of a hydatidiform mole, multiple pregnancy, fetal or placental abnormalities, antiphospholipid syndrome or severe renal disease [18, 29].

Organisational aspects of care

Multidisciplinary team approach

The significance of an experienced multidisciplinary team approach to the management of women with pre-eclampsia has been highlighted in many recent publications [1, 2, 4, 30, 31]. A common message is the importance of early

referral, involving the anaesthetist in the management [4, 31, 32] and ensuring that the woman with severe pre-eclampsia is stabilised before delivery [2, 4, 19].

Maintenance of clinical skills

In the most recent Confidential Enquiries into Maternal Deaths, the maintenance of clinical skills was highlighted as a major factor in reducing morbidity and mortality [1]. Particular emphasis was placed on regular audited training of all clinical staff for the early recognition and management of severely ill pregnant women [1, 2].

The use of an obstetric early warning chart has been proposed as an important clinical tool that may allow for more timely recognition of those women who are developing a critical illness [1, 2]. Widespread adoption of generic reportable parameters also allows benchmarking both nationally and internationally for outcomes in women with pre-eclampsia [33, 34].

Reduction of high blood pressure

Non-severe hypertension

Non-severe hypertension is defined as blood pressure with a systolic level of 140–159 mmHg and a diastolic of 90–109 mmHg. In a recent systematic review, there were no clear differences when antihypertensive interventions were compared with placebo or with no intervention, or when two anti-hypertensive medications were compared. Due to the risk of haemorrhagic stroke in the presence of systolic hypertension, most guidelines recommend lowering of non-severe blood pressure to a systolic level of 140–150 mmHg and a diastolic of 90–100 mmHg, using oral labetalol as the drug of choice [20, 35]. Thresholds vary depending on the existence of co-morbidities.

Safe agents include methyldopa, labetalol, nifedipine or isradipine, and some β -adrenoceptor blockers (metoprolol, pindolol, propranolol) and low-dose diazoxide [36]. Atenolol is not recommended due to fetal growth restriction. Angiotensin converting enzyme inhibitors and angiotensin type-2 receptor blockers are contraindicated (19, 37, 38; Level 3).

Severe hypertension

Severe hypertension is defined as systolic pressure ≥ 160 mmHg or diastolic pressure ≥ 110 mmHg and

should be treated (Level 2–). Systolic blood pressure > 180 mmHg is defined as a hypertensive crisis and a medical emergency and requires immediate and effective treatment [1]. Based on maternal mortality reports, this degree of hypertension, if left untreated, is associated with an increased risk of intracerebral haemorrhage [2]. Reducing severe levels of hypertension decreases the risk of mortality [38] (Level 3).

Drugs that can be safely used include labetalol (oral or intravenous), nifedipine (oral) and hydralazine (intravenous). The choice should be made depending on the experience of the clinician with a particular agent [39] (Level 1++). There is an absence of robust trials comparing hydralazine with intravenous labetalol or oral nifedipine [20]. The latter agents may be preferable due to reduced maternal and fetal complications [3, 40] (Level 1++). Labetalol should be avoided in women with severe asthma. Drugs that should be avoided for the reduction of blood pressure are high dose diazoxide, ketanserin, nimodipine and magnesium sulphate (MgSO_4) [39]. Continuous fetal heart rate monitoring should be employed until the blood pressure is stable [19].

Particular care should be taken to avoid precipitous falls in blood pressure, which may induce maternal or fetal complications, as a result of falling below critical perfusion thresholds. Elevated blood pressure should be lowered to levels of systolic 140–150 mmHg/diastolic 80–100 mmHg at a rate of 10–20 mmHg every 10–20 min. Consideration should also be given to the extent of placental transfer of the administered drug.

There is extensive experience with the safety and efficacy of intravenous hydralazine. This is usually administered by intermittent bolus of 5 mg intravenously or intramuscularly. A continuous infusion of 0.5–10.0 $\text{mg}\cdot\text{h}^{-1}$ is also typically employed in more refractory cases. The use of hydralazine is often accompanied by maternal tachycardia and cautious administration of up to 500 ml crystalloid is recommended before or at the same time as the initial dose of intravenous hydralazine to reduce the chance of a precipitous fall in blood pressure [20].

