Obstetrics

Acute fatty liver of pregnancy

DEFINITION Rare pregnancy-associated disorder characterised by fatty infiltration of the liver.

AETIOLOGY Likely to be due to a mitochondrial disorder affecting fatty acid oxidation.

ASSOCIATIONS/RISK FACTORS Nulliparity, multiple pregnancy, obesity, male fetus, pre-eclampsia.

EPIDEMIOLOGY UK prevalence estimated at 5 per 100 000.

HISTORY Often non-specific, normally in third trimester: nausea, vomiting, abdominal pain, jaundice, bleeding.

EXAMINATION Liver tenderness, jaundice, ascites, manifestations of coagulopathy. Fifty percent of women will have proteinuric hypertension.

PATHOLOGY/PATHOGENESIS Accumulation of microvesicular fat in haepatocytes, periportal sparing, small yellow liver on gross examination.

INVESTIGATIONS

Bloods: FBC (assess Hb, haemoconcentration, thrombocytopenia), clotting (↓ synthesis + consumption of clotting factors), LFT (↑ transaminases, mild hyperbilirubinaemia), U&E, glucose (hypoglycaemia common).

MANAGEMENT Delivery is necessary to halt deterioration. Treatment is supportive: fluid management; correction of hypoglycaemia; blood transfusion as appropriate; correction of coagulopathy with platelets/FFP/cryoprecipitate. Liver transplantation is rarely necessary.

COMPLICATIONS

Maternal: Death, haemorrhage (secondary to DIC), renal failure, hepatic encephalopathy, sepsis, pancreatitis.
Fetal: Death.

PROGNOSIS Maternal mortality 10–20%; perinatal mortality 20–30%.

Amniotic fluid embolism

DEFINITION Obstetric emergency in which amniotic fluid and fetal cells enter the maternal circulation causing cardiorespiratory collapse.

AETIOLOGY Unclear. Entry of amniotic fluid or fetal debris to the maternal circulation provokes either an anaphylactoid reaction or activation of the complement cascade.

ASSOCIATIONS/RISK FACTORS Often occurs in the absence of identifiable risk factors. Multiparity, ↑ maternal age, Caesarean section, uterine hyperstimulation, use of uterotonics, placental abruption, trauma, termination of pregnancy.

EPIDEMIOLOGY UK prevalence is 1.8 per 100 000 maternities.

HISTORY Sudden-onset dyspnoea ± chest pain, ?collapse.

EXAMINATION Tachypneoa, cyanosis, hypotension, tachycardia, evidence of coagulopathy.

PATHOLOGY/PATHOGENESIS The precipitating reaction causes pulmonary artery spasm, ↑ pulmonary arterial pressure and ↑ right ventricular pressure, resulting in hypoxia. Hypoxia leads to myocardial and pulmonary capillary damage and LVF. Post mortem reveals fetal squames and debris in the maternal pulmonary circulation.

INVESTIGATIONS
Bloods: ABG, FBC, clotting, U&E, X-match.
Imaging: CXR.
Other: ECG.

MANAGEMENT Largely supportive; manage in ITU.
Airway: Maintain patency.
Breathing: High-flow oxygen ± intubation.
Circulation: Two large-bore IV cannulae, fluid resuscitation, consider pulmonary artery catheter and ionotropic support, correct coagulopathy with FFP/cryoprecipitate/platelets, blood transfusion if necessary.

Consider delivery.

COMPLICATIONS Cardiac arrest, death, DIC, seizures, uterine atony and haemorrhage, pulmonary oedema, ARDS, renal failure.

PROGNOSIS In the UK: 37% mortality, of which 25% occurs within the first hour.

Cardiac disease in pregnancy

DEFINITION Cardiac disease in a pregnant woman.

AETIOLOGY

Congenital heart disease: PDA, ASD, VSD, coarctation of the aorta, Marfan's, Fallot's tetralogy, Eisenmenger's syndrome.

Acquired heart disease: Valvular defects, ischaemic heart disease.

Cardomyopathies: Including peripartum cardiomyopathy (new-onset cardiomyopathy and heart failure usually within time period of last month of pregnancy and 5 months post-partum).

ASSOCIATIONS/RISK FACTORS As for cardiac disease in general: family history, obesity, hypertension, smoking, ↑ age, diabetes.

EPIDEMIOLOGY Increasing prevalence due to ↑ maternal age, ↑ life expectancy for patients with congenital heart disease, ↑ immigrant populations.

HISTORY Assess new/deterioration of symptoms: SOB, palpitations, orthopnoea, paroxysmal nocturnal dyspnoea, decreased exercise tolerance, chest pain.

EXAMINATION

General: Pulse, BP, JVP, ?oedema, ?cyanosis.

Chest: Heart sounds, murmurs (*note:* ejection systolic common in pregnancy), basal crepitations.

Abdomen: Fundal height (associated with IUGR).

PATHOLOGY/PATHOGENESIS Dependent on aetiologies noted above. There is 40% increase in blood volume during pregnancy, hence cardiac strain. Women with cardiac disease are unable to increase cardiac output (uterine hypoperfusion, ↑risk of pulmonary oedema).

INVESTIGATIONS

Bloods: FBC, U&E, LFT.

Cardiac: ECG, echo.

Fetus: Serial USS for fetal growth, cardiac anomaly scan if there is maternal congenital heart disease.

MANAGEMENT Dependent on the aetiology.

General: Combined obstetric/cardiology care (tertiary referral centre).

Preconceptual: Assess cardiac status, address risks and absolute contraindications to pregnancy.

Antenatal: Optimise treatment, monitor fetal wellbeing, consider thromboprophylaxis.

Delivery: Consider optimum mode and timing of delivery, consult with anaesthetists, correct positioning, fluid management, consider antibiotic prophylaxis (structural defects).

Post-partum: Increase surveillance (period of haemodynamic change).

COMPLICATIONS

Maternal: Progression of disease, VTE, pulmonary oedema, death.

Fetal: PTL, ↑ congenital heart disease (if maternal congenital heart disease), IUGR, effects of teratogenic drugs for anticoagulation, fetal death.

PROGNOSIS High risk of maternal mortality if LVEF <40% or LVF. Pulmonary hypertension: 20–50% maternal mortality rate. Eisenmenger's: 50% maternal mortality rate. Patients with Marfan's syndrome with aortic root >4–4.5 cm are advised against pregnancy.

Chronic hypertension in pregnancy

DEFINITION Hypertension that is either present prior to conception (detected before 20/40) or persists after pregnancy.

AETIOLOGY Essential hypertension in >90–95% (cause unknown). Remainder are secondary to: *endocrine* (Cushing's, phaeochromocytoma, CAH), *renal* (renal artery stenosis, chronic renal disease), *vascular* (e.g. coarctation of aorta).

ASSOCIATIONS/RISK FACTORS Increasing age, ethnicity (Afro-Caribbean), obesity, smoking, diabetes, family history, pre-eclampsia.

EPIDEMIOLOGY Affects 1–5% of pregnancies.

HISTORY Largely asymptomatic.

EXAMINATION Blood pressure may be normal in first trimester due to ↓ systemic vascular resistance. Secondary causes include renal bruits, radiofemoral delay.

PATHOLOGY/PATHOGENESIS Chronic systemic inflammatory response increases susceptibility to pre-eclampsia. Placental pathology similar to pre-eclampsia (arterial occlusive changes, excess villous syncytial knots, infarction etc.) leads to hypoperfusion of the maternal space.

INVESTIGATIONS

Bloods: FBC, U&E, LFT, Urate.
Urinalysis: For proteinuria. If secondary causes are suspected: urinary catecholamines, renal artery USS and so on.
Fetus: Serial USS for fetal growth.

MANAGEMENT

Medication: Convert to non-teratogenic medications: methyldopa, nifedipine, labetalol (*note:* ACE inhibitors are teratogenic), aspirin 75 mg od (↓ risk of pre-eclampsia/IUGR).
Other: Monitor for pre-eclampsia, serial USS for fetal growth, uterine artery Dopplers at 24/40 (predicts pre-eclampsia).

COMPLICATIONS IUGR, superimposed pre-eclampsia (25%), placental abruption, prematurity.

PROGNOSIS Raised maternal and perinatal morbidity is related to superimposed pre-eclampsia.

Cord prolapse

DEFINITION Descent of the umbilical cord through the cervix past the presenting part in the presence of ruptured membranes: an *obstetric emergency*.

AETIOLOGY Potential space (e.g. unengaged presenting part) allows descent of the cord past the presenting part.

ASSOCIATIONS/RISK FACTORS Breech presentation, abnormal lie, multiple pregnancy (second twin), prematurity, low birthweight, unengaged presenting part, polyhydramnios, ARM.

EPIDEMIOLOGY Affects 0.1–0.6% of pregnancies.

HISTORY An abnormal FHR is detected, often after membrane rupture.

EXAMINATION Cord is felt or seen through the cervix below the presenting part.

PATHOLOGY/PATHOGENESIS Compression of the cord by the presenting part and arterial spasm prevents blood flow through the cord, causing asphyxia.

INVESTIGATIONS Unnecessary.

MANAGEMENT Place patient in Trendelenberg or knee–chest position. Manually elevate the presenting part (this may also be achieved through bladder filling). Emergency delivery is necessary, usually via Caesarean section. The neonatal team should be present at delivery.

COMPLICATIONS Hypoxic–ischaemic encephalopathy, fetal death.

PROGNOSIS Perinatal mortality rate is 91 per 1000, higher if it occurs outside hospital.

Diabetes in pregnancy

DEFINITION Pre-existing or new-onset diabetes in pregnancy.

AETIOLOGY

Pre-existing: Type 1 – failure of pancreas to produce insulin; type 2 – relative insulin deficiency associated with increased peripheral insulin resistance.
Gestational diabetes (GDM): Altered glucose tolerance in pregnancy.

ASSOCIATIONS/RISK FACTORS

GDM: ↑ maternal age, ethnicity (South Asian, Middle Eastern, Afro-Caribbean), obesity, smoking, PCOS, family history, previous macrosomic baby.

