Eclampsia
occurring in approximately 1:2000 pregnancies.

It may occur antepartum (40 per cent), intrapartum (20 per cent) or postpartum (40 per cent).
Eclampsia is obvious as a grand mal convulsion. However, other causes of fits such as epilepsy have to be considered.

Any convulsion in pregnancy should be considered to be eclamptic until proved otherwise.
**Symptoms**

- Frontal headache
- Visual disturbance (blurred vision and flashing lights)
- Epigastric pain
- General malaise and nausea
- Restlessness
• Agitation
• Hyper-reflexia and clonus
• Facial (especially periorbital) oedema
• Right upper quadrant tenderness
• Poor urine output
• Papilloedema
Management

- **Seizures due to eclampsia are usually self-limiting**
- Call for help and approach patient safely
- Turn her onto her side and try to stop her hurting herself during the fit
- **Most fits will stop spontaneously**
- Open airway + check breathing Check circulatio
- Place on her side
- Administer oxygen by face mask
- Auscultate chest (vs aspiration)
- Gain intravenous access and send off bloods for investigation
- Administer magnesium sulphate
- Control blood pressure
- Assess fetus
- Make plans for delivery **once stabilized**
An anaesthetist is needed urgently
- Open and protect airway
- Establish intravenous access
- 4g loading dose magnesium intravenously over 15 min
- This can be repeated (2 or 4 g depending on BMI)
If fits still persist

- Diazepam or thiopentone will be needed
- Both sedate and require an anaesthetist to intubate
- and protect the airway and to ventilate
- Blood pressure control should be established
- Once stable, if antenatal needs delivery by CS
- If fits recur general anaesthesia with muscle relaxation is then needed
The blood pressure should be reduced to safe levels.

- Antihypertensives can be
- either labetalol (can be given orally while intravenous access is obtained),
- oral nifedipine or intravenous hydralazine. If given intravenously, a bolus is initially used followed by an infusion that can be titrated to obtain a safe blood pressure.
- A gradual reduction of the blood pressure is optimal to precipitating fetal distress (in the form of a bradycardia) secondary to sudden drops in maternal blood pressure that reduce uterine blood flow.
Management of fluid balance can be problematic.

- In pre-eclampsia, there is intense peripheral vasoconstriction accompanied by a decrease in the plasma volume, together with redistribution of the extracellular fluid. The urine output falls, and overenthusiastic efforts to provide a fluid challenge may cause pulmonary and cerebral oedema.
In the absence of bleeding, no more than 80 mL/hour of fluids (oral and intravenous) should be given. Renal failure is uncommon and if it occurs is usually reversible.
If the gestation is less than 34 weeks, steroids should be given to improve lung maturity and decrease neonatal complications.

Delivery is often by Caesarean section, although if labour is well established, vaginal delivery is possible. If at all possible, clotting disorders must be corrected before delivery (by whatever means) is attempted.
HELPP syndrome – a combination of haemolysis, elevated liver enzymes and low platelets – is seen in 5–10 per cent of cases of severe pre-eclampsia. It is more common in multiparous women. It may be associated with disseminated intravascular coagulation,
- maternal mortality rate of 1.94 per 100,000 – more than twice that of the next most common cause (pre-eclampsia).
- Pregnancy is a hypercoagulable state because of an alteration in the thrombotic and fibrinolytic systems.
- There is an increase in clotting factors VIII, IX, X and fibrinogen levels, and a reduction in protein S and anti-thrombin (AT) III concentrations.
These physiological changes predispose a woman to thromboembolism and this is further exacerbated by venous stasis in the lower limbs due to the weight of the gravid uterus placing pressure on the inferior vena cava in late pregnancy and immobility,

Pregnancy is associated with a 6–10-fold increase in the risk of venous thromboembolic disease compared to the non-pregnant situation.
Risk factors for thromboembolic disease

Pre-existing
- Maternal age (35 years)
- Thrombophilia
- Obesity (80 kg)
- Previous thromboembolism
- Severe varicose veins
- Smoking
- Malignancy

Specific to pregnancy
- Multiple gestation
- Pre-eclampsia
- Grand multiparity
- Caesarean section, especially if emergency
- Damage to the pelvic veins
- Sepsis
- Prolonged bed rest
The major hereditary forms of thrombophilia currently recognized include: deficiencies of the endogenous anticoagulants protein C, protein S and AT III; abnormalities of procoagulant factors, factor V Leiden (caused by a mutation in the factor V gene) and the prothrombin mutation G20210A. It seems probable that there are still some thrombophilias not yet discovered or described. Heritable thrombophilias are present in at least 15 per cent of Western populations.
Acquired thrombophilia is most commonly associated with antiphospholipid syndrome (APS). APS is the combination of lupus anticoagulant with or without anti-cardiolipin antibodies, with a history of recurrent miscarriage and/or thrombosis. It may (or, more commonly, may not) be associated with other autoantibody disorders, such as systemic lupuerythematosus (SLE).
more than 50 per cent of women with pregnancy related VTE will have a thrombophilia. It is therefore vital that women with a history of thrombotic events are screened for thrombophilia. The
Deep vein thrombosis