Sodium nitroprusside is rarely used in pregnancy and has known maternal adverse effects of hypotension and paradoxical bradycardia in women with severe pre-eclampsia. Fetal cyanide toxicity is a complication of

prolonged treatment. Sodium nitroprusside should be used with extreme caution in situations of life-threatening hypertension, immediately before delivery in circumstances where clinicians are familiar with its use [3, 38]. It is administered as an intravenous infusion at 0.25–5.0 $\mu\text{g}\cdot\text{kg}\cdot\text{min}^{-1}$ [3].

The pharmacological agent of choice in women with pre-eclampsia and acute pulmonary oedema is glyceryl trinitrate. This is administered as an infusion of 5 $\mu\text{g}\cdot\text{min}^{-1}$, increasing every 3–5 min to a maximum dose of 100 $\mu\text{g}\cdot\text{min}^{-1}$ [3].

Treatment and prevention of seizures (eclampsia)

Eclampsia signifies severe disease and is associated with intracranial haemorrhage, cardiac arrest and death with a case fatality rate of 3.1% [1, 4, 41].

Treatment of seizures and prevention of recurrent seizures

Magnesium sulphate is the first-line drug treatment for seizures (eclampsia) and for recurrent seizures [42, 46] (Level 1++). Magnesium sulphate reduces the chance of maternal death compared with diazepam [46] (Level 1++) and is superior to diazepam, phenytoin and lytic cocktail (chlorpromazine, promethazine, pethidine) in reducing significantly the risk of seizure recurrence [42, 45, 46] (Level 1++). Morbidity related to pneumonia, mechanical ventilation and admission to an intensive care unit are significantly reduced with the use of MgSO_4 compared with phenytoin [45] (Level 1++). Both intravenous and intramuscular routes of administration have been used effectively. The regimen recommended by the Collaborative Eclampsia Trial is 4–5 g MgSO_4 intravenously over 5 min, followed by an infusion of 1 $\text{g}\cdot\text{h}^{-1}$ for 24 h. If recurrent seizures occur, an additional 2 g intravenous MgSO_4 should be administered [44].

Prevention of seizures

Magnesium sulphate is recommended as prophylaxis for eclampsia in women with severe pre-eclampsia [43] (Level 1++). Compared with placebo or no treatment, the use of MgSO_4 more than halves the risk of eclampsia and the number needed to treat to prevent one seizure in this group of women was 50. Magnesium was also

advantageous in reducing the risk of a first seizure when compared with other agents.

There is controversy regarding the use of MgSO_4 in mild disease. The number needed to treat to prevent one woman having a seizure was approximately 100, and side effects were more common in the MgSO_4 group, although none were life-threatening [43]. There was also an increase in the number of deliveries by caesarean section in the MgSO_4 group. Magnesium should be considered for seizure prophylaxis in women with mild pre-eclampsia (Level 1++).

When MgSO_4 was selectively administered only to women with severe pre-eclampsia instead of to all women with gestational hypertension, there were more women with eclampsia who then required general anaesthesia and experienced adverse neonatal outcomes compared with their controls [47] (Level 2–).

Clinical practice issues related to MgSO_4

Magnesium sulphate does not reverse or prevent the progression of the disease, nor does it significantly lower blood pressure and it is not recommended as an antihypertensive agent [19, 38]. Patient safety and clinical effectiveness are enhanced when hospitals, health centres, and emergency transport vehicles have guidelines for the safe use of MgSO_4 . Monitoring of MgSO_4 should utilise clinical parameters of urinary output, respiratory rate, oxygen saturation and patellar reflexes. Serum magnesium levels should be measured if toxicity is suspected. This is often apparent with serum levels of $> 3.5 \text{ mmol.l}^{-1}$. Toxicity is particularly likely in the presence of significant renal insufficiency. The drug treatment for MgSO_4 toxicity is 10% calcium gluconate (1 g) over 10 min.

Additional therapies for haematological and/or hepatic complications

Corticosteroids for HELLP

The syndrome of haemolysis, elevated liver enzymes, and low platelets is a severe form of pre-eclampsia. The exact criteria that are used to make the diagnosis are debated in the literature. The most widely used classifications are those of Sibai and Martin [16, 48]. Common to both are evidence of haemolysis as evidenced with raised lactate dehydrogenase, elevated liver transaminases, plus a platelet count of $< 100 \times 10^9 \text{.l}^{-1}$.