EPIDEMIOLOGY Prevalence is 2–5% but estimates vary. GDM accounts for 90% of diabetes in pregnancy.

HISTORY

Pre-existing: Usually known to mother.
GDM: Usually asymptomatic, detected on screening.

EXAMINATION

Abdomen: Fundal height (macrosomia/polyhydramnios).

PATHOLOGY/PATHOGENESIS

Type I: Autoimmune destruction of pancreatic islet cells.
Type II: Genetic component + influence of age and obesity on peripheral insulin resistance.
GDM: ↑ insulin resistance in pregnancy (↑ secretion of insulin antagonists, including HPL, glucagon and cortisol), altered carbohydrate metabolism, failure of normal pregnancy increase in insulin production.
Fetus: Hyperglycaemia in early pregnancy may affect development (congenital abnormalities); hyperglycaemia causes fetal hyperinsulinaemia and macrosomia.
Neonatal: Hypoglycaemia can occur (withdrawal of maternal glucose while fetal insulin levels remain high).

INVESTIGATIONS

Delivery: Aim for delivery after 38 completed weeks of pregnancy. Sliding scale in labour.
Pre-existing: Detailed anomaly scan, fetal cardiac scan, ophthalmic examination.
GDM: Screening (universal or selective) by GTT at 26–28/40.

MANAGEMENT

Pre-existing

Preconceptual: Optimisation of glucose control.
Medical: Optimise diet, consider converting oral hypoglycaemics to insulin. Likely to require increasing doses of insulin.
Pregnancy: Capillary blood glucose monitoring, monitor for pre-eclampsia, serial USS for fetal growth.
Delivery: Sliding scale in labour.
Postpartum: Return to pre-pregnancy doses of medications.

GDM

Medical: Diet control. Persistent hyperglycaemia may require insulin treatment.
Pregnancy/delivery: As for pre-existing.
Postpartum: Stop insulin after delivery, fasting blood glucose 6/52 postpartum.

COMPLICATIONS

Maternal: Progression of pre-existing nephropathy/neuropathy/retinopathy, miscarriage, pre-eclampsia, operative delivery.

Diabetes in pregnancy (continued)

Fetal/neonatal: Congenital abnormalities (pre-existing only), fetal death, polyhydramnios, polycythaemia, macrosomia (+ traumatic delivery), respiratory distress syndrome, neonatal hypoglycaemia, neonatal jaundice.

PROGNOSIS Dependent on adequacy of control.

GDM: 70% recurrence in future pregnancies, ↑ risk (40–60%) for type 2 diabetes.

Eclampsia

DEFINITION Grand mal seizures on a background of pre-eclampsia.

AETIOLOGY Unclear: see pathology/pathogenesis.

ASSOCIATIONS/RISK FACTORS Pre-existing pre-eclampsia.

EPIDEMIOLOGY UK incidence is 4.9 per 10 000 maternities, 44% postpartum, 18% intrapartum and 38% antenatal.

HISTORY Symptoms of impending eclampsia: headache, epigastric tenderness, visual disturbance, oedema, previous examination findings of hyperreflexia, clonus.

EXAMINATION Grand mal seizure.

PATHOLOGY/PATHOGENESIS Unclear. Mechanism related to cerebral vasospasm, hypertensive encephalopathy, tissue oedema, ischaemia and haemorrhage have been proposed.

INVESTIGATIONS

Bloods: (As for pre-eclampsia) FBC, clotting, U&E, urate, LFT, G&S, consider ABG.
Urine: ? proteinuria.
Imaging (post-seizure): CT head, CXR if chest signs.

MANAGEMENT

Airway and breathing: Apply oxygen, maintain patency, ventilation if appropriate.
Circulation: Manage on left tilt, ensure large-bore IV access, evaluate pulse and blood pressure.
Drugs: IV magnesium sulphate: 4 g loading dose followed by 1 g/h (monitor urine output, respiratory rate, patellar reflexes).
Recurrent seizures: Consider – further bolus magnesium sulphate, thiopentone, diazepam, IPPV and muscle relaxation.
Post-seizure: Assess chest, control blood pressure (consider IV labetalol/hydralazine), strict fluid management, (85 mL/h input, urine output >25 mL/h), may require CVP monitoring, deliver baby (only when mother stabilised), consider ITU.

COMPLICATIONS Cardiac arrest, death, permanent CNS damage (e.g. cortical blindness), CVA (1–2%), DIC, renal failure, ARDS.

PROGNOSIS Maternal mortality rate 1.8%; significant morbidity in 35%.

Epilepsy in pregnancy

DEFINITION A continuing tendency to have seizures.

AETIOLOGY Usually idiopathic.

ASSOCIATIONS/RISK FACTORS Family history, intracranial mass, previous intracranial surgery, head injury, cerebrovascular disease, CNS infections.

EPIDEMIOLOGY Affects 0.5% women of childbearing age.

HISTORY Known personal history of epilepsy.

EXAMINATION Often unremarkable.

PATHOLOGY/PATHOGENESIS

Seizure: Paroxysmal discharge of cerebral neurones results in convulsions.
Seizure types: Primary generalised, partial (focal) or complex partial, temporal lobe.
Altered seizure frequency in pregnancy: Related to ↑ renal and hepatic drug clearance, ↑ volume of distribution, ↓ absorption (e.g. during labour, nausea and vomiting), compliance issues (fear of congenital abnormalities).

INVESTIGATIONS

Bloods: (Effects of anticonvulsants) FBC (↑MCV), serum folate, serum anticonvulsant levels, LFT.
Fetus: Detailed fetal anomaly scan ± fetal echo.

MANAGEMENT

Preconceptual: Maximise control on least teratogenic monotherapy if possible, folic acid 5 mg od.
Medication: Remains the same (benefits outweigh risks of changing) if well controlled with phenytoin, carbamazepine, lamotrigine, valproate, phenobaritone or levetiracetam (may require ↑ doses and may need to monitor drug levels). If diagnosed in pregnancy, lamotrigine and carbamazepine are drugs of choice. Vitamin K from 36/40 if enzyme-inducing drugs taken.
Delivery: Continue medications, may require diazepam/lorazepam if seizures during labour.
Postnatal: IM neonatal vitamin K. Gradually reduce doses of any medications increased in pregnancy to baseline.

COMPLICATIONS

Maternal: Change in seizure frequency, causes around 5 maternal deaths a year in UK.
Fetal: Teratogenicity related to antiepileptic drugs – congenital abnormalities (orofacial, neural tube, congenital heart), ↑ risk childhood epilepsy, haemorrhagic disease of the newborn (if enzyme-inducing drugs taken).

PROGNOSIS Poorly controlled/multiple seizure types more likely to deteriorate in pregnancy. Long-term seizure-free patients unlikely to experience an increase in seizures. Highest risk is peri-partum.

Fetal distress in labour

DEFINITION Fetal hypoxia in labour, ± acidosis. Diagnosis is by intermittent auscultation, continuous CTG or FBS.

AETIOLOGY Insufficient oxygen transfer from mother to fetus results in an asphyxial insult.

ASSOCIATIONS/RISK FACTORS Hypertonic uterus, placental insufficiency, intrauterine infection, cord prolapse, placental abruption, IUGR, oligohydramnios, post-dates pregnancy, hypertensive disease.

EPIDEMIOLOGY UK incidence: 2–10% (*note:* diagnosis of fetal distress necessitates delivery).

HISTORY Detected via monitoring, ?features of underlying condition (e.g. hypertension, abruption).

EXAMINATION

General: Assess maternal pulse (maternal tachycardia can cause fetal tachycardia), BP (maternal hypovolaemia), temperature (signs of infection/chorioamnionitis).
Abdomen: Check for hypertonic/irritable uterus (hyperstimulation, abruption), clinical assessment of fetal growth/liquor (IUGR, oligohydramnios).
Vaginal: Assess cervical dilatation, colour of liquor, bleeding, check for cord prolapse.

PATHOLOGY/PATHOGENESIS Impairment of transfer of O_2 and CO_2 between mother and fetus (e.g. due to placental insufficiency/pathology, fetal conditions resulting in reduced oxygen-carrying capacity such as anaemia, infection) leads to hypoxia. Excess CO_2 is converted to carbonic acid, causing a respiratory acidosis and leading to a fall in pH. Buffering system exists to attempt to maintain pH. Can recover if oxygenation re-established. If insult is prolonged, lactate accumulates leading to metabolic acidosis – pH falls and base excess rises.

INVESTIGATIONS

Bloods: FBC, G&S (preparation for delivery).
CTG
FBS: pH >7.25 is reassuring unless CTG continues to deteriorate, <7.25 repeat at 30 mins (*note:* <7.2 requires delivery).

MANAGEMENT

General: Left tilt, IV access, stop oxytocin infusion if in progress.
Fetal bradycardia >6 minutes: Necessitates emergency delivery.
Persistent CTG abnormalities: (See *Cardiotocography* appendix) may require FBS.
Delivery: If fully dilated, <1/5 palpable abdominally and vertex at or below spines, consider instrumental delivery. Otherwise deliver by Caesarean section.

COMPLICATIONS

Maternal: Complications of operative delivery.
Fetal: Hypoxic ischaemic encephalopathy, death.

PROGNOSIS Depends on underlying cause and timeliness of intervention.

Group B streptococcal infection

DEFINITION Group B *Streptococcus* (GBS) is the most common cause of early-onset (<7 days) infection in neonates.

AETIOLOGY Commensal bacterium of the vagina and rectum, carried by 25% of women. Of the babies who come into contact with GBS during labour the majority will not be affected. Some will become colonised. A minority become seriously ill, often within the first 12 hours after delivery.

ASSOCIATIONS/RISK FACTORS Positive HVS/LVS/rectal swab/MSU, previously affected baby, pyrexia in labour, prolonged rupture of membranes, PTL.

EPIDEMIOLOGY Most frequent cause of early-onset neonatal infection. Incidence is 0.5 per 1000 births in UK.

HISTORY Often asymptomatic. Detected on MSU, HVS or LVS. ?Prolonged rupture of membranes. ?PTL.

