Subsequently, give oral thiamine 50 mg three times daily until vomiting has stopped.

**Other management on discharge from hospital**

Withhold iron tablets until vomiting has resolved, but ensure that they are taken subsequently, as iron-deficiency anaemia may have been an important consequence of the hyperemesis.

Try to help with any depression that is present and also, if resources to address intimate partner violence are available in the community, make sensitive inquiries of the woman or girl in case this is a contributing factor.

---

### 2.7 Medical disorders complicating pregnancy and delivery

#### 2.7.A Heart failure during pregnancy, including rheumatic heart disease

**BOX 2.7.A.1 Minimum standards**

- Oxygen.
- Furosemide.
- Digoxin.
- Nitroglycerine sublingual tablets.
- Blood transfusion.
- Morphine.

**Introduction**

Serious cardiac pathology may present either as heart failure where respiratory distress is the most obvious finding, or as cardiogenic shock (see shock section later).

**Causes of heart failure during pregnancy**

There are five main causes of heart failure in pregnancy:

1. severe anaemia
2. structural heart disease
3. circulatory overload (e.g. excessive IV fluids)
4. hypertension in severe pre-eclampsia
5. hypertrophic cardiomyopathy (HCM) and peripartum cardiomyopathy.

Heart failure can result from:

- left ventricular volume overload (aortic and mitral valve incompetence) or excessive pulmonary blood flow (e.g. congenital heart defects)
- left heart obstruction (aortic stenosis, mitral stenosis, hypertension)
- primary pump failure (severe anaemia, myocarditis, cardiomyopathy or arrhythmia)
- over-transfusion (a particular risk in hospital with IV blood or fluid infusions, especially in the anaemic mother).

**Clinical signs**

These include the following:

- respiratory distress (raised rate and some chest wall recession)
- tachycardia out of proportion to respiratory difficulty

**Jugular venous pressure**

Normal levels of jugular venous pressure (JVP) are 4–5 cm above the sternal angle. In heart failure the JVP can be raised so that the external jugular vein is filled up to or above the angle of the jaw (see Figure 2.7.A.1).

**Treatment of severe decompensated heart failure**

- Assess ABC.
- Sit the patient upright and ensure bed rest.
- Give a high concentration of oxygen via face mask with reservoir bag.
- If there are signs of shock (poor pulse volume or low blood pressure with extreme pallor and depressed conscious level), treat for cardiogenic shock with inotropes (if available).
- If there are signs of pulmonary oedema, give IV furosemide 40 mg (and repeat as required).
The following are useful investigations if available:

- full blood count (to exclude severe anaemia)
- serum urea and electrolytes
- infection screen, including blood cultures
- 12-lead electrocardiogram
- chest X-ray
- echocardiogram.

Subsequent treatment of heart failure

- Dietary sodium restriction.
- Loop diuretics (furosemide) for moderate/severe pulmonary oedema.
- Treatment with oral hydralazine or oral nifedipine (modified release version) as a vasodilator (instead of glyceryl trinitrate used in the acute scenario above).
- Treatment with a B-blocker (preferably a B1 cardio-selective B-blocker such as atenolol) can be beneficial. They are NOT used in the acute presentation of a patient with severe decompensated heart failure. They are contraindicated in asthma and may be associated with intra-uterine growth restriction, but are not associated with congenital malformations.
- Continued treatment with digoxin (as above) should be considered if significant symptoms persist.

The following useful investigations if available:

- 375–750 micrograms, then after 6 hours 187.5–375 micrograms.
- Or give an oral digoxin loading dose (instead of IV) of 375–750 micrograms, then after 6 hours 187.5–375 micrograms, then after a further 6 hours 125–250 micrograms over 10 minutes.

- Maintenance digoxin dose IV or oral:
  - 125–750 micrograms once daily.
  - Reduce the dose in renal impairment. Be alert for low K⁺ levels.

- Consider thromboprophylaxis. This treatment must take into account any bleeding risk and the timing of delivery.

- Check for severe anaemia (especially if the haemoglobin concentration is < 5.0 g/dL), for which partial exchange transfusion may be helpful. Partial exchange transfusion can be achieved with a cannula in a large vein in the antecubital fossa. Withdraw 25 mL of anaemic blood and infuse 50 mL of new blood over 5 minutes, and repeat up to 10 times. An alternative is careful transfusion of packed cells (hang the bag vertically for 15 minutes) to allow the red blood cells to separate from the plasma. Transfuse only the red blood cell component with 40 mg IV furosemide for each unit of 500 mL infused.

- Provided the patient is not hypotensive (systolic blood pressure > 90 mmHg) and has no serious obstructive valvular disease, give a glyceryl trinitrate tablet 500 micrograms sublingually and repeat up to a total of 3 tablets.

- Give morphine 3 mg over 5 minutes, and consider repeating after 15 minutes. Morphine is effective in reducing the afterload and, in addition, will reduce anxiety and pain both of which are likely to make heart failure worse.

- For patients with persisting heart failure load with digoxin IV, 250–500 micrograms over 10 minutes, then after 6 hours 125–250 micrograms over 10 minutes, then after a further 6 hours 125–250 micrograms over 10 minutes.

- Treatment with a B-blocker (preferably a B1 cardio-selective B-blocker such as atenolol) can be beneficial.

- Treatment with oral hydralazine or oral nifedipine (modified release version) as a vasodilator (instead of glyceryl trinitrate used in the acute scenario above).

- Limit infusion of IV fluids to decrease the risk of circulatory overload, and maintain a strict fluid balance chart.

- Ensure adequate analgesia.

- If an oxytocin IV infusion is required, use a higher concentration at a slower rate while maintaining a fluid balance chart (e.g. the concentration may be doubled if the number of drops per minute is decreased by half).

- Increase the rate of oxytocin infusion until regular strong contractions are established, and then maintain infusion at that rate.

- Avoid sustained, bearing-down efforts during the second stage if possible.

- If it is necessary to decrease the woman’s workload during delivery, perform an episiotomy and assist delivery by vacuum extraction or forceps.

- Ensure active management of the third stage of labour. Oxytocin given on delivery of the baby must be given very slowly IV (5 units diluted in 20 mL of 0.9% saline over 5–10 minutes) to avoid hypotension.

- Do not give ergometrine.

Note: Heart failure is not an indication for Caesarean section.

Management of anaesthesia if Caesarean section is needed

It is assumed that epidural anaesthesia/analgesia is unlikely to be possible or appropriate in low-resource settings. Avoid spinal anaesthesia if there is a fixed cardiac output, such as aortic/mitral stenosis or heart failure associated with valvular disease. If you are giving a general anaesthetic, take precautions against aspiration and minimise the risk of an increase in blood pressure associated with intubation by premedication with either morphine (5 mg initially IV) or lignocaine (1 mg/kg IV). If the patient is considered to have insufficient cardiovascular stability for general anaesthetic, undertake Caesarean section under local infiltration anaesthesia.

In all of the above situations the surgeon should be ready to start operating immediately when anaesthesia is established, so that the operating time is as short as possible. As above, oxytocin given for active management of third stage must be given very slowly IV (5 units diluted in 20 mL of 0.9% saline over 5–10 minutes) to avoid hypotension.

