

the initial clinical condition of the child; the extent and duration of the surgery; the metabolic, fluid and thermal challenges experienced; anticipated postoperative course; and the likelihood of complications). Some procedures require a period of postoperative ventilation (e.g. diaphragmatic hernia, cardiac and some intra-abdominal procedures), but in many cases, return to the neonatal unit or HDU is anticipated.

Extubation – at the end of surgery and anaesthesia, reversal of neuromuscular blockade with atropine and neostigmine is the rule. The inhalational agents are washed out with 100% oxygen and the tracheal tube is not removed until adequate spontaneous respiration and vigorous bodily movements are achieved. The tracheal tube is well tolerated in an awake baby, though a phase of coughing, salivating, irregular breathing and breath-holding often occurs during awakening from anaesthesia. Resist the temptation to extubate the trachea until the baby virtually does it for you. Laryngospasm is almost as common on extubation as before intubation. It may be associated with hypoxia and bradycardia. It requires good airway management, PEEP, 100% oxygen and, occasionally, succinylcholine and re-intubation.

Analgesia – the adequacy of pain relief can be reviewed in the recovery ward. Adjustment may be required.

Apnoea and other monitoring – postoperative monitoring is essential. Apnoea is one of the most common problems in the first 24 hours postoperatively in neonates aged less than 56 weeks' postconceptional age. Use of an apnoea mattress or transthoracic impedance monitoring through the ECG leads is commonly used. An oxygen saturation monitor is a late detector of apnoeic episodes; however, it also gives information on heart rate. Blood pressure is a useful trend monitor. Temperature, blood glucose, central venous pressure, urine output, and observation of stoma or wound drainage are invaluable.

A thermoneutral environment is ideal and best provided by an enclosed incubator or an open resuscitaire cot with servo-control of the overhead heater.

Fluids and feeding – postoperative fluid management (Figure 3) varies with surgical procedure and the general condition of the neonate. It is often decided by the surgical team. Electrolytes should be checked daily, with supplements of potassium as appropriate. Total parenteral nutrition may be required for some patients. Minor surgery not involving the gut may not require intravenous fluids, merely an early return to normal feeding.

Discharge criteria – few neonates are suitable for day-case anaesthesia. Most require overnight admission to re-establish normal feeding, to ensure adequate analgesia and for apnoea monitoring. ◆

FURTHER READING

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Pre-eclampsia and HELLP syndrome

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In *The Confidential Enquiries into Maternal Deaths in the United Kingdom*, for 1999,¹ hypertensive disorders remained one of the highest causes of direct maternal death, after thromboembolic disease and early pregnancy causes. Fifteen women died from pre-eclampsia or eclampsia; five were associated with HELLP (haemolysis, elevated liver enzymes and low platelet count) syndrome.

Pre-eclampsia is difficult to define and several different definitions are used in the literature. Peripheral oedema should not be part of the definition. It is generally agreed that hypertension in pregnancy (Figure 1), is blood pressure greater than 140/90 mm Hg, using the Korotkoff phase V sound for the diastolic value, on two occasions, 4 hours apart. Significant proteinuria is defined as more than 300 mg of proteinuria in a 24-hour collection of urine. When these parameters are breached, perinatal and maternal morbidity and mortality rates increase. HELLP syndrome can deteriorate very rapidly, resulting in fetal and maternal demise.

Pathophysiology

There is a two-stage theoretical model (Figure 2) for development of pre-eclampsia.²

Stage one involves reduced placental perfusion. This allows screening in high-risk women using Doppler waveflow studies of the uterine arteries at 20 and 24 weeks of pregnancy, looking for diastolic notches or raised pulsatility index, which suggests an increased risk of developing pre-eclampsia and/or intrauterine growth restriction. The reduced oxygen delivery, from the embarrassed placental circulation, increases the release of cytokines and other factors into the general circulation, thus causing the systemic features of the disease.

The second stage is thought to be brought about largely through the mechanism of oxidative stress, generated in the placenta,

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Classification of pregnancy hypertension

Pre-existing hypertension/chronic hypertension

- 3–5% of pregnancies
- Blood pressure $\geq 140/90$ mm Hg, before week 20 of pregnancy or hypertension diagnosed in pregnancy that persists 6 weeks after delivery

Gestational (transient) hypertension

- 6–7% of pregnancies
- Blood pressure developing after week 20 of pregnancy, which settles within the 6 weeks of the puerperium, with no associated features

Pre-eclampsia – de novo

- 2–8% of pregnancies
- Superimposed on pre-existing hypertension. 15–25% of women with pre-existing hypertension
- Worsening control of hypertension, associated with new-onset proteinuria or a doubling in any proteinuria measured quantitatively, during the first trimester, after week 20 of pregnancy