EXAMINATION Usually unremarkable.

PATHOLOGY/PATHOGENESIS Gram-positive *Streptococcus* characterised by the presence of Group B Lancefield antigen. Also known as *Streptococcus agalactiae*.

INVESTIGATIONS

Microbiology: HVS, LVS, rectal swab for MC&S. There is no national screening programme for GBS in the UK (no clear evidence of benefit, and concerns about antibiotic resistance/anaphylaxis).

MANAGEMENT IV antibiotics in labour (benzylpenicillin, clindamycin if penicllin-allergic). If detected antenatally on MSU, treat with antibiotics.

COMPLICATIONS

Neonatal: Septicaemia, pneumonia, meningitis, death.

PROGNOSIS Treatment is 80% effective in preventing early-onset GBS infection. Mortality is 10% in early-onset neonatal infection.

HIV in pregnancy

DEFINITION Human immunodeficiency virus – a retrovirus that attacks T lymphocytes.

AETIOLOGY HIV is present in vaginal fluid, semen, blood and breast milk of infected patients. Transmission: sexual contact, blood-borne, vertical.

ASSOCIATIONS/RISK FACTORS Increased risk of vertical transmission with ↑ viral load, ↓ CD4 count, prolonged rupture of membranes (>4 hours), breastfeeding.

EPIDEMIOLOGY Increasingly common due to increased life expectancy in individuals with HIV infection. Prevalence 0.38% in London, 0.06% in rest of UK.

HISTORY May be asymptomatic until progression to AIDS (average 8–10 years). ?Febrile seroconversion illness.

EXAMINATION No clinical features in HIV. May present with opportunistic infection, or rarely AIDS-defining illness (e.g. *Pneumocystis carinii* pneumonia (PCP), Kaposi's sarcoma, oesophageal candidiasis).

PATHOLOGY/PATHOGENESIS In women who do not breastfeed, over 80% vertical transmission occurs late in the third trimester (>36 weeks) and at delivery. Less than 2% occurs in the first two trimesters.

INVESTIGATIONS

Bloods: Routine HIV testing at antenatal booking, regular viral load and CD4 count.
Monitoring for drug toxicity: FBC, U&E, LFT, lactate and blood glucose.

MANAGEMENT

Antenatal: HAART (a combination of at least three antiretrovirals) if CD4 count $>350 \times 10^6$/L. Aim to suppress viral load to undetectable levels (<50 copies/mL). If CD4 count and viral load acceptable, can start antiretrovirals from 28 to 32 weeks.
Intrapartum: If viral load detectable or non-compliant with HAART, advise Caesarean section. IV zidovudine infusion given from 4 h prior to delivery until cord is clamped.
Viral load undetectable: Consider vaginal delivery, avoid FBS/FSE or rupture of membranes for >4 h.
Neonatal: Antiretrovirals for 4–6 weeks, PCR testing at birth, 3 weeks, 6 weeks and 6 months, HIV antibody test at 18 months. Avoid breastfeeding.

COMPLICATIONS Side-effects of HAART – pre-eclampsia, obstetric cholestasis and other liver dysfunction, lactic acidosis, glucose intolerance, GDM.

PROGNOSIS No evidence to suggest that pregnancy accelerates progression to AIDS. Appropriate measures as above reduce transmission rate from 28% to <2%.

Infections in pregnancy – *chicken pox*

DEFINITION Primary infection caused by the varicella zoster virus.

AETIOLOGY Transmission by physical contact, aerosol route, vertical.

ASSOCIATIONS/RISK FACTORS No prior immunity, immigrant population from tropical or subtropical areas (*note:* can be transmitted via patients with shingles).

EPIDEMIOLOGY Complicates 3 per 1000 pregnancies, 90% immunity in women in the UK.

HISTORY Fever, malaise, pruritic rash (becomes vesicular then crusts over).

EXAMINATION Vesicular rash.

PATHOLOGY/PATHOGENESIS DNA virus (herpes family), highly infectious.

Spread: Aerosol route, direct contact with vesicular fluid, indirectly via fomites.

Incubation period: 1–3 weeks, infectious from 48 hours prior to the rash forming until vesicles crust over.

Dormant period: Following primary infection the virus lies dormant in sensory nerve root ganglia and may reactivate as shingles.

INVESTIGATIONS

Bloods: Varicella zoster IgM (active infection) or IgG (immunity).

USS: Fetal anomaly scan (fetal varicella syndrome).

MANAGEMENT

Non-immune mother: Give VZIG.

Established chicken pox: Aciclovir (800 mg 5 × daily for 7 days), if within 24 h of onset of rash (caution prior to 20 weeks).

Maternal infection near term: Avoid elective delivery for 5–7 days after rash appears (allows placental transfer of maternal antibodies).

Neonate: Requires VZIG if delivered within 7 days before or after onset of maternal rash.

COMPLICATIONS

Maternal: Pneumonia (10%), hepatitis, encephalitis, death (rare).

Fetal: Fetal varicella syndrome (if maternal infection before 28/40): skin scarring, eye defects, limb deformities, neurological abnormalities.

Neonatal: Varicella infection of the newborn (if maternal infection 1–4 weeks prior to delivery to 1 week postpartum).

PROGNOSIS After exposure, VZIG reduces risk of maternal infection to 50% in the non-immune. No increased risk of miscarriage.

Infections in pregnancy – *cytomegalovirus*

DEFINITION Common viral infection, associated with a severe congenital syndrome in the fetus.

AETIOLOGY

Transmission: Sexual contact, blood-borne, contact with infected bodily fluids (saliva or urine), vertical.

ASSOCIATIONS/RISK FACTORS Higher socioeconomic class (less likely to have immunity through childhood infection), immunosuppression (e.g. HIV).

EPIDEMIOLOGY There is 50% immunity in pregnant women. 1% of seronegative pregnant women will contract CMV antenatally.

HISTORY Often asymptomatic, ?fever, malaise, fatigue.

EXAMINATION Often no clinical signs, ?lymphadenopathy.

PATHOLOGY/PATHOGENESIS DNA virus (herpes family). Following primary infection can remain dormant and then reactivate (e.g. in immunosuppression). Incubation period 1–2 months.

INVESTIGATIONS

Bloods: CMV IgM (current infection)/IgG (immunity).
USS: Fetal anomaly scan.
Other: Amniocentesis for CMV PCR (6–9 weeks after primary infection).

MANAGEMENT No treatment to prevent transmission to fetus. May offer termination of pregnancy if evidence of CNS damage. Neonatal gancyclovir can attenuate audiological complications.

COMPLICATIONS Increased risk of miscarriage and stillbirth, congenital CMV (IUGR, microcephaly, intracerebral calcification, blindness, sensori-neural deafness, hepatosplenomegaly, skin rash, pneumonitis, mental retardation).

PROGNOSIS Rate of transmission to the fetus is 40%. Of these, 10% will develop the congenital syndrome. Ninety percent of babies who are symptomatic at birth will have later neuro-developmental problems.

Infections in pregnancy – *hepatitis B*

DEFINITION Infection caused by the hepatitis B virus.

AETIOLOGY Transmission by sexual contact, blood-borne, vertical.

ASSOCIATIONS/RISK FACTORS Multiple sexual partners, unprotected sexual intercourse, intravenous drug use, ↑ prevalence in South-East Asian immigrant population.

EPIDEMIOLOGY Affects 1 per 100 000 pregnancies.

HISTORY Fever, myalgia, nausea, vomiting, jaundice, abdominal pain (*note:* asymptomatic in 70%).

EXAMINATION Jaundice, hepatomegaly, right upper quadrant tenderness.

PATHOLOGY/PATHOGENESIS Double-stranded DNA virus (Hepadanavirus family) replicates in the liver and causes hepatic dysfunction. Incubation period 2–6 months.

INVESTIGATIONS

Bloods: HBsAg (infection), core antibody (anti-HBc IgM – acute infection), hepatitis B e-markers (HBeAg – high infectivity), LFTs.

MANAGEMENT

Delivery: Caesarean section is *not* indicated, avoid FBS/FSE.
Postnatal: Mothers *can* breastfeed.
Neonatal vaccination: (If mother is HBsAg positive) at birth, 1 month, 2 months and booster at 1 year.
Neonatal HBIG: (Within 48 h of delivery) appropriate if birthweight <1500 g, if acute infection occurred in pregnancy, if mother is HBeAg positive or HBeAg negative + anti-HBe negative, or if e-markers are unknown.

COMPLICATIONS

Maternal: 10% develop chronic disease (chronic hepatitis, cirrhosis), ↑ risk of hepatocellular carcinoma, 1% develop fulminating hepatitis.
Neonates: 90% of babies infected with hepatitis B develop chronic hepatitis.

PROGNOSIS With appropriate treatment, vertical transmission rate is 10%.

Infections in pregnancy – *herpes simplex*

DEFINITION Infection caused by the herpes simplex virus (HSV).

AETIOLOGY Transmission by physical contact, sexual contact, vertical.

ASSOCIATIONS/RISK FACTORS Unprotected sexual intercourse, immunosuppression (e.g. HIV), other STIs.

EPIDEMIOLOGY Herpes simplex infects 2% of pregnant women.

HISTORY Burning sensation, pain, pruritis, dysuria (*note:* may be asymptomatic).

EXAMINATION Clusters of vesicles with surrounding erythema, can progress to ulcerated lesions which crust over, ?associated lymphadenopathy.

PATHOLOGY/PATHOGENESIS DNA virus (herpes family) of two types – 1: oral, 2: genital.

Dormant period: Following primary infection, HSV remains dormant in nerve ganglia and can be reactivated to form recurrent lesions.

Spread to neonate: Mainly direct contact with infected maternal secretions (but transplacental transmission possible), risk of neonatal transmission at vaginal delivery – 41% with primary lesions, 2% with recurrent lesions.

INVESTIGATIONS Usually diagnosed clinically.

Microbiology: Swabs for viral culture/PCR, STI screen.

Bloods: HSV antibody (primary infection in third trimester).