Post-operative management must ensure adequate analgesia with morphine.
Cardiac consequences of rheumatic heart disease and their effects on pregnancy

Introduction

After an episode of acute rheumatic fever, there may be permanent valve damage. Rheumatic heart disease occurs when acute valve inflammation is followed by scarring and fibrosis, resulting in various degrees of shortening, thickening, rigidity, deformity, retraction and fusion of the valve cusps. The commonest valve lesions are mitral regurgitation, mitral stenosis and aortic regurgitation. Rheumatic heart disease is most severe and progressive in the following:

- patients who initially have severe carditis
- patients who have recurrent episodes of acute rheumatic fever. The prognosis is more favourable if recurrences are prevented. After single episodes, residual cardiac disease may disappear or improve, and valve damage only worsens in a few cases. It is therefore crucial to maintain continuous antibiotic prophylaxis to prevent further valve damage.
- Asymptomatic rheumatic valve disease often becomes clinically relevant during pregnancy. A history of rheumatic fever should be sought at booking and ideally the heart auscultated for murmurs. It should be remembered as a differential diagnosis in a woman presenting with shortness of breath, a cardiac arrhythmia or heart failure.

When a cardiology referral and/or surgical intervention is not available

- Medical management is supportive, aiming to maximise cardiac function. The most dangerous period is delivery and shortly afterwards. In general, the more normal the delivery the less stress there is on the heart.
- Regular follow-up and rational drug therapy can make a significant difference. Use routine medications (diuretics, B-blocker, digoxin and nitrates) to maximum effect.
- Bed rest and the avoidance of heavy work are essential.
- Treat anaemia and any other coexisting conditions.
- Advise hospital delivery.
- Venesection can be used to decrease venous load for the patient in life-threatening situations where preload is high.

Note that there is a risk of teratogenicity with ACE inhibitors during the first trimester, and problems with placental and renal function later in pregnancy. There is also diminished placental perfusion with diuretics although these should not be withheld if clinically indicated. ACE inhibitors may be used after delivery and during breastfeeding.

Effects of rheumatic heart disease

Mitral regurgitation

Mitral regurgitation is the commonest valve lesion.

Clinical features

These include the following:

- easy fatigue (caused by low cardiac output)
- shortness of breath on exertion (caused by pulmonary oedema and inability to increase cardiac output)
- orthopnoea, paroxysmal nocturnal dyspnoea and haemoptysis
- hyperdynamic apical impulse
- apical impulse displaced laterally and inferiorly
- a blowing apical pansystolic murmur radiating to the left axilla; there may be a third heart sound and a short low-frequency mid-diastolic murmur from increased trans-mitral flow
- there may be basal creptations
- chest X-ray demonstrates cardiomegaly and left atrial enlargement (a double density on the right heart border and elevation of the left main bronchus)
- the ECG demonstrates left atrial enlargement (broad bifold P waves in lead II and a prominent negative component to the P in V1) and left ventricular hypertrophy
- signs of pulmonary hypertension.

Management

- Urgent referral for a cardiology opinion, as surgery is likely to be necessary (if available).
- Annual echocardiograph (if available), as progressive left heart dilation may result in irreversible left ventricular dysfunction if referral is delayed until symptoms develop.
- Medical treatment for heart failure, but patients who are unwell enough to require this may be more appropriately treated by mitral valve repair or mitral valve replacement with a mechanical valve or bioprosthesis, if possible locally.

Mitral stenosis

Features

- Mild stenosis does not cause symptoms, moderate stenosis causes shortness of breath on exertion, and severe stenosis causes easy fatigue, shortness of breath at rest, orthopnoea (shortness of breath on lying down), paroxysmal nocturnal dyspnoea and haemoptysis.
- There is a low-frequency mid-diastolic murmur that is maximal at the apex, accentuated by exercise.
- There is a loud first heart sound and diastolic opening snap.
- The murmur becomes longer as the severity of stenosis increases.
- In severe cases, there are signs of pulmonary hypertension.
- Chest X-ray and ECG show left atrial enlargement when there is moderate mitral stenosis, and chest X-ray shows pulmonary oedema when stenosis is severe.

Management

- Symptoms are treated with diuretics and a low-sodium diet. Digoxin is indicated only in rare cases where there is atrial fibrillation secondary to left atrial enlargement.
- Symptomatic patients and those with pulmonary hypertension should be referred for cardiology review (if available), as surgery is often necessary (open or closed mitral commissurotomy, mitral valve replacement and percutaneous catheter balloon mitral commissurotomy).

Aortic regurgitation

- This is less common than mitral regurgitation, and frequently occurs in combination with mitral valve disease.
- Symptoms occur when left ventricular dysfunction develops secondary to chronic left ventricular volume overload.
- Once the symptoms have appeared, deterioration is often rapid.
- Symptoms include exercise intolerance, shortness of
breath on exertion, orthopnoea (shortness of breath on lying down), paroxysmal nocturnal dyspnoea, haemoptysis and chest pain.

- Examination reveals a blowing decrescendo early diastolic murmur that is maximal at the mid to lower left sternal border. The murmur is loudest when sitting forward with the breath held in expiration.

**Signs of moderate to severe aortic regurgitation**

- The murmur lengthens and may be present throughout diastole.
- Hyperdynamic apex.
- Apical impulse displaced laterally and inferiorly.
- Wide pulse pressure.
- Collapsing pulses.
- Basal crepitations.
- Visible pulsations in the suprasternal notch and neck vessels.
- Systolic murmur at the upper right sternal border (from increased aortic valve flow).

**Management**

- Cardiology assessment, as surgery may be necessary (if available). Marked cardiomegaly on chest X-ray or multiple ventricular ectopics on the ECG should prompt referral.
- An echocardiogram is needed at least annually (if available), as it is important to assess left ventricular dilation and function to ensure that surgery is performed before irreversible left ventricular dysfunction develops.
- Exercise tolerance may be improved by medical treatment for heart failure.

**2.7.B Asthma**

**BOX 2.7.B.1 Minimum standards**

- Oxygen.
- Salbutamol by metered-dose inhaler and nebuliser.
- Aminophylline.
- Magnesium sulphate.
- Adrenaline.
- Prednisolone/hydrocortisone.

**Assessment**

**Features of severe asthma**

- Too breathless to feed or talk.
- Recession/use of accessory muscles.
- Respiratory rate > 40 breaths/minute.
- Pulse rate > 120 beats/minute.

**Features of life-threatening asthma**

- Conscious level depressed/agitated.
- Exhaustion.
- Poor respiratory effort.
- SaO₂ < 85% in air/cyanosis.
- Silent chest.

Asthma complicates 3–4% of pregnancies. Pregnancy is associated with worsening of the symptoms in one-third of affected mothers.

- A chest X-ray is indicated only if there is severe dyspnoea, uncertainty about the diagnosis, asymmetry of chest signs (possible pneumothorax) or signs of severe infection.
- Transcutaneous PCO₂, arterial or capillary blood gases (if available) can be helpful in very severe asthma.
- Continuous pulse oximetry is valuable (if available), as hypoxaemia is a major feature of all severe asthma attacks.
- Do not give prostaglandins other than misoprostol (the latter is safe in pregnancy). For the prevention and treatment of postpartum haemorrhage, give oxytocin 10 units IM or ergometrine 500 micrograms IM or both (Syntometrine IM).
- Do not give labetalol for hypertension in patients with asthma.