Eclampsia

- Rare in developed countries, affecting 1/2000 deliveries, but can vary from 1/100 to 1/1700, in less developed countries
- Characterized by convulsions in any woman with hypertension in pregnancy, or who develops hypertension following the convulsion

HELLP (Haemolysis, Elevated Liver enzymes, Low Platelets) syndrome

- About 3% of women with pre-eclampsia
- One of the more severe manifestations of pre-eclampsia

in response to the impaired placentation. How this causes the subsequent systemic changes in the mother is not known, but magnification of the oxidative stress in the placenta, to oxidative stress at the level of the maternal endothelium is thought to occur, through cytokine release, leading to cell injury, vasoconstriction and activation of the coagulation cascade.

Predisposing factors

It is important to recognize women at risk of developing pre-eclampsia because therapeutic interventions can significantly reduce the risk of prematurity and intrauterine growth restriction for the fetus and reduce the risk of maternal complications, particularly in women at high risk.

Age – women under 20 and over 35 years of age have a significantly increased risk of developing pre-eclampsia.

Body mass index above 30 – obesity is an independent risk factor for pre-eclampsia. It increases anaesthetic risk and is associated with increased risk of cardiovascular disease, diabetes and hypertension.

Ethnicity – there is a suggestion that pre-eclampsia is more common and/or more severe in particular ethnic groups (e.g. West Africans). This may be associated with a higher incidence of chronic hypertension, but further work is necessary to confirm this.

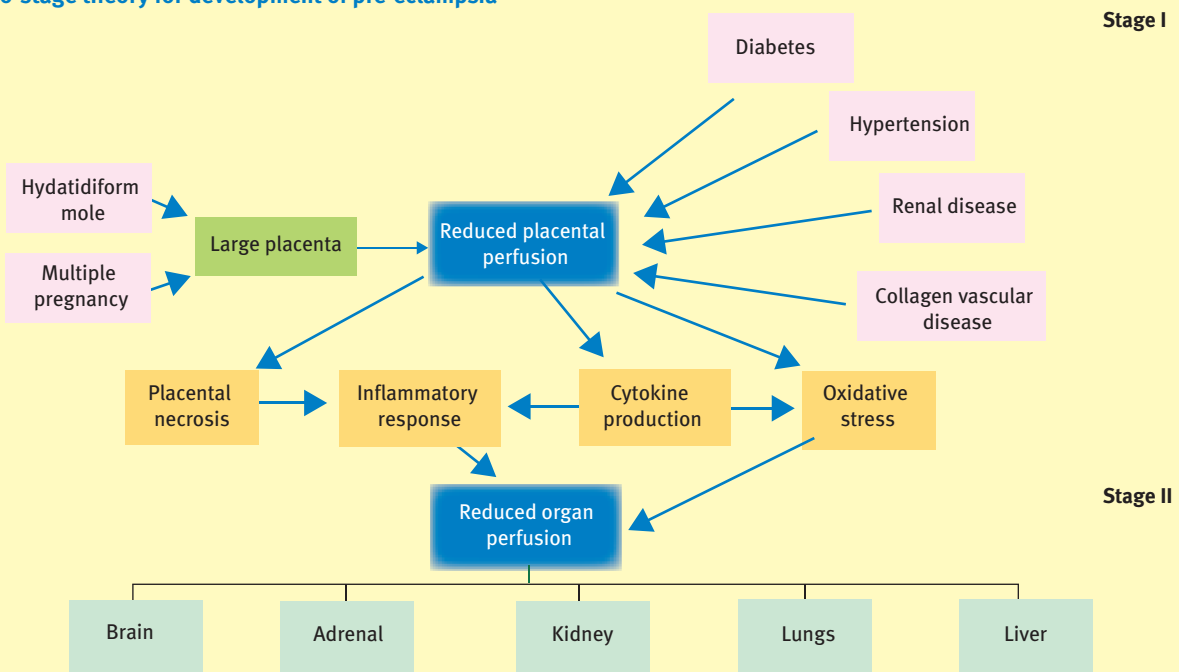
Cigarette smoking decreases the risk of developing pre-eclampsia by up to 40%.

Family history – the risk of pre-eclampsia is increased by up to 25% if there is a history of pre-eclampsia in the mother of the pregnant woman and by up to 40% if she has an affected sister.

Paternity – men who have fathered a previous pre-eclamptic pregnancy are twice as likely to father a pre-eclamptic pregnancy in a new relationship. A change in partner also increases the risk

1

Two-stage theory for development of pre-eclampsia



2

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