A systematic review concluded that there was insufficient evidence either to refute or to support adjuvant corticosteroid use with either dexamethasone (10–12 mg), betamethasone (12 mg) or prednisolone [49]. Consistent with observational studies, corticosteroids were shown to increase the platelet count if given in the antenatal period, and the authors concluded that corticosteroids may be justified in clinical situations where an increased rate of recovery in platelet count is clinically useful [49]. This did not translate to improvement in outcomes and the clinical relevance of this is unclear. On the basis of the available evidence, it is also not clear whether administration of corticosteroids to increase platelet count to generate a number at which one could safely undertake neuraxial anaesthesia is beneficial or harmful [50].

Postpartum use of dexamethasone was compared with placebo in a randomised controlled trial that found no difference in key maternal morbidity and mortality indices, and no difference in the use of blood products between the two groups. This finding did not support the use of dexamethasone in the postpartum period [51].

Platelet transfusions and other therapies

A significant decrease in platelet numbers may be associated with abnormal bleeding. In the non-obstetric population, a level of $< 50 \times 10^9 \text{.l}^{-1}$ is considered significant in the context of surgery or major haemorrhage. It is recommended that platelet counts should not be allowed to decrease below $50 \times 10^9 \text{.l}^{-1}$ in the acutely bleeding pregnant woman [52].

Other interventions aimed at limiting the disease such as plasma exchange or plasmapheresis have been described, but require additional investigation before any recommendation may be made [19].

Intravenous fluids

Acute pulmonary oedema is a leading cause of death in women with pre-eclampsia and is a frequent cause for admission to intensive care [53–55]. In observational studies, the use of either crystalloid or colloid solutions has been associated with transient improvements in maternal cardiovascular system parameters. However, in one large trial and a systematic review, volume expansion demonstrated no advantage when compared with no plasma volume expansion [53, 55]. In a recent

retrospective review comparing two tertiary referral obstetric units, one with a restrictive fluid policy and one without, the rate of acute pulmonary oedema was strongly associated with increased intravenous fluid administration in the unit that had unrestricted fluid policies for women undergoing labour, caesarean delivery and seizure prophylaxis with MgSO_4 [34] (Level 3). Acute pulmonary oedema is associated with positive fluid balances of > 5500 ml [56, 57] (Level 3). In addition, evidence suggests that the use of intravenous fluids to increase plasma volume or treat oliguria in a woman with normal renal function and stable serum creatinine levels is not recommended [53].

Specific clinical contexts

Neuraxial blockade in women with pre-eclampsia

The incidence of major complications after neuraxial techniques in pregnant women in general is approximately 1/20 000–30 000 for spinal anaesthesia, and 1/25 000 for epidural analgesia [58]. In the absence of contraindications, lumbar neuraxial analgesia is appropriate for women with pre-eclampsia during labour [20, 59, 60] (Level 1+) and neuraxial anaesthesia is the preferred method for anaesthesia for caesarean birth in women with pre-eclampsia [61] (Level 1+).

Coagulopathy in pre-eclampsia is usually due to thrombocytopenia and, less commonly, disseminated intravascular coagulation. Studies investigating coagulation in women with pre-eclampsia using thromboelastography found that if the platelet count was greater than $100 \times 10^9 \text{ l}^{-1}$, there were no abnormalities of coagulation detectable [62]. Platelet counts of $< 100 \times 10^9 \text{ l}^{-1}$ in women with severe pre-eclampsia were associated with hypocoagulation and should prompt additional investigation of coagulation status.

Based on current evidence, it is not possible to be definitive regarding a lower limit of the platelet count. Current standards of practice have been drawn indirectly from a number of sources using a variety of outcome measures including the lower limit of thromboelastography maximal amplitude in healthy pregnant women and findings in women with severe pre-eclampsia with platelet counts $< 100 \times 10^9 \text{ l}^{-1}$. A common conclusion of this work has been that a platelet count $> 75 \times 10^9 \text{ l}^{-1}$, in the absence of other coagulation

abnormalities, would not be expected to be associated with increased likelihood of neuraxial anaesthetic complications in the setting of pre-eclampsia [62–64]. Any difference between different techniques and their relative safety have not been adequately studied. In the absence of other coagulation abnormalities, the risk of haematoma associated with neuraxial anaesthesia with platelet counts $> 75 \times 10^9 \text{ l}^{-1}$ is very low.