**Aortic stenosis**

The two commonest causes of aortic valve stenosis are progressive wear of a congenital bicuspid aortic valve and rheumatic fever (the most common cause in developing countries, and usually also with aortic regurgitation).

**Clinical features**

- Chest pain (angina from inadequate coronary artery perfusion).
- Fainting, usually with exertion or excitement.
- Shortness of breath due to heart failure.
- Sudden death.
- On examination, delayed upstroke and reduced magnitude of the carotid pulse and an ejection systolic heart murmur.
- The ECG shows left ventricular hypertrophy and sometimes ST changes of myocardial ischaemia.
- The chest X-ray shows a normal sized heart, dilated aortic root and pulmonary venous congestion. Sometimes there is calcification of the aortic valve.

**Management**

- Avoid strenuous exercise.
- Avoid endocarditis.
- Refer for a specialist opinion if possible.
- Diuretics can be helpful, but surgery is usually required.
The aim should be for the patient to need her salbutamol inhaler no more than 1–2 times/day. If use in excess of this occurs, the patient should be commenced on inhaled steroids or have her current dose of inhaled steroids increased.

If the maximum dose of inhaled steroids is reached, then long acting B2-agonists and slow release theophylline should be considered if available.

If not available, or ineffective, oral prednisolone can be added at the lowest dose required to maintain control. Oral prednisolone is associated with an increased risk of infection and gestational diabetes, and complicates control of established diabetes. Its long term use has other potential side-effects for the mother, such as osteoporosis, but it should not be withheld if required to maintain control of maternal disease.

### Emergency treatment of severe asthma

- **OXYGEN**
  - Prop up in lateral tilt position
  - Salbutamol inhaler 100 microgram/puff: 2 puffs over approximately 1 minute

- **Continue oxygen**

- **Is the mother improving?**
  - **YES**
    - Shake salbutamol inhaler: 2 puffs over approximately 1 minute
    - Repeat x 5 cycles as long as improving, with each cycle given every 5–10 minutes
    - Give oral prednisolone 30–60 mg, or if not able to take oral medicine hydrocortisone 100 mg IV/IM 6-hourly
  - **NO**
    - Nebuliser driven by oxygen
      - Salbutamol 5 mg
      - Continuous salbutamol by nebuliser or inhaler (if nebuliser not available)
      - Repeat dose as soon as the last one finishes
    - If NOT improving
      - Subcutaneous/IM adrenaline 0.5–1 mg
      - If NOT improving
        - IV aminophylline 250 mg as loading dose over 15 minutes
        - Then IV infusion of 1 mg/kg/hour

- **If the patient is not responding, or their condition is deteriorating:**
  - Nebulised salbutamol may be given continuously.
  - In acute severe asthma, 2 g of magnesium sulphate IV in 50 mL of 0.9% saline over 10–15 minutes can improve respiratory function.

- IV salbutamol 250 micrograms over 10 minutes is an alternative to magnesium sulphate or aminophylline, followed by IV infusion of 1–5 micrograms/kg/minute (but monitoring ECG and checking K⁺ levels regularly is necessary; extra potassium may be needed, and monitoring of plasma K⁺ levels is essential if this drug is given IV).

- In severe cases in the absence of other measures, adrenaline can be effective. It should be given subcutaneously or IM (dose = 500 micrograms to 1 mg), but may be given IV in life-threatening asthma as follows.
  - Place 1 mg of adrenaline in 10 mL of 0.9% saline and give 1 mL of this solution. Wait for 1 minute and then keep on repeating 1 mL doses IV every minute until the patient improves or the whole 1 mg (10 mL) has

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**FIGURE 2.7.B.1 Pathway of care for pregnant mother with severe asthma.**

---
been given. The risk of cardiac side effects (tachycardia, cardiac arrhythmias) is low if adrenaline is given in this way.

- In patients with poor respiratory effort, depressed conscious level and poor oxygenation despite maximum oxygen therapy:
  - Attempt to support ventilation with a bag-valve-mask.
  - Summon experienced support (an anaesthetist) if available, and consider intubation for mechanical ventilation with IV ketamine or halothane induction.

**Indications for intubation and positive pressure ventilation (if available)**

These include the following:

- increasing exhaustion
- progressive deterioration in clinical condition (e.g. a silent chest)
  - oxygenation decreasing and/or oxygen requirement increasing
  - pCO₂ increasing (if measurable from arterial/capillary gas)
- sudden deterioration
- massive atelectasis
- pneumothorax.

If the patient is responding and improving, continue inhaled salbutamol as often as indicated.

**Other measures**

- Reassure the patient, and avoid upsetting them by performing unnecessary invasive procedures.
- Give IV steroids to cover labour/delivery for prevention of Addisonian crisis in patients with a history of taking significant doses of oral steroids in the recent past, especially if long term.
- Restrict IV fluids to two-thirds of the normal requirements.
- Give antibiotics only if there are signs of infection (fever and other signs of pneumonia; chest X-ray may be helpful).
- When the patient has recovered, review their maintenance treatment and inhaler technique.

**How to give drugs such as aminophylline or magnesium sulphate safely IV without syringe drivers or pumps**

- **Bolus doses:**
  - The safest way to give these is slowly by hand using the syringe.
- **IV infusions:**
  - Where volume overload is not an issue, the simplest method is to add the drug (e.g. aminophylline) to 500-mL bags of Ringer-lactate or Hartmann’s solution or other available/appropriate fluid and run over 12–24 hours.
  - Where volume overload is an issue, a microburette (if available) can be used to give small volumes of IV fluids or drugs safely (see Figure 2.7.B.2). The chamber in the figure holds 100 mL (1 drop/second = 1 mL/minute).

![Figure 2.7.B.2 Burette for careful infusion.](image.png)
2.7.C Anaphylaxis

**BOX 2.7.C.1 Minimum standards**
- Adrenaline.
- Hydrocortisone and prednisolone.
- Nebulised adrenaline.
- Antihistamine.
- Nebulised or inhaled salbutamol.

**Introduction**
Anaphylaxis is an allergic reaction to ingested, inhaled or topical substances, which may present as one or more of stridor, shock or respiratory distress. Common causes include allergy to penicillin, to radiographic contrast media, to blood transfusion, to insect bites and to certain foods, especially nuts. Anaphylaxis can occur with any drug.

**Clinical features**
Consider the possibility of anaphylaxis in a patient with any of the symptoms and signs listed in Table 2.7.C.1, especially when any of the following are present:
- a history of previous severe reaction
- rapidly progressive or increasingly severe symptoms
- a history of asthma, eczema or rhinitis (atopy)
- current treatment with beta-blockers.

This condition is potentially life-threatening, and may result in a change in conscious level, collapse, and respiratory or cardiac arrest. Some patients carry their own adrenaline.