MANAGEMENT

Antenatal: Aciclovir (200 mg 5 × daily for 5 days) in primary infection.

Delivery with primary HSV: If within 6 weeks of likely delivery, advise Caesarean section. If opts for vaginal delivery, give IV aciclovir intrapartum, avoid prolonged ruptured membranes/FSE/FBS.

Delivery with recurrent HSV: Does not necessitate Caesarean section. Women may opt for Caesarean if lesions detected at onset of labour – can offer daily aciclovir from 36/40 to reduce likelihood of lesions.

COMPLICATIONS

Maternal: Disseminated herpes (encephalitis, hepatitis, disseminated skin lesions) rare but more common in pregnancy.

Neonatal: 1 per 60 000 live births – can affect skin/eyes/mouth, CNS or multiple organs.

PROGNOSIS Neonatal mortality from 2% (local disease) to 50% (disseminated disease).

Infections in pregnancy – *listeriosis*

DEFINITION Infection caused by the bacterium *Listeria monocytogenes*.

AETIOLOGY Found in soil, decayed matter and animals. Transmission by faeco-oral route (soft cheese, paté, unpasteurised dairy products, unwashed salads), vertical (transplacentally or during delivery).

ASSOCIATIONS/RISK FACTORS Increased risk of infection in pregnancy and immunosuppression.

EPIDEMIOLOGY Rare: 1 per 20 000 pregnancies in UK.

HISTORY Diarrhoea, vomiting, malaise, fever, sore throat, myalgia (*note*: often asymptomatic).

EXAMINATION No specific signs.

PATHOLOGY/PATHOGENESIS Gram-positive bacillus, transmission as noted above.

INVESTIGATIONS

Microbiology: Blood culture, amniotic fluid culture, placental culture (*note*: serological testing is not reliable).

MANAGEMENT IV antibiotics (penicillin and aminoglycoside, e.g. gentamicin).

COMPLICATIONS

General: Septicaemia, pneumonia, meningitis.
Pregnancy: Miscarriage, chorioamnionitis, PTL, fetal death.

PROGNOSIS Good prognosis if treated; poor prognosis with septicaemia (mortality 50%), meningitis (70%) or if neonatal infection occurs (80%).

Infections in pregnancy – *parvovirus B19*

DEFINITION Erythema infectiosum (Fifth disease) caused by parvovirus B19.

AETIOLOGY Transmission by aerosol route, also blood-borne.

ASSOCIATIONS/RISK FACTORS Most commonly affects children but also affects susceptible adults.

EPIDEMIOLOGY Common infection, with 60% immunity in adults by age 20 years. Affects 1 per 400 pregnancies.

HISTORY Rash, malaise, fever, arthropathy (*note:* 25% of cases are asymptomatic).

EXAMINATION Rash – commonly 'slapped cheek' appearance (erythema infectiosum). May have purpura, erythema multiforme.

PATHOLOGY/PATHOGENESIS Small single-stranded DNA virus, incubation period 13–20 days. Infective from 10 days prior to onset of rash until appearance of rash. Risk period of transmission to fetus is between 4/40 and 20/40. No intrauterine transmission under 4/40. Low risk of fetal hydrops after 20/40.

INVESTIGATIONS

Bloods: Parvovirus serology IgM (active infection), IgG (immunity), rubella serology (similar presentation).
USS: Fetal anomaly scan 4 weeks after onset of illness, and then at 1–2 weekly intervals until 30/40.

MANAGEMENT

Maternal: Symptomatic treatment (mild, self-limiting illness).
Fetus: Intrauterine blood transfusion (if fetal hydrops).

COMPLICATIONS

Maternal: Aplastic anaemia, arthritis, myocarditis, nephritis.
Fetus: Miscarriage (15%), fetal hydrops (3%).

PROGNOSIS Untreated fetal hydrops has 50% mortality (reduced to 18% by intrauterine blood transfusion). Does not cause congenital abnormalities.

Infections in pregnancy – *rubella*

DEFINITION Infection caused by the rubella virus.

AETIOLOGY Transmission by aerosol route, vertical (transplacental).

ASSOCIATIONS/RISK FACTORS Non-immunity (↑ rates in ethnic minorities).

EPIDEMIOLOGY Rare: 97% of women are vaccinated or immune in the UK.

HISTORY Fever, malaise, coryzal symptoms, arthralgia, rash.

EXAMINATION Lymphadenopathy, maculopapular rash (usually starting behind the ears, spreading to head and neck, then to the rest of the body).

PATHOLOGY/PATHOGENESIS RNA virus (Togaviridae family) with incubation period 6–21 days, infectious from 1 week prior to 5 days after onset of rash.

INVESTIGATIONS

Bloods: Rubella serology IgM (active infection), IgG (immunity).
USS: Fetal anomaly.

MANAGEMENT Symptomatic treatment of mother. May offer TOP if confirmed infection in first trimester.

COMPLICATIONS

Maternal: Miscarriage, pneumonia, arthropathy, encephalitis, ITP.
Fetal: Death, congenital rubella syndrome (deafness, VSD, PDA, cataracts, CNS defects, IUGR, hepatosplenomegaly, thrombocytopenia, rash).

PROGNOSIS Highest risk is congenital rubella syndrome with infection in first trimester (90%), 5–10% risk between 14 and 16/40, low risk after 20/40. Rubella and congenital rubella syndrome very rare owing to routine childhood vaccination since 1988 (MMR).

Infections in pregnancy – *toxoplasmosis*

DEFINITION Infection caused by the protozoon *Toxoplasma gondii*.

AETIOLOGY Transmission by faeco-oral route (found in infected meat and cat faeces).

ASSOCIATIONS/RISK FACTORS Household cats, ↑ incidence in rural areas, ↑ incidence in France.

EPIDEMIOLOGY Affects 1 per 2000 pregnancies.

HISTORY Fever, malaise, arthralgia (*note:* often asymptomatic).

EXAMINATION No specific clinical signs, ?fever/lymphadenopathy.

PATHOLOGY/PATHOGENESIS Parasite excreted in cat faeces, with incubation period 5–23 days. ↑ risk of vertical transmission with increasing gestational age (5% in the first trimester, 80% in the third trimester). However risk of congenital toxoplasmosis *reduces* with increasing gestational age (60–80% in the first trimester, 5% in the third trimester).

INVESTIGATIONS

Bloods: Toxoplasma IgM (active infection), IgG (immunity).
USS: Fetal anomaly scan.
Other: Amniocentesis (to detect fetal infection).

MANAGEMENT

Antibiotics: Spiramycin (↓ risk of vertical transmission).
Termination: May offer termination of pregnancy with USS evidence of fetal infection.

COMPLICATIONS Miscarriage, PTL, fetal death, congenital toxoplasmosis (hydrocephalus, retinochoroiditis, intracranial calcification, IUGR, hepatosplenomegaly, thrombocytopenia, rash).

PROGNOSIS Dependent on the severity of congenital toxoplasmosis.

Intrauterine death

DEFINITION Death of a fetus after 24/40.

AETIOLOGY

Maternal: Prolonged pregnancy, diabetes, pre-eclampsia, hypertension, obstetric cholestasis, SLE, antiphospholipid syndrome, thrombophilias, infections, Rhesus isoimmunisation, haemoglobinopathies, uterine rupture, maternal trauma, maternal death.

Fetal: Congenital abnormalities, genetic abnormalities, IUGR, infections (parvovirus, listeria, TORCH), hydrops fetalis, multiple gestation.

Placental: Placental insufficiency, abruption, vasa praevia.

Intrapartum factors: Birth asphyxia, shoulder dystocia, cord accident.

Other: Idiopathic.

ASSOCIATIONS/RISK FACTORS As for aetiology; also associated with ↑ maternal age, smoking, recreational drug use, previous intrauterine death.

EPIDEMIOLOGY Affects 5 per 1000 pregnancies.

HISTORY Reduced or absent fetal movements, symptoms of underlying conditions.

EXAMINATION

Abdomen: Absent FH, may have↓ fundal height, features of underlying conditions.

PATHOLOGY/PATHOGENESIS Dependent on the cause.

INVESTIGATIONS

USS: Confirm absent fetal heart movements (two accredited operators).

Bloods: FBC, U&E, LFT, CRP, HbA1c, clotting, thrombophilia screen, TORCH screen, parvovirus serology, Kleihauer (feto-maternal haemorrhage).

Other: Placental histology, fetal karyotype, post mortem.

MANAGEMENT Offer induction of labour.

Post-delivery: Offer bromocriptine or cabergoline to suppress lactation, arrange bereavement counselling, discuss consent for post mortem, follow-up with results.

COMPLICATIONS DIC, postnatal depression.

PROGNOSIS Unlikely to recur if no cause found, but manage subsequent pregnancies as 'high risk'.

Intrauterine growth restriction

DEFINITION Slowing of fetal growth such that it fails to reach its growth potential.

AETIOLOGY

Maternal: Hypertension, pre-eclampsia, diabetes, drug/alcohol abuse, smoking, renal disease, thrombophilia, ↑maternal age.
Fetal: Chromosomal abnormalities, infection (e.g. CMV, rubella), multiple pregnancy.
Other: Placental insufficiency.

ASSOCIATIONS/RISK FACTORS As for aetiology; previous IUGR.

EPIDEMIOLOGY Affects 3–5% of pregnancies.

HISTORY History of any aetiological factors noted above; enquire about fetal movements.

EXAMINATION

Abdomen: ↓fundal height.

PATHOLOGY/PATHOGENESIS

Symmetrical IUGR: Head and body are proportionally small, normally early onset, seen in chromosomal abnormalities.
Asymmetrical IUGR: Typically later onset, abdominal circumference disproportionately smaller than the head, seen with placental insufficiency.

INVESTIGATIONS

USS: Anomaly scan, growth (abdominal and head circumference), liquor volume (?oligohydramnios), umbilical artery Doppler (abnormal if end-diastolic flow absent or reversed), middle cerebral artery Doppler (may show redistribution of blood to brain).
CTG: Fetal wellbeing.