- pain in the calf with varying degrees of redness or swelling.
- unilateral leg odema symptoms should ring alarm bells.
- calf is tender to gentle touch
- Compression ultrasound has a high sensitivity and specificity in diagnosing proximal thrombosis
- Venography is invasive, requiring the injection of contrast medium and the use of x-rays. It does, however, allow excellent visualization of veins both below and above the knee.
Pulmonary embolus

- Presentation is of mild breathlessness, or inspiratory chest pain
- Tachycardic (90 bpm) with a mild pyrexia (37.5°C)
- Rarely, massive PE may present with sudden cardiorespiratory collapse
If PE is suspected

- initial electrocardiogram (ECG),
- chest x-ray and arterial blood gases should be performed to exclude other respiratory diagnoses
- ultrasound and if positive treat with a presumptive diagnosis of PE.
- ventilation perfusion (V/Q) scan or computed tomography pulmonary angiogram (CTPA should be performed. In both cases the radiation to the fetus is below the threshold considered safe.
- d-dimer is now commonly used as a screening test for thromboembolic disease in non-pregnant women,
Warfarin is given orally and prolongs the prothrombin time (PT). Warfarin is rarely recommended for use in pregnancy (exceptions include women with mechanical heart valves) as it crosses the placenta and can cause limb and facial defects in the first trimester and fetal intracerebral haemorrhage in the second and third trimesters.
Low molecular weight heparins (LMWHs) are now the treatment of choice. They do not cross the placenta and have been shown to be at least as safe and effective as unfractionated heparin (UFH) lower and fewer haemorrhagic complications Safe and easy to administer.
Following delivery, women can choose to convert to warfarin (with the need for stabilization of the doses initially and frequent checks of the international normalized ratio (INR) or remain on LMWH. Both warfarin and LMWH are safe in women who are breastfeeding.

Graduated elastic stockings should be used for the initial treatment of DVT and should be worn for two years following a DVT to prevent post phlebitic syndrome.
Antenatal thromboprophylaxis risk assessment and management

High risk

- Single previous VTE+
  - Thrombophilia or family history
  - Unprovoked/oestrogen-related previous
- recurrent VTE (>1)

High risk
Requires antenatal prophylaxis with LMWH
Refer to trust-nominated thrombosis in pregnancy expert/team
Intermediate risk

- Single previous VTE with no family history or thrombophilia
- Thrombophilia + no VTE
- Surgical procedure, e.g. appendicectomy
- Medical comorbidities, e.g. heart or lung disease, SLE, cancer, inflammatory conditions, nephrotic syndrome, sickle cell disease,
- intravenous drug user

Consider antenatal prophylaxis with LMWH
Seek trust-nominated thrombosis in pregnancy expert/team advice
Lower risk

- Age >35 years
- Obesity (BMI >30 kg/m2)
- Parity ≥3
- Smoker
- Pre-eclampsia
- Dehydration/hyperemesis/OHSS
- Multiple pregnancy or ART
- Gross varicose veins
- Current systemic infection
- Immobility, e.g. paraplegia, SPD, long-distance travel

Mobilization and avoidance of dehydration
Postnatal thromboprophylaxis risk assessment and management

High risk

Any previous VTE+
Anyone requiring antenatal LMWH

At least 6 weeks postnatal prophylactic LMWH
Intermediate risk

- Caesarean section in labour
- Asymptomatic thrombophilia (inherited or acquired)
- Medical comorbidities, e.g. heart or lung disease, SLE, cancer, inflammatory conditions,
- nephrotic syndrome, sickle cell disease,
- intravenous drug user
- BMI >40 kg/m²
- Prolonged hospital admission

At least 7 days postnatal prophylactic LMWH
Lower risk

- Age >35 years
- Obesity (BMI >30 kg/m²)
- Parity ≥ 3
- Smoker
- Elective Caesarean section
- Any surgical elective in puerperium
- Pre-eclampsia
- Mid-cavity rotational operative delivery
- Prolonged labour (>24 hours)
- PPH >1 litre or blood transfusion
- Gross varicose veins
- Current systemic infection
- Immobility, e.g. paraplegia, SPD, long-distance travel

Mobilization and avoidance of dehydration