**Treatment**
- Remove or stop the allergen if possible.
- Adrenaline 1 mg is given IM, unless there is intractable shock or cardiac arrest on presentation, in which case give adrenaline IV as follows:
  - Place 1 mg of adrenaline in 10 mL of 0.9% saline and give 0.5–1 mL of this solution. Wait for 1 minute and then keep on repeating 1 mL doses IV every minute until the patient improves or the whole 1 mg (10 mL) has been given.
- IV/IM hydrocortisone, 100–300 mg (if IV by slow injection) or oral prednisolone 40 mg stat.
- Nebulised adrenaline if there is stridor.
- Nebulised salbutamol 5 mg by oxygen driven nebulizer or adrenaline if there is wheezing.
- Antihistamine; chlorphenamine 10–20 mg by slow intravenous injection.
- Intubation and ventilation (if available) will be required for severe cases.

**TABLE 2.7.C.1 Anaphylaxis: symptoms and signs**

<table>
<thead>
<tr>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td>Burning sensation in mouth</td>
<td>Coughing and/or wheezing</td>
</tr>
<tr>
<td></td>
<td>Itching of lips, mouth and throat</td>
<td>Loose bowel movements</td>
</tr>
<tr>
<td></td>
<td>Feeling of warmth</td>
<td>Sweating</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>Irritability</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain</td>
<td></td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td>Urticarial rash</td>
<td>Bronchospasm</td>
</tr>
<tr>
<td></td>
<td>Angio-oedema</td>
<td>Tachycardia</td>
</tr>
<tr>
<td></td>
<td>Conjunctivitis</td>
<td>Pallor</td>
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<td></td>
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<td></td>
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</tr>
</tbody>
</table>

Mild anaphylaxis can lead to moderate and then severe anaphylaxis, and then to death, unless treated.
Section 2.7

Remove allergen

Assess airway

Partial obstruction/stridor

Complete obstruction

No problem

Assess breathing

Wheeze

Apnoea

No problem

Assess circulation

No pulse

Shock

No problem

Reassess ABC

Airway deterioration

No problem

Observe

Adrenaline 0.5 – 1 mg IM (0.5 – 1 mL of 1 in 1000)
Nebulised adrenaline 5 mL of 1 in 1000
Repeat nebuliser every 10 minutes as required
Hydrocortisone 100 mg IV 6-hourly

Intubation or surgical airway

Adrenaline 1 mg IM (1 mL of 1 in 1000)
Nebulised adrenaline 5 mL of 1 in 1000 or salbutamol 5 mg using oxygen if possible
Repeat nebuliser every 10 minutes as required
Hydrocortisone 100 mg IV 6-hourly
If no response aminophylline 250 mg in 10 mL 0.9% saline IV over 15 minutes

Bag-mask ventilation

Cardiac compressions plus bag-mask ventilation

Ringer-lactate or Hartmann’s 500 mL to 1 L IV rapidly as bolus

REPEAT ADRENALINE 1 mg IM (1 mL of 1 in 1000) EVERY 10 MINUTES AS LONG AS SEVERE ANAPHYLAXIS CONTINUES

FIGURE 2.7.C.1 Pathway of care for anaphylaxis in pregnancy. ABC, airway, breathing and circulation.
Two children with pneumonia receiving oxygen from one oxygen concentrator; the only one available in a ward of 26 beds.

Two senior midwives in Liberia trained in advanced obstetrics undertaking an emergency Caesarean section.
2.7.D Diabetes mellitus

Introduction
Diabetes mellitus is associated with increased maternal mortality and morbidity, as well as increased perinatal mortality and morbidity, including congenital malformations. Pregnancy causes changes in the maternal physiology to make it a diabetogenic state. Women who have pre-existing diabetes have an increased insulin requirement in pregnancy. Previously healthy women may develop gestational diabetes. Both type 2 diabetes and gestational diabetes are more common in certain ethnic groups, including South Asians, and are more common in those with a high body mass index (BMI).

Before the discovery of insulin, maternal mortality in diabetics and perinatal mortality in their infants were extremely high. Insulin has led to a dramatic improvement in maternal survival, but in comparison with non-diabetic pregnancy there is still a three- to fivefold increase in perinatal mortality, and an increase in congenital malformations. These risks can be reduced by strict attention to the control of the diabetes both before and during pregnancy.

Diabetes predisposes to pre-eclampsia.

Management

Before pregnancy
- Advise any diabetics of reproductive age about the importance of close monitoring and modified treatment in pregnancy.
- Obesity: give dietary advice.
- Tight control of diabetes: aim for blood glucose levels of less than 7.5 mmol/litre and HbA1c levels within normal limits.
- The mother should take folic acid 5 mg daily if planning pregnancy.

In early pregnancy
- Nausea and vomiting are common.
- Hypoglycaemia is common in insulin-treated diabetes. Provide glucagon at home if possible, and explain its use to other household members. Alternatively, counsel the patient to keep sugar-containing foods close by. Inform the patient and others about the signs of hypoglycaemia.
- It is not always necessary to convert mothers treated with oral hypoglycaemic agents to insulin. Metformin is commonly used in these circumstances (initially 500 mg with breakfast for 1 week, then 500 mg twice daily with breakfast and tea, and then 500 mg three times daily with breakfast, lunch and tea).
- As soon as possible, assess the gestational age. Early ultrasound scan can detect anencephaly, but 20 weeks’ gestation is usually the best time to look at the spine and heart if facilities are available.

During pregnancy

Type 1 diabetes (insulin dependent)
Close control of diabetes is needed. Expect insulin requirements to increase by up to 50% above pre-pregnant levels. There is an increased risk of congenital abnormalities, macrosomia, polyhydramnios, preterm labour and pre-eclampsia. Plan delivery with care. The risks of infection and development of diabetic ketoacidosis are high. Signs of hyperglycaemia include a gradual onset of drowsiness and polyuria, dehydration, hypotension, difficulty breathing, and a ketotic smell to the breath. Signs and symptoms of hypoglycaemia may be of rapid onset, leading to unconsciousness, particularly if the mother has taken insulin but has not taken her usual food. Awareness of impending hypoglycaemia in those with type 1 diabetes is often reduced in pregnancy. These patients must be advised about the possible effects on safety during driving.

The insulin requirement often escalates rapidly, especially in the late second and early third trimester, and in order to maintain control of the blood glucose, frequent medical review every 1 to 2 weeks coupled with frequent self-assessment of blood glucose levels, is likely to be required for women with type 1 diabetes.

Type 2 diabetes
Women who are diet-controlled before pregnancy require careful monitoring of blood sugar levels in pregnancy, and may need metformin and/or insulin.

Gestational diabetes
This is often undiagnosed, and should be suspected if any of the following are present:
- a family history of diabetes
- a past history of a large baby, stillbirth or gestational diabetes
- recurrent glycosuria
- a high BMI (overweight)
- a relevant ethnic background.

All women with diabetes should ideally be monitored more regularly in the antenatal clinic for complications such as

Diagnosis of diabetes with a glucose tolerance test

<table>
<thead>
<tr>
<th>TABLE 2.7.D.1 Seventy-five-gram oral glucose loading dose results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting plasma glucose concentration (mmol/litre)</td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Gestational impaired glucose tolerance</td>
</tr>
<tr>
<td>Normal</td>
</tr>
</tbody>
</table>
International Maternal & Child Health Care

pre-eclampsia, polyhydramnios and a large or small for gestational age infant.