Analgesia for labour

Lumbar neuraxial analgesia reduces pain-mediated hypertensive responses, and the presence of a functioning epidural catheter enables the use of the epidural catheter for titrating local anaesthetic to ensure surgical anaesthesia [60, 65]. If neuraxial analgesia is contraindicated, intravenous opioid analgesia has been administered with good effect [59, 66]. Intravenous fluid loading should not be used in women who have severe pre-eclampsia before establishing low-dose analgesia including combined-spinal epidural analgesia [20].

Anaesthesia for caesarean birth

Neuraxial anaesthesia is the preferred anaesthetic technique for delivery by caesarean section (Level 1+).

Single-shot spinal, combined-spinal epidural, and epidural anaesthesia have all been used effectively. There is no evidence that one technique has an advantage over the other.

Hypotension requiring vasopressor medication during neuraxial anaesthesia is less common in women with pre-eclampsia than in healthy women. If hypotension occurs, it may be successfully managed with titrated doses of intravenous ephedrine (3–5 mg bolus) or phenylephrine (50–100 μg bolus) [61, 67] (Level 1+).

The use of adrenaline-containing local anaesthetic solutions for epidural boluses to provide surgical anaesthesia appears to be safe, and is widely used to minimise systemic absorption of local anaesthetics. There has been a single case report of a hypertensive crisis with absorbed adrenaline, emphasising the need for close observation of these women [68].

General anaesthesia in women with pre-eclampsia

General anaesthesia may be necessary in a small number of cases for a variety of reasons including coagulopathy, pulmonary oedema or eclampsia. Pulmonary oedema

with underlying systolic dysfunction may indicate peripartum cardiomyopathy. There is minimal evidence to guide practice in the choice of anaesthesia for women in the post-eclamptic period. Some groups have advocated general anaesthesia if symptoms and signs of cerebral oedema are present, or in women with depressed levels of consciousness before caesarean section. However, if the woman is stable with a normal level of consciousness and no neurological deficits, in the absence of other contraindications, neuraxial anaesthesia is an acceptable choice [30].

If general anaesthesia is used, particular attention and extreme vigilance should be given to ablating the hypertensive response to intubation, as this has been identified as a cause of direct maternal mortality [1, 2]. Drugs that have been used for this purpose include alfentanil, fentanyl, remifentanyl, MgSO₄ lidocaine and esmolol. It has been recommended that clinicians use the drug(s) with which they are most familiar [69, 70] (Level 1–). Care must also be taken to avoid complications on emergence from anaesthesia including hypertension, aspiration and acute pulmonary oedema.

Monitoring of the woman with severe pre-eclampsia

During labour, blood pressure measurement should be hourly in women with mild or moderate hypertension, or continually monitored in women with severe hypertension [20].

Intra-arterial blood pressure measurement enables continuous blood pressure recording and facilitates repeated blood sampling for assessment of respiratory function, electrolytes, acid-base balance and haematological and liver abnormalities [71–73] as well as the monitoring of cardiac output by minimally invasive cardiac output monitors [74, 75]. Central venous catheters are not commonly used and pulmonary artery catheters are rarely used. Transthoracic echocardiography offers advantages in this setting as it provides structural and functional information about cardiac performance, diastolic function, and responses to interventions [3, 76–78]. Common transthoracic echocardiographic findings in women with pre-eclampsia include pericardial effusions, diastolic dysfunction and alterations in systolic function with either increased or decreased systolic function [79–82].

The use of oxytocic agents and management of postpartum haemorrhage

The management of life-threatening postpartum haemorrhage in the setting of severe pre-eclampsia is a complex and challenging situation. Reversible causes of uterine atony need to be addressed and mechanical methods to facilitate uterine contraction should be utilised. Syntocinon is the drug of choice for uterine contraction in the setting of severe hypertension and should be carefully titrated to haemodynamic responses [20]. The use of ergometrine has been associated with hypertensive crises and death in women with pre-eclampsia; therefore, ergometrine should not be used for uterine contraction [1, 2] (Level 3). Misoprostol is associated with elevations in blood pressure, although to a lesser degree than ergometrine.