MANAGEMENT

Normal Doppler results: Aim delivery >37/40 unless abnormalities arise, with regular fetal monitoring.
Abnormal Doppler results: Steroids if pre-term, consider delivery, paediatrician at delivery, continuous CTG intrapartum.

COMPLICATIONS Stillbirth, PTL, intrapartum fetal distress, birth asphyxia, meconium aspiration, postnatal hypoglycaemia, neurodevelopmental delay, ↑ risk type 2 diabetes and hypertension in adult life.

PROGNOSIS Increased perinatal morbidity and mortality, increased neurodevelopmental delay if onset <26/40.

Malposition

DEFINITION Abnormal position of fetal vertex in relation to the maternal pelvis.

AETIOLOGY Fetal head normally engages in the OT position, rotating to the OA position with simultaneous flexion. Malpositions in labour include OP, OT and deflexion of the head.

ASSOCIATIONS/RISK FACTORS Cephalopelvic disproportion (e.g. android pelvis), epidural, inadequate uterine contractions.

EPIDEMIOLOGY Ten percent of labours start OP, most correct during labour.

HISTORY ? Prolonged labour.

EXAMINATION

Abdomen: OP suspected if the lower abdomen appears flattened.
Vaginal: Determine position through palpation of fetal fontanelles, assess for caput/moulding.

PATHOLOGY/PATHOGENESIS NA.

INVESTIGATIONS

CTG: For fetal wellbeing, frequency of contractions.
Bloods: FBC, G&S (preparation for delivery).

MANAGEMENT

Labour: Hydrate, augment with oxytocin infusion, offer epidural (↑ likelihood obstetric intervention, ↓ urge to push prior to full dilatation if OP).
Delivery: (If failure to progress) if fully dilated, <1/5 palpable abdominally and vertex at or below spines – vaginal delivery with manual rotation / Kielland's forceps / rotational ventouse delivery. Otherwise offer Caesarean section.

COMPLICATIONS

Maternal: Exhaustion, risks obstetric intervention.
Fetal: Fetal distress, complications of instrumental delivery.

PROGNOSIS Good; 75% of OP positions spontaneously rotate to OA.

Malpresentation

DEFINITION Any presentation where the presenting part is not the fetal vertex.

AETIOLOGY Associated with fetal/maternal factors that affect rotation.

ASSOCIATIONS/RISK FACTORS

Maternal: Placental site abnormalities, uterine abnormalities, obstructed lower segment (fibroids, pelvic abnormalities) grand multiparity (uterine laxity).
Fetal: Multiple gestation, prematurity, fetal malformation, polyhydramnios, macrosomia.

EPIDEMIOLOGY Prevalence at term: breech 3–4%, transverse lie 0.3%, face 0.002%, brow 0.001%, compound 0.1%.

HISTORY Asymptomatic, detected on examination.

EXAMINATION

Breech

Abdomen: Palpable head at fundus, soft breech in pelvis.
Vaginal: Soft presenting part, ischial tuberosities, anus or genitalia may be felt.
Footling breech: Foot felt/seen through the cervix.

Transverse lie

Abdomen: No presenting part felt in pelvis, uterus appears wide, fundal height may be low.
Vaginal: No presenting part felt in pelvis.

Face

Vaginal: Facial landmarks felt.

Brow

Vaginal: Supraorbital ridges ± base of nose felt.

PATHOLOGY/PATHOGENESIS NA.

INVESTIGATIONS

Breech or transverse: USS to confirm lie.

MANAGEMENT

Breech

ECV may be attempted after 37 weeks.

Persistent breech: Recommend Caesarean section (↑ fetal/neonatal morbidity with vaginal delivery).

Transverse lie

Requires delivery by Caesarean section, ECV occasionally attempted.

Brow

If persistent or second stage, deliver by Caesarean section.

Face

Mentoposterior position: Deliver by Caesarean section.
Mentoanterior: May attempt vaginal delivery.

Compound

Commonly this is fetal arm alongside fetal head – manage expectantly.

COMPLICATIONS

Maternal: Morbidity resulting from operative delivery, prolonged/obstructed labour, uterine rupture (especially transverse), PPH, ↑ perineal injury (mentoanterior face presentation).

Malpresentation (continued)

Fetal: ↑ perinatal mortality/morbidity (difficult delivery, prematurity, congenital abnormalities), cord prolapse (asphyxia).

PROGNOSIS Dependent on aetiology, presentation and timeliness of appropriate management.

Multiple pregnancy

DEFINITION Pregnancy involving more than one fetus.

AETIOLOGY

Monozygous: Division of fertilised egg – DCDA: splitting 3 days, 2 chorions, 2 amnions; MCDA: splitting 4–7 days, single placenta, one chorion, 2 amnions; MCMA: splitting 8–12 days, single placenta, one amnion, one chorion.

Dizygous: Fertilisation of >1 ovum by different sperm – DCDA: separate placentae, amnions and chorions.

ASSOCIATIONS/RISK FACTORS Previous history, family history, ovulation, African race, ↑ maternal age.

EPIDEMIOLOGY Incidence of twins, 11%; incidence of triplets, 1 per 8100.

HISTORY

First trimester: Incidental finding on ultrasound, hyperemesis (increased β-HCG).

Later in pregnancy: Large-for-dates, multiple fetal parts on abdominal examination.

EXAMINATION

Abdomen: ↑fundal height, multiple fetal parts, >1 FH.

PATHOLOGY/PATHOGENESIS NA.

INVESTIGATIONS Confirmation by USS: establishment of chorionicity (almost 100% accurate in first trimester only), nuchal translucency (*note:* serum screening unreliable).

MANAGEMENT

Antenatal: Serial USS for fetal growths (dichorionic: 4-weekly from 24/40; monochorionic: 2-weekly scans from 18 weeks), monitor FBC (↑ anaemia), monitor BP (↑ eclampsia), GTT (↑ diabetes).

Vaginal delivery: May be attempted if first twin cephalic (need continuous monitoring and active management in third stage).

Caesarean section: Recommended for delayed delivery of second twin, fetal distress of either twin, malpresentation of second twin after delivery of first (ECV or internal version may be attempted prior), non-vertex presentation of first twin, monoamniotic twins, higher order births.

COMPLICATIONS

Maternal: Miscarriage, hyperemesis, pre-eclampsia, anaemia (↑ plasma volume and fetal iron requirements), PPH, APH, diabetes (↑ steroid load), operative delivery, postnatal depression.

Fetal: Prematurity, polyhydramnios, congenital malformations, IUGR, fetal death, discordant growth.

Monochorionic twins: Twin–twin transfusion syndrome (placental anastomoses produce haemodynamic imbalance resulting in polyhydramnios and hydrops in one twin and oligohydramnios and IUGR in the other).

Intrapartum: Cord prolapse, twin interlocking, ↑ perinatal morbidity of second twin (vaginal delivery).

PROGNOSIS Dependent on complications; twin fetal mortality rate 4 times higher than singletons.

Obstetric cholestasis

DEFINITION Pruritis in pregnancy, which resolves on delivery, associated with abnormal liver function in the absence of any other identifiable pathology.

AETIOLOGY Complex, likely genetic and hormonal factors.

ASSOCIATIONS/RISK FACTORS Previous history, family history, ethnicity (South Asian, Chilean, Bolivian), multiple pregnancy.

EPIDEMIOLOGY UK prevalence: 0.7% of pregnancies.

HISTORY Normally occurs in the second half of pregnancy. Generalised pruritis in the absence of rash (often worse at night, may be more intense over the palms and soles), rarely associated with dark urine and steatorrhoea.

EXAMINATION Often entirely normal but excoriations may be present.

PATHOLOGY/PATHOGENESIS Increased susceptibility to cholestatic effect of oestrogens, ?related to impaired sulfation, may also be related to a defect in membrane phospholipid which may be inheritable.

INVESTIGATIONS

Bloods: LFT (↑ transaminases/GGT, occasionally mild hyperbilirubinaemia, may be preceded by symptoms therefore consider fortnightly repeat if normal), bile acids (raised), clotting (may be abnormal due to ↓ vitamin K absorption).

Diagnosis of exclusion, therefore need: PET screen (FBC, U&E, LFT, clotting), liver USS, hepatitis serology (A, B, C and E), EBV and CMV serology, liver autoantibodies (AMA, ASMA).

MANAGEMENT

Monitoring: Weekly LFT and clotting, serial USS for fetal + intermittent CTG monitoring (limited in prediction of fetal compromise).

Medications: Chlorpheniramine (control pruritis), ursodeoxycholic acid (↓ serum bile acids and pruritis, no effect on fetal compromise), vitamin K (↓ fetal/maternal haemorrhage), dexamethasone (if no response to UCDA).

Delivery: Induce at 37/40 owing to increased risk of fetal death.

Postpartum: Ensure resolution of abnormal LFTs after 10/7 postpartum.

COMPLICATIONS

Maternal: PPH (↓ vitamin K).

Fetal: Death, intracranial haemorrhage (↓ vitamin K), fetal distress, preterm delivery.

PROGNOSIS Complete recovery is made postnatally, but 90% recurrence in future pregnancies. Risk of fetal death is 2–3%.

Oligohydramnios

DEFINITION Decreased volume of amniotic fluid, below fifth centile, or deepest pool less than 2 cm.

AETIOLOGY Rupture of membranes, placental insufficiency, fetal urinary tract pathology/malformations.

ASSOCIATIONS/RISK FACTORS Chromosomal abnormalities, post-term pregnancies, IUGR, pre-eclampsia, medication (ACE inhibitors, indomethacin), multiple pregnancy (e.g. TTTS).

EPIDEMIOLOGY Affects 4% of pregnancies.

HISTORY History of fluid leak PV with rupture of membranes (*note:* commonly asymptomatic).

EXAMINATION

Abdomen: ↓ fundal height, fetal parts easily palpable.
Speculum: Assess for ruptured membranes if clinically appropriate.

PATHOLOGY/PATHOGENESIS Reduced amniotic fluid volume by loss of fluid or reduced fetal urine output (↓ placental function or fetal urinary tract pathology).