Management of delivery in women with diabetes

For spontaneous labour, induction of labour and elective Caesarean section:
1. Measure glucose on admission and hourly during labour.
2. Site an IV line with 500 mL of 0.9% saline containing 10% dextrose and potassium chloride 10 mmol, and give at a rate of 60 mL/hour.

Avoid the routine use of insulin in labour in low resource settings because of lack of experience and lack of blood glucose stick tests. In mothers who were using insulin during pregnancy and those where blood glucose is > 7 mmol/litre on two successive occasions one hour apart in labour, the insulin requirements shown in Table 2.7.D.2 below can be used.

**TABLE 2.7.D.2 Insulin requirements**

<table>
<thead>
<tr>
<th>Blood glucose concentration (mmol/litre)</th>
<th>Hourly subcutaneous injections of insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2.0</td>
<td>No insulin; dextrose only</td>
</tr>
<tr>
<td>2.0–4.0</td>
<td>1 unit</td>
</tr>
<tr>
<td>4.1–9.0</td>
<td>2 units</td>
</tr>
<tr>
<td>9.1–11.0</td>
<td>3 units</td>
</tr>
<tr>
<td>11.1–16.9</td>
<td>4 units</td>
</tr>
</tbody>
</table>

NOTE: for blood glucose, 1 mmol/litre = 18 mg/dL.

- If the glucose level is > 17 mmol/litre, expert advice should be sought.
- Aim for a glucose level of 4–9 mmol/litre.
- Reduce insulin by half at delivery, and aim to resume the pre-pregnancy insulin dosage 24 hours after delivery. If the mother is breastfeeding, her insulin requirement may be lower.
- Women who have developed gestational diabetes usually have normal blood glucose levels soon after the delivery of the placenta. Their diabetic medication should be stopped postnataally, and their blood sugar levels should be monitored.
- Mothers who have had gestational diabetes should have a glucose tolerance test at 6 weeks postnataally. They are at risk of developing type 2 diabetes, and appropriate dietary and lifestyle advice should be provided. A fasting blood glucose test annually should also be recommended.

Diabetic ketoacidosis (DKA)

DKA is the commonest endocrine emergency, and should be suspected in patients with any of the following:
- dehydration
- abdominal pain
- ketone smell on the breath
- acidosis
- acidic breathing
- unexplained coma.

Patients die from hypokalaemia and cerebral oedema. Patients who are 5% dehydrated, or who are vomiting or drowsy or clinically acidic, need emergency care as follows.

**Primary assessment and resuscitation**

**Airway**
- If the airway is not open, use an airway-opening manoeuvre, and consider an airway adjunct such as an oropharyngeal airway or intubation (if available and subsequently supported).
- The nares and oropharynx may need gentle suctioning under direct observation.
- If the patient is unconscious and the airway is unprotected, the recovery position should be adopted to minimise the risk of aspiration of vomit.

**Breathing**

Give a high concentration of oxygen through a face mask with a reservoir, if the airway is adequate.

If breathing is inadequate, ventilate with oxygen via a bag-valve-mask-reservoir device, and ask for experienced senior help to intubate (if this is available and sustainable).

**Circulation**

- Gain IV access using a short wide-bore cannula (14- to 16G).
- External or internal jugular vein access is an option if peripheral access is impossible. Long saphenous vein cut-down may also be considered.
- Take blood for a full blood count, urea and electrolytes, blood culture, cross-matching, glucose stick test and laboratory blood glucose (if available).
- Give a 500-mL rapid IV bolus of 0.9% saline or Plasma-Lyte 148.
- An antibiotic such as cefotaxime 1 gram IV 6-hourly, or the locally available equivalent, is an appropriate antibiotic for those in whom an infection is likely to have precipitated the DKA. Although, of course, antibiotic therapy must be tailored to the specific cause.

**Diagnosis**

- History:
  - polydipsia
  - polyuria
  - weight loss.
- Clinical:
  - acidic respiration
  - dehydration
  - drowsiness
  - abdominal pain and/or vomiting.
- Biochemical:
  - high blood glucose on finger-prick test
  - ketones and glucose in urine.

**Secondary assessment and emergency treatment**

The following in particular need to be assessed.

**Degree of dehydration**

- 3%: dehydration is only just clinically detectable
- 3–5%: dry mucous membranes and reduced skin turgor
Ileus
- Insert a nasogastric tube.
- Ensure by clinical assessment, and by abdominal X-ray if appropriate, that there is no other cause of the acute abdomen, including intestinal obstruction.

Cerebral oedema
Look for irritability, slow pulse, high blood pressure and papilloedema (a late sign).

Infection
DKA can cause a leucocytosis but not fever. If fever is present, look for and treat infection.

Ileus
- Insert a nasogastric tube.
- Look for abdominal distension, vomiting, and dehydration.

Additional emergency treatment
General
- After resuscitation with fluid boluses, calculate the fluid requirement (see below).
- Avoid excessive fluid replacement, as this is a risk factor for cerebral oedema.
- Do not give hypotonic IV solutions (e.g. 0.18% saline with 4% glucose, or 5% glucose): they are risk factors for cerebral oedema.
- Continue to give IV fluids until the patient is drinking.
- After fluids are running, calculate the rate of insulin infusion (blood glucose levels will already be falling).
- Use a continuous low-dose IV infusion of insulin (there is no need for an initial bolus), or in resource-limited situations use regular subcutaneous injections of short-acting insulin based on a sliding scale according to blood glucose measurements. Details below.
- Continue to give IV fluids until the patient is tolerating enteral fluids.

Fluid and electrolyte management
- Calculate the patient’s fluid requirement. This is equal to maintenance plus deficit (see Figure 2.7.D.1).
  - Maintenance
  - Deficit (litres) = percentage dehydration x body weight (kg)/100
  - Only plan to correct up to an 8% deficit, as any more risks over-infusion.
- Ignore the volume of fluids used to resuscitate/treat shock.
- Give the total fluid requirement over 24 hours:

Glucose > 12 mmol/litre: give 0.9% saline or Plasma-Lyte 148.
Glucose < 12 mmol/litre: give 0.9% saline or Plasma-Lyte 148 containing 5% dextrose (by adding 100 mL of 50% glucose to 900 mL of 0.9% saline or Plasma-Lyte 148).
Sodium 135–155 mmol/litre: correct by rehydration over 24 hours.
Sodium > 155 mmol/litre: correct by rehydration over 48 hours using 0.9% saline or Plasma-Lyte 148.
Expect the sodium level to rise initially as the glucose level falls and water is removed from the circulation.
If the plasma sodium level initially falls (as well as the glucose level), this may precipitate cerebral oedema.

Bicarbonate
- Administration of bicarbonate is rarely, if ever, necessary.
- Continuing acidosis usually indicates insufficient fluid resuscitation.
- Consider the use of bicarbonate in patients who are profoundly acidotic (pH < 7.0 if measurable) and shocked. Its only purpose is to improve cardiac contractility in severe shock.