Postpartum management

Ongoing management of women with severe pre-eclampsia should be by adequately trained staff in the appropriately monitored setting [2, 83].

Analgesia

There are few studies examining different analgesic options for women with pre-eclampsia after caesarean birth. Neuraxial techniques, local anaesthetic techniques, opioids, paracetamol and tramadol have not been examined to any significant degree in this population [84]. Non-steroidal anti-inflammatory agents are frequently used for analgesia after childbirth; however, these agents have well-documented adverse effects and contraindications and there have been specific case reports of hypertensive crises in women with pre-eclampsia [85] (Level 3). Alternative agents should be used (Level 4).

Thromboprophylaxis

Thromboprophylaxis should be considered for all women with pre-eclampsia, with consideration given to timing of agents in relation to neuraxial anaesthesia.

Discontinuation of intravenous MgSO₄

Based on the findings of the Collaborative Eclampsia Trial, MgSO₄ infusions have commonly been continued for 24 h postpartum. However, in a recent study of

women with mild pre-eclampsia, the MgSO_4 infusion was continued for 12 h postpartum, and there was no difference in the clinical course compared with those women who received MgSO_4 for 24 h [86] (Level 1+). Using clinical assessment rather than time, Isler and colleagues suggested several parameters for consideration of cessation including: absence of headache; visual changes and epigastric pain; sustained blood pressure of less than 150/100 mmHg without antihypertensive therapy; and a spontaneous diuresis $> 100 \text{ ml.h}^{-1}$ for at least $< 2 \text{ h}$ [87].

Management of postpartum complications

Management of acute pulmonary oedema

Pulmonary oedema may occur in up to 2.9% of women with pre-eclampsia with only 30% of cases occurring before delivery [88]. Management of acute pulmonary oedema has been recently reviewed [89]. In addition to stabilisation of the mother and expediting resolution of the acute pulmonary oedema, consideration needs to be given to delivery of the fetus, if acute pulmonary oedema occurs in the antenatal period. Treatment follows similar practices to those used in the non-obstetric population. Oxygen saturation monitoring and oxygen supplementation either via non-invasive ventilation devices or intubation and ventilation are used depending on the severity of the respiratory compromise. Intravenous furosemide (bolus 20–40 mg over 2 min) is used to promote venodilation, with repeated doses of 40–60 mg after approximately 30 min, if there is inadequate diuretic response (maximum dose 120 mg.h^{-1}). Intravenous morphine 2–5 mg, fluid restriction and strict fluid balance and positioning such that the head is elevated and antenatal uterine displacement is maintained, should also be used [90, 91].

Management of oliguria in the immediate postpartum period

Oliguria in the postpartum period is multifactorial and in the presence of normal renal and respiratory function usually requires no treatment. The use of furosemide or low-dose dopamine for the management of oliguria in a woman with normal renal function is not recommended [92] (Level 1++).

Management of severe hypertension in the immediate postpartum period

Deaths due to severe hypertension and morbidity from stroke occur in the postpartum period. Although it is clear that antenatal hypertension may persist in the postpartum period, it is not clear what leads to new onset hypertension in this period. Commonly used drugs in this situation include hydralazine, nifedipine, frusemide and methyldopa [39, 93] (Level 1++).

Management of acute renal decompensation

Acute renal failure may occur in women with pre-eclampsia, particularly if there is pre-existing kidney disease. Indications for haemodialysis include persistent acidaemia, hyperkalaemia, volume overload and uraemia. Women with pre-eclampsia requiring renal replacement therapy should be transferred to a Level-3 (intensive care) unit for management [83].

Breastfeeding in women with pre-eclampsia

Ideal drugs have low milk:maternal plasma ratios. Satisfactory drugs in this group include methyldopa, β -adrenoceptor blockers with high plasma protein binding e.g. oxprenolol, angiotensin converting enzyme inhibitors (captopril, enalapril) and some dihydropyridine calcium channel blockers (nifedipine [93] Level 1++). Methyldopa is associated with increased risk of postnatal depression. Magnesium sulphate is excreted in the breast milk but is safe for breastfed infants [94]. Bromocriptine used for lactation suppression is associated with hypertension [95].

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