INVESTIGATIONS

USS: Diagnosis + assessment of liquor volume, fetal growth, umbilical artery Dopplers, exclude fetal anomalies.
CTG: Fetal wellbeing.

MANAGEMENT

If PROM/PPROM: See relevant sections.
Term: Delivery is appropriate (IOL if no contraindications).
Pre-term: Monitor with serial USS for growth, liquor volume and Dopplers, regular CTGs, deliver if further abnormalities arise (*note:* amnioinfusion has a very limited role in modern obstetrics).

COMPLICATIONS

Labour: ↑ incidence CTG abnormalities, meconium liquor, emergency Caesarean section.
Neonate: pulmonary hypoplasia, limb deformities.

PROGNOSIS Dependent on gestation at time of presentation: increased perinatal mortality rates with early-onset oligohydramnios.

Placental abruption

DEFINITION Separation of the placenta from the uterine wall prior to delivery.

AETIOLOGY Often idiopathic, may occur secondary to raised pressure on maternal side of placenta (e.g. hypertension) or mechanical trauma.

ASSOCIATIONS/RISK FACTORS Hypertensive disorders, previous history of abruption, PPROM, abdominal trauma, smoking, cocaine usage, polyhydramnios.

EPIDEMIOLOGY Affects 1–2% of pregnancies.

HISTORY Constant abdominal pain, PV bleed (if revealed).

EXAMINATION

General: ?shocked.
Abdomen: Hypertonic 'woody', tender uterus.
Speculum: Assess bleeding.
Vaginal: (Ensure not known placenta praevia) cervical dilatation (consider ARM if term and no fetal distress).
CTG: Fetal wellbeing.

PATHOLOGY/PATHOGENESIS As the placenta separates, retroplacental bleeding results in further placental detachment which may lead to fetal and maternal compromise. Haemorrhage may be concealed (20%) or revealed (80%).

INVESTIGATIONS

Bloods: FBC, clotting, U&E, X-match.
USS: Exclude placenta praevia (*note:* abruption unlikely to be evident on USS unless very large).

MANAGEMENT Depends on gestation and severity of abruption.

Mild abruption

If preterm and stable, may be managed conservatively with close monitoring; IOL if at term.

Severe abruption or maternal/fetal compromise

Requires immediate delivery by Caesarean section, airway, breathing, circulation (two large-bore IV cannulae), fluid resuscitation, correction of coagulopathy with FFP/cryoprecipitate/platelets, blood transfusion if necessary.

COMPLICATIONS

Maternal: Haemorrhage (APH + ↑ risk PPH), DIC, renal failure, 'Couvelaire' uterus (extravasation of blood into the myometrium and beneath the peritoneum).
Fetal: Birth asphyxia, death.

PROGNOSIS

Maternal: Mortality 0.5% in severe abruption.
Fetal: Mortality 3.3% in severe abruption.

Placenta praevia

DEFINITION A placenta wholly or partly inserting into the lower segment.

AETIOLOGY Unknown.

ASSOCIATIONS/RISK FACTORS Multiple pregnancy, ↑maternal age, previous uterine surgery, previous placenta praevia, smoking.

EPIDEMIOLOGY Affects 0.5% of pregnancies.

HISTORY Usually detected on routine ultrasound. Acutely, presents with painless PV bleeding in the second/third trimester.

EXAMINATION

General: May be shocked (tachycardia, hypotension)
Abdomen: Usually soft, non-tender, ?malpresentation.
Vaginal: Contraindicated.
Speculum: Gentle speculum examination permitted (assess bleeding).

PATHOLOGY/PATHOGENESIS Unknown. Abnormal implantation thought to occur where blood supply disrupted (e.g. uterine scar). Bleeding occurs secondary to shearing forces.
Minor placenta praevia: Placenta close to, but not covering, internal cervical os.
Major placenta praevia: Placenta overlying internal cervical os.

INVESTIGATIONS

Bloods: FBC, U&E, clotting, X-match.
CTG: Fetal wellbeing.
USS: Confirm placental site.
MRI: May be used to determine placenta accreta (placenta adherent to uterine wall) or placenta increta/percreta (placenta invades into or through the uterine wall).

MANAGEMENT

Vaginal delivery: Only possible if lower placental edge >2 cm from internal os and fetal head below placenta.
Caesarean section: If no bleeding, aim for 38/40.
Mild self-limiting bleed, no fetal/maternal compromise: Admit for monitoring, steroids if preterm, anti-D if Rhesus negative.
Severe bleed or fetal/maternal compromise: Manage as for severe placental abruption or maternal/fetal compromise (see above).

COMPLICATIONS

Maternal: Haemorrhage (APH + ↑ risk PPH), DIC, hysterectomy.
Fetal: IUGR, death.

PROGNOSIS Maternal mortality is 1 per 300.

Polyhydramnios

DEFINITION Increased volume of amniotic fluid, above 95th centile, or deepest pool greater than 8 cm.

AETIOLOGY Idiopathic, failure of fetal swallowing (neurological, chromosomal anomalies), fetal GI tract abnormality (duodenal/oesophageal atresia), congenital infections, fetal polyuria (diabetes, TTTS).

ASSOCIATIONS/RISK FACTORS As above.

EPIDEMIOLOGY Affects 1–4% of pregnancies.

HISTORY Symptoms of underlying aetiology, maternal discomfort.

EXAMINATION

Abdomen: ↑ fundal height, impalpable fetal parts, tense abdomen.

PATHOLOGY/PATHOGENESIS Raised amniotic fluid volume caused by increased fetal urine production or failure of fetal swallowing/intestinal absorption.

INVESTIGATIONS

USS: Diagnosis + assessment of liquor volume, fetal growth, umbilical artery Dopplers, exclude fetal anomalies.
Other: Exclude maternal diabetes.

MANAGEMENT

Amnioreduction: Only if gross polyhydramnios is causing significant discomfort.
Cyclo-oxygenase inhibitors: Occasionally used to decrease fetal urine output.
Diabetes: Optimise diabetic control.
Other: Paediatrician present at delivery.

COMPLICATIONS PTL, malpresentation, placental abruption, cord prolapse, complications of underlying pathology, PPH, ↑ risk Caesarean section.

PROGNOSIS Increased perinatal morbidity and mortality, related to PTL/congenital anomalies.

Postnatal depression

DEFINITION

Postpartum blues: Mild, self-limiting low mood in the postnatal period.
Postnatal depression: Pervasive low mood in the postnatal period.
Puerperal psychosis: Acute onset of psychotic illness in the postnatal period.

AETIOLOGY Poorly understood.

ASSOCIATIONS/RISK FACTORS Past psychiatric history, previous postnatal depression, antenatal or delivery complications, primigravidity (postnatal blues), social isolation.

EPIDEMIOLOGY

Postpartum blues: >80% of new mothers.
Postnatal depression: 10% of new mothers.
Puerperal psychosis: 1 per 1000 new mothers.

HISTORY

Postpartum blues: Emotional lability, irritability, poor sleep and concentration (often day 3–5 postnatally).
Postnatal depression: Low mood, decreased appetite, early-morning waking, anhedonia, anxiety (often occurs up to 6 weeks postnatally).
Puerperal psychosis: Mania, delusions, hallucinations, thoughts of self-harm (often presents day 3–7 postnatally).

EXAMINATION Mental state examination, depression scales may be used.

PATHOLOGY/PATHOGENESIS Poorly understood, thought to be related to falling levels of oestrogens, progesterone and cortisol postnatally.

INVESTIGATIONS NA.

MANAGEMENT

Postpartum blues: Reassurance, support (self-limiting).
Postnatal depression: Depends on severity – counselling, CBT, antidepressants.
Puerperal psychosis: Psychiatric emergency requiring inpatient admission – antidepressants, antipsychotics, ?ECT.

COMPLICATIONS Poor emotional attachment to child, long-term psychiatric morbidity, suicide (up to 5% with puerperal psychosis), infanticide (up to 4% with puerperal psychosis).

PROGNOSIS Recurrence in subsequent pregnancies: postnatal depression 30%, puerperal psychosis 20%.

Postpartum haemorrhage

DEFINITION Blood loss of >500 mL at vaginal delivery or >1000 mL at Caesarean section.

Primary PPH: Within 24 hours.
Secondary PPH: 24 hours to 6 weeks.

AETIOLOGY

Primary PPH: Uterine atony (80%), trauma to perineum/vagina/cervix, retained placenta/membranes, coagulopathy.
Secondary PPH: Endometritis, retained placenta/membranes.

ASSOCIATIONS/RISK FACTORS Multiple pregnancy, polyhydramnios, APH, grand multiparity, fibroid uterus, previous PPH, prolonged labour, augmented labour, instrumental delivery, high birthweight infant, personal or family history of bleeding disorder, anticoagulant use, Caesarean section, pyrexia in labour, episiotomy, placental site abnormalities.

EPIDEMIOLOGY Affects 5% of deliveries.

HISTORY

Primary: Excessive PV bleed after delivery (*note:* verify whether placenta is complete).
Secondary: PV bleed, abdominal pain, PV discharge, fever.

EXAMINATION

Primary
General: Shock (tachycardia, hypotension), signs of anaemia.
Abdomen: ?atonic uterus (above umbilicus)
Speculum: Exclude trauma (perineal/vaginal/cervical).
Vaginal: Evacuate clots from cervix (inhibits contraction).

Secondary
Abdomen: Tender uterus.
Speculum: Assess bleeding, ?cervical os open.
Vaginal: ?uterine tenderness.

PATHOLOGY/PATHOGENESIS See the aetiologies noted above. Placental blood flow >500 mL/min at term. With atony, poor uterine contraction (compresses spiral arteries) leads to heavy blood loss.

INVESTIGATIONS

Primary
Bloods: FBC, U&E, clotting, X-match.

Secondary
Bloods: FBC, U&E, clotting, X-match, CRP.
Microbiology: HVS.
USS: ?retained products.

MANAGEMENT

Primary
Airway, breathing, circulation (two large-bore IV cannulae), fluid resuscitation, blood transfusion if necessary.