The maximum volume of 8.4% sodium bicarbonate for half-correction of acidosis is calculated according to the following formula, and given over 60 minutes:

$$\text{Volume (mL) of 8.4% NaHCO}_3 = \frac{1/3 \times \text{weight (kg)} \times \text{base deficit (mmol/litre)}}{2}$$

If blood gas analysis cannot be undertaken:
- The kidneys will resolve the acidosis (if they are working) if the patient receives adequate fluid and insulin therapy.
- If you cannot measure pH, then do not give bicarbonate except in extremis.

Potassium
In diabetic ketoacidosis there is always massive depletion of total body potassium, although initial plasma levels may...
be low, normal or even high. Levels in the blood will fall once insulin is started.

Do not give potassium if any of the following are present:
- anuria
- peaked T waves on the ECG
- serum potassium level > 7.0 mmol/litre.

If biochemical assessment of the K⁺ is not possible, it should be assumed that K⁺ replacement is necessary as long as the urine output is adequate, and there are no peaked T-waves present on the ECG (where available).
- In resource-limited settings, hypokalaemia is most safely corrected enterally using ORS with or without additional oral potassium supplements (aim for a total of 100 mmol/day).
- Potassium rich foods may also be given, e.g. coconut milk and bananas.
- If oral supplementation is not possible or the patient is severely ill: start IV potassium supplements with 20 mmol/litre of IV fluid given after the start of initiating therapy with insulin and fluids as long as sufficient urine is being passed at > 30 mL/hour.
- Run the IV infusion (20 mmol in 1 litre over 4 to 8 hours (42 to 84 drops per minute (dpm) if using a standard IV giving set with a drop factor of 20). It should not be given at a rate exceeding 20 mmol in 2 hours (126 dpm) as this is dangerous. Given the difficulty in accurately

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**FIGURE 2.7.D.1** Pathway of care for severe diabetic ketoacidosis in pregnancy. AVPU scale: alert, responds to verbal stimulus, responds to pain, unresponsive; ORS, oral rehydration solution.
monitoring transfusion rates without an electronic pump, a large margin of error should be used. 

- Stop IV supplementation when the patient can take oral supplements.

**Insulin**

In resource-limited settings, give subcutaneous doses of short-acting soluble insulin 6-hourly at 0.6 units/kg/dose (i.e., 0.1 units/kg/hour). Give half the dose if the blood sugar level is falling too fast.

Always have an IV glucose solution (10% or 50%) available to treat any hypoglycaemia that develops.

- **In well-resourced settings**, make up a solution of 1 unit/mL of human soluble insulin (e.g., Actrapid) by adding 50 units of insulin to 50 mL of 0.9% saline or Plasma-Lyte 148 in a syringe pump. Using a Y-connector, attach this to the IV fluids that are already running. Do not add insulin directly to the fluid bags. The solution should then run at 0.1 units/kg/hour (0.1 mL/kg/hour).

- If the blood glucose level falls by more than 5 mmol/litre/hour, reduce the infusion rate to 0.05 units/kg/hour.

- If the blood glucose level is less than 12 mmol/litre, and a dextrose-containing fluid has been started, consider reducing the insulin infusion rate.

- Do not stop the insulin infusion while dextrose is being infused, as insulin is required to switch off ketone production.

- If the blood glucose level falls below 7 mmol/litre, consider adding extra glucose to the infusion.

- If the blood glucose level rises out of control, re-evaluate the patient for sepsis or another condition.

- Discontinue the insulin infusion 30 minutes after the first subcutaneous injection, to avoid rebound hyperglycaemia.

**Other management**

**Urine output**

- Urinary catheterisation may be useful in patients with impaired consciousness.

- Document all fluid input and output.

- Test all urine samples for glucose and ketones.

- If a massive diuresis continues, the fluid input may need to be increased.

**Gastric aspirate**

- If large volumes of gastric aspirate occur, replace these volume for volume with 0.9% saline or Plasma-Lyte 148 plus 5 mmol/litre potassium chloride (KCl).

**Biochemistry**

- Check urea and electrolytes, blood pH/bicarbonate (if available), and laboratory blood glucose 2 hours after the start of resuscitation, and then at least 4-hourly.

- Do not expect ketones to have disappeared completely before changing to subcutaneous insulin.

**Never give an IV insulin infusion without a syringe driver. This is not safe. It is better to use a sliding scale of subcutaneous rapid-acting insulin.**

**Cerebral oedema**

Cerebral oedema in DKA:

- is unpredictable

- occurs more often in new diabetics

- has a mortality of around 80%.

**Signs and symptoms**

These include the following:

- headache

- confusion

- irritability

- reduced conscious level

- fits

- small pupils

- increasing blood pressure

- slowing pulse

- possible respiratory impairment.

**Management**

- Exclude hypoglycaemia.

- Give 20 grams of 20% mannitol over 15 minutes as soon as cerebral oedema is suspected. Repeat every 4–6 hours.

- Restrict IV fluids to two-thirds maintenance, and replace the deficit over 72 hours rather than 24 hours.

- Arrange for the patient to be intubated. Keep the PaCO₂ in the range 3.5–5.0 kPa (if this is possible and sustainable).

- Keep the sodium (Na⁺) concentration higher than 135 mmol/litre.

- Keep the head in the midline and 30-degrees elevated.

If there is a fever, treat it actively with environmental measures, or with paracetamol, if more than 38.0°C.

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**2.7.E Reduced consciousness and coma**

**Introduction**

In resource-limited countries, severe pre-eclampsia, eclampsia, malaria, meningitis (including TB), HIV infection, head injury and drug ingestion are the most common causes of reduced conscious level and coma in pregnancy.

**Pathophysiology**

Raised intracranial pressure (RICP) is an important component of the most severe cases. This can occur gradually or rapidly (e.g., due to intracranial bleeding or cerebral oedema). The initial physiological compensating mechanisms include a reduction in the volume of cerebrospinal fluid and in the volume of venous blood within the cranium. However, when these fail, the cerebral perfusion pressure (CPP) falls and arterial blood flow to the brain is reduced.

Cerebral perfusion pressure (CPP) = mean arterial pressure (MAP) – intracranial pressure (ICP).

A severely increased pressure within the skull will
cause pressure effects which are classically recognised in two main sites where brain tissue is pushed against the bone:

1. **Central syndrome**: cerebellar tonsils herniate through the foramen magnum. This is known as **coning**. The syndrome consists of slowing pulse, rising blood pressure and irregular respiration.

2. **Uncal syndrome**: the uncus (part of the hippocampal gyrus) is pushed through the tentorium. It may be unilateral. This leads to third cranial nerve compression and ipsilateral dilated pupil, followed by oculomotor palsy and failure of lateral gaze. Later effects include hemiplegia.

**Raised intracranial pressure (RICP)**

In a patient with impaired conscious level or with a Glasgow Coma Scale score of < 9, who was previously well and is not post-ictal, the following signs indicate raised ICP:

<table>
<thead>
<tr>
<th>Absolute signs of raised ICP:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Papilloedema</td>
</tr>
<tr>
<td>Absence of pulsation of retinal vessels</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs suggesting raised ICP:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal oculocephalic reflexes</td>
</tr>
<tr>
<td>Do not test patients with neck injuries in this way</td>
</tr>
<tr>
<td>(a) Rotation of the head to the left or right normally causes the eyes to move in the opposite direction; abnormal if there is no response or a random response</td>
</tr>
<tr>
<td>(b) Flexure of neck usually causes eye gaze deviation upwards; abnormal if there is loss of this reflex</td>
</tr>
<tr>
<td>Abnormal posture May need to be elicited by a painful stimulus</td>
</tr>
<tr>
<td>(a) Decorticate: arms flexed, legs extended</td>
</tr>
<tr>
<td>(b) Decerebrate: arms extended, legs extended (see Figure 5.16.A.3)</td>
</tr>
<tr>
<td>Abnormal pupillary responses Unilateral or bilateral suggests raised ICP</td>
</tr>
<tr>
<td>Abnormal breathing patterns Ranges from hyperventilation to Cheyne–Stokes breathing to apnoea</td>
</tr>
<tr>
<td>Cushing’s triad Slow pulse, raised blood pressure and abnormal pattern of breathing – a late sign of raised ICP</td>
</tr>
</tbody>
</table>

**Primary assessment and resuscitation**

**ABC**

**Call for help. Ideally an anaesthetist should be present to manage the airway and support breathing.**

The first steps in the management of the patient with decreased conscious level are to assess and if necessary support airway, breathing and circulation.

**Airway**

The patient with a reduced level of consciousness is more likely to have a compromised airway as the tongue falls into the back of the mouth. There is also a risk of aspiration.

**Look, listen and feel**

Assess the airway, open it if closed and keep it open, either by assigning someone to continue airway-opening manoeuvres or by using adjuncts such as an oropharyngeal airway (see Section 1.13). Never use such an airway if the patient is conscious enough to have a gag reflex, as it may worsen airway obstruction and cause vomiting. Give oxygen at a rate of 15 litre/minute or as high a flow rate as is available, via a tight-fitting face mask with a reservoir bag. If an anaesthetist is present, intubation can be performed to protect the airway; otherwise adopt the recovery position (see Figure 2.7.E.1). Careful suction of the nose and/or mouth may be helpful.

**Assess and stabilise.**

**ABCD**

**Insecure airway**

**Inadequate breathing**

**URGENT**

Airway establishment and airway protection

Intubation and ventilation (if possible)

The patient will require support if:

- breathing is insufficient
- gag or cough reflex is absent
- GCS score is < 9, or AVPU score is P or U
- there is impending herniation due to raised ICP
- there is evidence of effects of inadequate breathing on other systems.

If the airway is adequate, give high concentration O₂ and support breathing if required.

**Breathing**

Assess the breathing for depth and frequency, and give high-flow oxygen via a face mask and reservoir bag. If breathing is absent or inadequate (gasping or agonal breaths only), provide assisted ventilation using a bag-valve-mask with a reservoir and oxygen.

Inadequate airway and breathing in coma can lead to a rise in arterial pCO₂ that can cause a dangerous rise in intracranial pressure.

**Circulation**

Inadequate perfusion of blood to the brain initially produces confusion and later causes coma. Measurement of the blood pressure in addition to other markers for shock is
crucial in recognising hypovolaemia after haemorrhage, or unconsciousness after an eclamptic fit with hypertension.

If the intracranial pressure is high, cerebral perfusion will be compromised if hypotension occurs. However, excessive fluid administration should be avoided.

- Establish IV access quickly.
- Take blood samples and send them to the lab for a full blood count, blood smear for malarial parasites, electrolytes, liver function tests, blood glucose and blood culture.

**Neurological failure**
Assess neurological failure as follows:

- Use the AVPU scale.
- Check blood glucose levels: If the blood sugar level is low or suspected to be low (< 2.5 mmol/litre or < 45 mg/dL), give 100 mL of 25% glucose IV over 15 minutes (dilute 50 mL of 50% glucose with 50 mL of Ringer-lactate or Hartmann’s solution) and then give 10% dextrose in Ringer-lactate or Hartmann’s solution over 4 hours (add 100 mL of 50% glucose to each 400 mL of Ringer-lactate or Hartmann’s solution infused).
- Check the pupils for signs suggesting raised intracranial pressure (RICP) or opiate overdose.
- Check for neck stiffness which may suggest meningitis.
- Look for other signs of raised intracranial pressure, as outlined above.

Further assessment of conscious level can be aided by the Glasgow Coma Scale score and documentation of pupil function.

### TABLE 2.7.E.1 Glasgow Coma Scale (GCS)

<table>
<thead>
<tr>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>To verbal stimuli</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>No response to pain</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td></td>
</tr>
<tr>
<td>Obey verbal command</td>
<td>6</td>
</tr>
<tr>
<td>Localises to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws from pain</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion to pain (decorticate)</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension to pain (decerebrate)</td>
<td>2</td>
</tr>
<tr>
<td>No response to pain</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Orientated and converses</td>
<td>5</td>
</tr>
<tr>
<td>Disorientated and converses</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>No response to pain</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
</tr>
</tbody>
</table>

A GCS score of < 9 is likely to need airway protection by intubation if skills are available to undertake this safely.

### TABLE 2.7.E.2 Pupillary changes

<table>
<thead>
<tr>
<th>Pupil size and reactivity</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small reactive pupils</td>
<td>Metabolic disorders</td>
</tr>
<tr>
<td></td>
<td>Medullary lesion</td>
</tr>
<tr>
<td>Pinpoint pupil</td>
<td>Metabolic disorders</td>
</tr>
<tr>
<td></td>
<td>Narcotic/organophosphate ingestion</td>
</tr>
<tr>
<td>Fixed mid-sized pupils</td>
<td>Midbrain lesion</td>
</tr>
<tr>
<td>Fixed dilated pupils</td>
<td>Hypothermia</td>
</tr>
<tr>
<td></td>
<td>Severe hypoxemic/ischaemic brain injury</td>
</tr>
<tr>
<td></td>
<td>Barbiturate ingestion (late sign)</td>
</tr>
<tr>
<td></td>
<td>During and post seizure</td>
</tr>
<tr>
<td></td>
<td>Anticholinergic drugs</td>
</tr>
<tr>
<td>Unilateral dilated pupil</td>
<td>Rapidly expanding ipsilateral lesion</td>
</tr>
<tr>
<td></td>
<td>Tentorial hemiation</td>
</tr>
<tr>
<td></td>
<td>Third cranial nerve lesion</td>
</tr>
<tr>
<td></td>
<td>Epileptic seizures</td>
</tr>
</tbody>
</table>

**Secondary assessment and emergency treatment**
Secondary assessment occurs after stabilisation of ABCD. During secondary assessment, continue to monitor the patient, and if there is any change, reassess ABC and treat any residual problems.

**Diagnostic pointers**
As soon as possible during resuscitation, gain as much information about the history as possible:

- the possibility of eclampsia, which means that magnesium sulphate may be required
- recent trauma
- endemic area for infections such as malaria, sleeping sickness and encephalitis
- pre-existing neurological problem
- past history of epilepsy
- ingestion of poisons
- underlying chronic condition (renal, cardiac, diabetes).