Atony: Bimanual compression, uterotonics (oxytocin bolus + infusion, IM ergometrine, IM carboprost, PR misoprosol), transfer to theatre.
Consider: Intra-uterine balloon insertion, uterine artery embolisation, laparotomy and insertion of brace suture, hysterectomy.
Trauma: Requires suturing (consider transferring to theatre).

Postpartum haemorrhage (continued)

Retained products: Manual evacuation in theatre.
Coagulopathy: Correct with FFP/cryoprecipitate/platelets.

Secondary
Resuscitate as appropriate, IV antibiotics, ERPC only if unavoidable (increased risk of uterine perforation).

COMPLICATIONS Death, hysterectomy, ↑VTE, renal failure, DIC, Sheehan's syndrome (pituitary hypoperfusion following massive PPH causes pituitary necrosis and hypopituitarism).

PROGNOSIS Fourth most common cause of maternal death in the UK. Leading cause of maternal mortality worldwide.

Pre-eclampsia

DEFINITION Proteinuric hypertension in pregnancy, developing after 20/40.

AETIOLOGY See pathology/pathogenesis below.

ASSOCIATIONS/RISK FACTORS Nulliparity, ↑maternal age, family history, previous history of pre-eclampsia, pre-existing hypertension, new partner, pre-existing renal disease, diabetes, PCOS, multiple pregnancy, obesity.

EPIDEMIOLOGY Affects 2–5% of pregnancies.

HISTORY Headache, oedema, visual disturbance, right upper quadrant pain due to liver capsule swelling (*note:* often asymptomatic).

EXAMINATION

General: ↑BP, oedema (especially facial), hyperreflexia, clonus, ?papilloedema
Abdomen: ?RUQ tenderness, ?reduced fundal height.

PATHOLOGY/PATHOGENESIS Impaired trophoblastic invasion into the spiral arteries during placentation. Increased resistance in the uteroplacental circulation leads to hypoperfusion/ischaemia releasing inflammatory mediators which cause widespread endothelial damage, end-organ dysfunction and oedema.

INVESTIGATIONS

Bloods: FBC (↓platelets, haemoconcentration), U&E and urate (renal impairment), LFT (↑ transaminases), clotting.
Urine: Urinalysis (proteinuria), MSU (exclude UTI), 24 h urine collection (significant proteinuria >0.3 g protein per 24 hours).
USS: Fetal growth, liquor volume and umbilical artery Dopplers.
CTG: Fetal wellbeing.

MANAGEMENT Depends on gestational age and severity of condition. The condition will not resolve until after delivery.

Mild/moderate pre-eclampsia: Regular monitoring of BP with urinalysis, regular blood testing, serial USS for fetal growth, regular CTGs, antihypertensives (e.g. methyldopa, labetalol, nifedipine), aim for delivery after 37 weeks.
Severe pre-eclampsia/evidence of fetal compromise: (*Note:* requires delivery) antihypertensives (labetalol, nifedipine, hydralazine), seizure prophylaxis (IV magnesium sulphate), fluid restriction and strict fluid balance (catheterise, consider CVP monitoring), steroids for lung maturity if preterm.

COMPLICATIONS

Maternal: Eclampsia, abruption, CVA, pulmonary oedema, cerebral oedema, renal failure, liver failure, DIC, HELLP syndrome (haemolysis, elevated liver enzymes, low platelets)
Fetal: IUGR, death.

PROGNOSIS Severe pre-eclampsia has 10% recurrence in subsequent pregnancies.

Prelabour rupture of membranes (PROM) at term

DEFINITION Spontaneous rupture of membranes prior to onset of labour at term.

AETIOLOGY Natural physiological mechanisms including Braxton Hicks contractions and cervical ripening lead to weakening of the membranes.

ASSOCIATIONS/RISK FACTORS None known.

EPIDEMIOLOGY Affects 8% of pregnant women.

HISTORY Sudden gush of fluid loss PV, followed by constant trickle.

EXAMINATION
General: Assess for signs of infection (fever, tachycardia).
Vaginal: Avoid if possible (↑ risk infection).
Speculum: (If history uncertain) confirm pooling of liquor in vagina, note liquor colour.

PATHOLOGY/PATHOGENESIS NA.

INVESTIGATIONS
Microbiology: Consider HVS/LVS.

MANAGEMENT

Clear liquor and no known GBS
Expectant management for 24 hours (majority of women will labour). Offer augmentation of labour after 24 hours (may opt for expectant management for up to 72 hours with 4-hourly temperature and 24-hourly fetal monitoring). Augment labour with prostaglandin or oxytocin infusion. Antibiotic cover (benzylpenicillin or clindamycin if penicillin allergic) dependent on unit protocol.

Meconium or known GBS or pyrexia
Augment labour immediately (antibiotics if known GBS/pyrexia).

Postnatal
Neonatal observation required for at least 12 hours.

COMPLICATIONS Increased risk of ascending infection.

PROGNOSIS Sixty percent of women will labour over the next 24 hours.

Preterm prelabour rupture of membranes (PPROM)

DEFINITION Spontaneous rupture of membranes prior to onset of labour in a pregnancy <37 weeks.

AETIOLOGY Weakening of the membranes strongly linked to an infective cause (often subclinical).

ASSOCIATIONS/RISK FACTORS Also associated with APH, trauma, UTI, previous PROM/PTL, uterine abnormalities, cervical incompetence, smoking, multiple pregnancy, polyhydramnios.

EPIDEMIOLOGY Affects 2% of pregnancies.

HISTORY Sudden gush of fluid loss PV, followed by constant trickle.

EXAMINATION
General: Assess for signs of infection (fever, tachycardia).
Vaginal: Avoid (↑ risk infection)
Speculum: Confirm pooling of liquor in vagina, note liquor colour.

PATHOLOGY/PATHOGENESIS See the aetiological link noted above.

INVESTIGATIONS
Bloods: FBC, CRP (?infection)
Microbiology: MSU, HVS/LVS.
CTG: Fetal wellbeing.
USS: Confirm presentation, estimated fetal weight.

MANAGEMENT Admit for monitoring (48–72 h), steroids (fetal lung maturity), antibiotic (erythromycin 250 mg 4 × daily for 10 days), monitor temperature 4-hourly, regular CTG. Consider tocolysis in the presence of uterine activity only if intrauterine transfer required or for steroid cover. If subsequently managed as an outpatient: weekly HVS and bloods, twice daily temperature check (monitor for infection). Aim to deliver after 34/40, or earlier if evidence of infection. If <23/40, discuss TOP (extremely poor outcomes).

COMPLICATIONS
Maternal: Sepsis, placental abruption.
Fetal: Chorioamnionitis, cord prolapse, PTL, pulmonary hypoplasia, limb contractures, death.

PROGNOSIS Increased perinatal mortality predominantly due to sepsis, prematurity and pulmonary hypoplasia.

Preterm labour (PTL)

DEFINITION Onset of labour prior to 37 weeks' gestation.

AETIOLOGY May be idiopathic. Infection (often subclinical) contributes increasingly to aetiology with decreasing gestation. See pathology/pathogenesis below.

ASSOCIATIONS/RISK FACTORS Infection of genital tract, UTI, multiple pregnancy, polyhydramnios, cervical incompetence/previous cervical surgery, systemic infective illness, previous PTL, uterine abnormalities, APH.

EPIDEMIOLOGY Affects 6% of deliveries in the UK.

HISTORY Regular painful contractions, although may present as diffuse pains or cramping. ? PV bleed, ?SROM.

EXAMINATION

General: Signs of infection (tachycardia, fever).
Abdomen: Contractions, abdominal tenderness (may indicate abruption, chorioamnionitis).
Speculum: ?liquor pooling.
Vaginal: Assess cervical dilatation.

PATHOLOGY/PATHOGENESIS Relationship with systemic infective illness may be due to direct spread of infection or release of inflammatory mediators. Decidual haemorrhage is also associated with release of inflammatory mediators. PTL in polyhydramnios/multiple pregnancy is related to uterine over-distension.

INVESTIGATIONS

Bloods: FBC, CRP (evidence of infection).
Microbiology: MSU, LVS/HVS.
CTG: Fetal wellbeing.
USS: Confirm presentation, estimated fetal weight, cervical length.
Fetal fibronectin: (Sample taken at speculum examination) predictive of likelihood to labour (used for high negative predictive value).

MANAGEMENT Administer steroids (fetal lung maturation), tocolysis used to allow steroid cover e.g. oxytocin antagonists (atosiban), nifedipine or GTN (contraindicated if APH or evidence of infection), antibiotics if SROM, fetal monitoring.

COMPLICATIONS Respiratory distress syndrome, intracranial haemorrhage, sepsis, necrotising enterocolitis, neurodevelopmental delay, cerebral palsy, neonatal death.

PROGNOSIS Dependent on gestation at delivery, accounts for over 20% of perinatal mortality. There is 20% risk of PTL in subsequent pregnancies.

Prolonged labour

DEFINITION Poor progress in labour.

First stage

Primip: Cervical dilatation of less than 2 cm in 4 hours.
Multip: Cervical dilatation of less than 2 cm in 4 hours or a slowing in progress.

Second stage

A delay in delivery of >2 h (primp) or >1 h (multip) from active pushing.

Third stage

Failure to deliver the placenta after >30 min (active management), >1 h (physiological). *See* National Institute for Health and Clinical Excellence clinical guideline, *Intrapartum Care: Management and Delivery of Care to Women in Labour*, 2007.

AETIOLOGY

First/second stage

'Powers': Frequency and duration of contractions.
'Passenger': Fetal size, malpresentation, malpositioning.
'Passages': CPD.

Third stage

Failure of the placenta to detach from the placental bed.

ASSOCIATIONS/RISK FACTORS

First/second stage

See the aetiologies noted above. *Also:* primigravidity, ↑maternal age, big baby, short stature, obesity, induction of labour, epidural (delay in second stage), cervical surgery, pelvic trauma, fetal malformations.

Third stage

Previous retained placenta, previous injury to uterus, pre-term delivery, induction of labour, multiparity.