Remember to treat the treatable components. The cause of coma may not be certain, so it is always important to address ABC. If the patient’s condition is unstable or deteriorating, return to ABC.

Always consider the possibility of eclampsia and the need for magnesium sulphate.

If there is no other clear cause for the coma treat with antibiotics for presumed meningitis (usually a third-generation cephalexin, or whatever is locally available and appropriate), and in endemic areas, also treat as for cerebral malaria (see Section 2.8.D).

Take the patient’s temperature (core and peripheral).

- Fever may be associated with sepsis (but lack of fever does not exclude sepsis) or poisoning (ecstasy, cocaine or salicylates).
- Hypothermia is found in poisoning with ethanol or barbiturates.

Rash: purpura suggests meningococcal disease; bruises suggest trauma (consider domestic violence).
Evidence of poisoning, ingestion or drug use: smell, residue around nose/mouth, needle tracks.

Other issues in addition to ABC regarding the management of coma

The prognosis depends on the cause of coma and the state of the patient, in particular the level of consciousness on admission, and the initial response to appropriate interventions. The presumptive cause of coma guides the treatment. Consider the following interventions:

- Assess and maintain electrolyte balance (avoid hypotension; use Ringer-lactate or Hartmann’s solution plus added 5% glucose, not 1/5 N dextrose saline. Add 50 mL of 50% glucose to each 450 mL of Ringer-lactate or Hartmann’s solution infused). If possible keep the serum sodium level in the normal range (135–145 mmol/litre).
- Treat seizures if present, and give prophylactic anticonvulsants if the patient has repeated seizures.
- Insert a nasogastric tube to aspirate the stomach contents. Perform gastric lavage in circumstances such as drug ingestion.
- Regulate the body temperature, and avoid hyperthermia (i.e. temperatures above 37.5°C).
- Undertake appropriate medical management of RICP, if noted:
  - Support ventilation (maintain a pCO₂ of 3.5–5.0 kPa, if measurable).
  - Give mannitol, 20 grams of 20% mannitol IV over 15 minutes, 2-hourly as required, provided that the serum osmolality is not greater than 325 mOsm/litre (if measurable).
  - Give dexamethasone (for oedema surrounding a space-occupying lesion) 10 mg initially IV, then 4 mg IV 6-hourly for 48 hours.
- Catheterisation is needed for bladder care and output monitoring, as well as for avoidance of retention, which can worsen RICP.
- Plan for continued regular clinical assessment, mainly nursing observations.
- Prevent the patient from falling out of the bed.
- Provide nutritional support: parenteral and/or oral feeding to prevent malnutrition during the period of unconsciousness.
- Skin care: prevent bed sores by turning the patient.
- Eye padding: to avoid xerophthalmia.
- Family counselling, support and consent in the case of invasive procedures.
- Appropriate surgical intervention if indicated.
- Chest physiotherapy to avoid hypostatic pneumonia.
- Restrict fluids to 60% of maintenance if evidence of water retention is seen.
- Prevent deep vein thrombosis by physiotherapy.
- Maintain oral and dental hygiene.
- Give appropriate care for central and peripheral venous access to avoid infection by maintaining sterility when handling the sites.
- Prevent hospital-acquired infection.

Reassessment

When the patient is stable, undertake a full examination of systems and neurological examination.

- Skin: rash, bruising, haemorrhage, neurocutaneous stigmata.
- Scalp: trauma.
- Ears/nose: discharge (blood, serous fluid, infection).
- Neck: tenderness, stiffness/rigidity.
- Odour: poisoning, ingestion, metabolic disorders.
- Abdomen: liver, spleen.
- Eyes: pupils, fundi (papilloedema, retinal haemorrhages, sub-conjunctival haemorrhages), movements.

Also assess the following:

- AVPU and Glasgow Coma Scale scores: re-evaluate regularly.
- Posture/tone: lateralisation.
- Deep tendon reflexes: lateralisation.
- If there are lateralising signs, and if the patient is stable enough, consider a CT scan (if available);

The CT scan may show cerebral oedema, haemorrhages, or hypoxic/ischaemic encephalopathy.

Specific topics in coma

Meningitis or encephalitis

The following organisms cause meningitis:

- Neisseria meningitides:
  - risk of mortality (> 5%) and permanent serious neurological sequelae.
- Haemophilus pneumonia:
  - less common where routine Hib vaccination is available.
- Streptococcus pneumoniae:
  - common in resource-limited countries
  - occurs with underlying immune compromise, especially HIV infection

![Pathway of care in coma. ABC, airway, breathing and circulation; ICP, intracranial pressure.](figure.png)
Section 2.8

— may follow a head injury if there is damage to the dura and/or meninges.

There is a risk of coning and death if a diagnostic lumbar puncture is performed in a patient with significantly raised intracranial pressure.

**Diagnosis of meningitis or encephalitis**

Classic signs and symptoms include the following:
- headache
- vomiting
- neck stiffness
- opisthotonus
- photophobia
- rash
- altered consciousness.

Poisoning (see Section 7.4).
Malaria in pregnancy (see Section 2.8.D).
Eclamptic coma (see Section 2.5.E).

2.8 Infections complicating pregnancy, delivery and after birth

### 2.8.A Pneumonia

#### BOX 2.8.A.1 Minimum standards
- Oxygen.
- Antibiotics (IV and oral).
- Chest X-ray.

**Clinical findings**

A high fever is usually associated with pneumonia and bacterial tracheitis. In the absence of stridor and wheeze, breathing difficulties in association with a significant fever are likely to be due to pneumonia.

Examination of the chest may show reduced air entry, bronchial breathing and crepitations. Pleuritic chest pain, neck stiffness and abdominal pain may be present if there is pleural inflammation. Pleural effusions and empyema are complications.

Always consider HIV infection and TB.

**Emergency treatment of pneumonia**
- Give antibiotics for 7 days:
  - ampicillin 2 grams IV/IM 6-hourly plus gentamicin 80mg IV/IM 8-hourly or 5 mg/kg IV/IM every 24 hours for most cases of community-acquired pneumonia
  - cefuroxime 500mg IV/IM 8-hourly or flucloxacillin 500mg IM or IV slowly every 6 hours for suspected or bacteriologically diagnosed *Staphylococcus aureus*
  - erythromycin 500 mg every 6 hours orally for *Chlamydia or Mycoplasma pneumoniae*
  - or whatever is available locally and appropriate.
- Sit the patient upright.
- Maintain hydration.
  - Extra fluid may be needed to compensate for fluid loss from fever.
  - Fluid restriction may be needed because of inappropriate ADH secretion, revealed by oliguria < 30 mL per hour or rising blood urea levels.
- Chest X-ray is indicated.
- Large pleural effusions/empyemas should be diagnosed where possible by ultrasound, and pleural drainage undertaken under ultrasound cover (do not place a chest drain into the heart, liver or an undiagnosed tumour or hydatid cyst) (see Section 8.3). Remember that in advanced pregnancy the diaphragm is elevated.
  - Effusions/empyemas adjacent to the heart on the left side may cause pericarditis and cardiac arrhythmias. (Listen regularly for a pericardial rub, and ideally monitor an ECG if available until the patient is stable.)