EPIDEMIOLOGY

First/second stage

Contributes to 30–50% of overall Caesarean section rates.

Third stage

Affects 0.8–1.2% of births.

HISTORY Assess risk factors as above, review partogram.

EXAMINATION

First/second stage

General: Maternal exhaustion, dehydration, blood-stained urine (sign of obstructed labour).
Abdomen: Fetal size, lie, presentation, engagement.
Vaginal: Cervical dilatation, station, presentation, position, presence of membranes, signs of obstruction (caput/moulding), assess pelvic capacity.

Third stage

Failure of placental delivery with controlled cord traction.

PATHOLOGY/PATHOGENESIS See the aetiologies noted above.

INVESTIGATIONS

Bloods: FBC, G&S (preparation for delivery/theatre).
CTG: Fetal wellbeing (first/second stage).

Prolonged labour (continued)

MANAGEMENT

First stage

Fluid rehydration, adequate pain relief, ARM if appropriate, augmentation of labour with oxytocin infusion (caution in multips). Caesarean section for malpresentation. Consider FBS/delivery if signs of fetal distress.

Second stage

Consider oxytocin. If fully dilated, <1/5 palpable abdominally and vertex at/below spines: instrumental delivery. Otherwise Caesarean section.

Third stage

Administer oxytocin, if still retained after 30 minutes needs manual removal of placenta (often in theatre).

COMPLICATIONS

First/second stage

Maternal: Risks of instrumental delivery or Caesarean section, PPH, uterine rupture, fistula formation (rare in UK).

Fetal: Fetal distress, complications of instrumental delivery, ↑ risk shoulder dystocia.

Third stage

PPH, infection.

PROGNOSIS

First/second stage

While many cases respond to intervention, prolonged labour still accounts for a high proportion of Caesareans.

Third stage

Good prognosis.

Prolonged pregnancy

DEFINITION Pregnancy that proceeds beyond 42 weeks.

AETIOLOGY Unknown.

ASSOCIATIONS/RISK FACTORS Previous post-term pregnancy, primiparity, obesity.

EPIDEMIOLOGY Affects 5–15% of pregnancies.

HISTORY Failure to enter spontaneous labour by 42 weeks. Ensure adequate fetal movements and accurate dating from early USS.

EXAMINATION
Abdomen: Fundal height, auscultate FH.
Vaginal: Assess cervix for induction of labour.

PATHOLOGY/PATHOGENESIS Placental function decreases post-term (histology shows calcification, syncytial knotting), associated with increased rates of perinatal morbidity and mortality.

INVESTIGATIONS
USS: Liquor volume, growth.
CTG: At least twice-weekly for fetal wellbeing.

MANAGEMENT All women should be offered membrane sweeping from 40–41/40 (↑ likelihood spontaneous labour). Induction of labour should be offered at between 41 and 42 weeks. Women's decisions to decline induction should be respected, but they should be advised that no monitoring techniques are predictive of fetal death.

COMPLICATIONS Increased risk of stillbirth (from 1 per 1000 at 37/40 to 3 per 1000 at 42/40), ↑ risk perinatal morbidity/mortality, ↑ risk Caesarean section, ↑ meconium staining of liquor.

PROGNOSIS Recurrence of 30% in subsequent pregnancies.

Rhesus isoimmunisation

DEFINITION Development of Rhesus antibodies in a Rhesus-negative mother after exposure to Rhesus-positive blood cells (most commonly RhD antigen).

AETIOLOGY A sensitising event exposes a RhD-negative mother to a RhD-positive fetus' red blood cells (can occur through RhD-positive blood transfusion but rare in the UK). Maternal antibodies develop to RhD. In subsequent pregnancies involving a RhD-positive fetus, maternal IgG antibodies will cross the placenta, forming complexes with RhD on fetal erythrocytes and destroying them. Sensitising events include delivery, APH, miscarriage, ectopic pregnancy, TOP and invasive prenatal testing.

ASSOCIATIONS/RISK FACTORS Previous pregnancy with insufficient/without anti-D prophylaxis, previous blood transfusion (rare if occurred in the UK).

EPIDEMIOLOGY Fifteen percent of Caucasian populations are RhD-negative. Without prophylaxis, isoimmunisation occurs in 1%.

HISTORY Often picked up on routine antenatal screening, patient may be aware of diagnosis from previous pregnancies. May have poor obstetric history.

EXAMINATION NA.

PATHOLOGY/PATHOGENESIS IgG antibodies against RhD-positive fetal red cells cause haemolysis leading to anaemia, hyperbilirubinaemia and subsequent hydrops fetalis (fetal heart failure associated with accumulation of fluid subcutaneously and in body compartments – e.g. ascites, pericardial effusion).

INVESTIGATIONS Regular monitoring of maternal antibody levels. Once threshold level for prediction of anaemia is reached, needs monitoring in a fetal medicine centre with USS markers of anaemia (e.g. MCA Doppler). Monitoring may also include: measurement of amniotic fluid bilirubin concentration, or cordocentesis.

MANAGEMENT

Prevention: Anti-D immunoglobulin prophylaxis is recommended for all RhD-negative mothers at 28/40 and 34/40, following any sensitising event, and after delivery (if baby is RhD-positive).

Rhesus isoimmunisation in current pregnancy: May necessitate fetal blood transfusions during pregnancy, and exchange transfusion in the neonate following delivery.

COMPLICATIONS Hydrops fetalis, intrauterine death, neonatal death, neonatal kernicterus.

PROGNOSIS Dependent on the severity of disease.

Shoulder dystocia

DEFINITION Difficulty with delivering the fetal shoulders following delivery of the head.

AETIOLOGY Bony impaction of the anterior fetal shoulder behind the maternal symphysis pubis.

ASSOCIATIONS/RISK FACTORS High birthweight baby (although 50% occur with normal birthweight babies), post-dates, previous shoulder dystocia, diabetes, obesity, instrumental delivery, prolonged labour.

EPIDEMIOLOGY Affects 1% of deliveries.

HISTORY Delay in delivery of fetal shoulders, 'turtle-necking' of fetal head.

EXAMINATION Evidence of shoulder dystocia as above.

PATHOLOGY/PATHOGENESIS NA.

INVESTIGATIONS NA.

MANAGEMENT The mnemonic HELPERR below is often used. Each manoeuvre should be attempted for up to 30 seconds. Meticulous documentation is mandatory.
H: Call for help.
E: Evaluate for episiotomy.
L: Legs – the McRoberts manoeuvre, hyperflexing the maternal thighs on to the abdomen.
P: Pressure suprapubically on to the posterior aspect of the fetal shoulder.
E: Enter manoeuvres – for internal rotation of the fetal shoulders into the oblique plane.
R: Remove the posterior arm.
R: Roll the patient on to all-fours.

Manoeuvres of last resort include deliberate clavicular fracture, symphysiotomy, general anaesthesia and the Zavanelli manoeuvre (replacement of the fetal head followed by Caesarean section).

COMPLICATIONS
Maternal: PPH, uterine rupture, extensive perineal tears (↑ third/fourth degree tears), symphyseal separation.
Fetal: Death, hypoxic–ischaemic encephalopathy, fractured humerus/clavicle, brachial plexus injury.

PROGNOSIS Perinatal mortality is 1–2%. A quarter of infants suffer brachial plexus injury (1–2% long-term dysfunction). 10–15% recurrence in future pregnancies.

Venous thromboembolism (VTE) in pregnancy

DEFINITION

Deep vein thrombosis (DVT): Formation of blood clot in deep veins (usually of the leg).
Pulmonary embolism (PE): Distal spread of a thrombus to the pulmonary vasculature.

AETIOLOGY Thrombus formation initiated by endothelial injury/stasis/hypercoagulability (Virchow's triad). Commonly occurs in deep veins of leg/pelvis and embolises to pulmonary vasculature. Pregnancy is a pro-coagulant state.

ASSOCIATIONS/RISK FACTORS

General: ↑maternal age, thrombophilia, obesity, personal or family history of PE, smoking, immobility, post-surgery.
Pregnancy: Caesarean section (especially emergency), instrumental delivery, infection, pre-eclampsia, multiple pregnancy, hyperemesis/dehydration.

EPIDEMIOLOGY Affects 1 per 6000 pregnancies.

HISTORY

DVT: Classically red, hot, swollen tender calf.
PE: Pleuritic chest pain, dyspnoea, cough, haemoptysis.

EXAMINATION

Deep vein thrombosis

Unilateral lower limb oedema, erythema, tenderness, may have low-grade pyrexia.

Pulmonary embolism

General: Tachycardia, tachypnoea, low-grade pyrexia, reduced O_2 saturation on pulse oximetry, cardiorespiratory collapse (rare).
Chest auscultation: May be normal or reveal reduced air entry, crepitations.
Cardiovascular: ?loud P2.

PATHOLOGY/PATHOGENESIS See the aetiologies noted above.

INVESTIGATIONS

Deep vein thrombosis

Duplex USS.

Pulmonary embolism

General: ABG (hypoxia/hypocapnia), ECG (sinus tachycardia/S1Q3T3).
Imaging: CXR, duplex USS (if both negative, then V/Q or CTPA).
Bloods: (Prior to anticoagulation) FBC, U&E, LFT, clotting.

MANAGEMENT

Prevention: All obstetric patients should be evaluated for thromboprophylaxis (e.g. compression stockings, LMWH).
Treatment: LMWH, initiated on clinical suspicion and discontinued if diagnosis excluded. Continue for remainder of pregnancy. Discontinue at start of labour or 24 hours prior to planned delivery. Continue treatment 6/52 postnatally (can convert to PO anticoagulation 3/7 after delivery).
Massive PE: ABC, multidisciplinary management, IV unfractionated heparin preferred. May rarely require thrombolysis, thoracotomy or surgical embolectomy.

COMPLICATIONS Death, effects of long-term anticoagulation, post-thrombotic leg syndrome.

PROGNOSIS Pulmonary embolism is the largest single cause of maternal death in the UK, resulting in 15 maternal deaths/year.