FINAL YEAR MBBS · CLINICAL VIVA

Obstetrics Viva Voce

Common long and short cases for MBBS clinical exams
Based on *Obstetrics by Ten Teachers*

Prof. Dr. Tayyaba Majeed
Gynaecologist and Obstetrician, Hameed Latif Hospital, Lahore

Published by Prof. Zahid Mahmood · professorzahid.com · 0300-4130159
700 viva questions with model answers · 14 cases

How to use this book

Each chapter is one common examination case. It opens with the typical case you may meet in the exam, followed by 50 questions that examiners commonly ask, from simple to hard: history and examination, diagnosis, investigations, management and complications.

The answers are short model answers, the way an examiner wants to hear them. Read the question, answer it aloud first, then check the model answer. Guidance follows UK practice as taught in Ten Teachers (NICE and RCOG); follow your local hospital protocols where they differ.

Contents

Case 1	Normal Pregnancy and Antenatal Booking	50 questions
Case 2	Anaemia in Pregnancy	50 questions
Case 3	Pre-eclampsia and Eclampsia	50 questions
Case 4	Diabetes in Pregnancy	50 questions
Case 5	Antepartum Haemorrhage: Placenta Praevia and Abruption	50 questions
Case 6	Preterm Labour and PPROM	50 questions
Case 7	Breech Presentation	50 questions
Case 8	Twin Pregnancy	50 questions
Case 9	Previous Caesarean Section	50 questions
Case 10	Rhesus Negative Pregnancy	50 questions
Case 11	Fetal Growth Restriction	50 questions
Case 12	Prolonged Pregnancy and Induction of Labour	50 questions
Case 13	Postpartum Haemorrhage	50 questions
Case 14	Heart Disease in Pregnancy	50 questions

CASE 1

Normal Pregnancy and Antenatal Booking

> **Typical exam case:** A 25-year-old primigravida at 12 weeks attends for her booking visit. She has no medical problems. Take an obstetric history, examine her and plan her antenatal care.

HISTORY AND EXAMINATION

Q1. How do you calculate the expected date of delivery?

By Naegele's rule: add 9 months and 7 days to the first day of the last menstrual period, assuming a regular 28-day cycle. The dating scan at 11–14 weeks is more accurate and is preferred.

Q2. What do the terms gravida and para mean?

Gravida is the total number of pregnancies, including the current one. Para is the number of births after 24 weeks, live or stillborn. Losses before 24 weeks are written after a plus sign, e.g. G3 P1+1.

Q3. What are the key parts of an obstetric history?

Presenting pregnancy (LMP, EDD, symptoms), past obstetric history, gynaecological history (smears, contraception), past medical and surgical history, drugs and allergies, family history, and social history including smoking, alcohol and domestic abuse.

Q4. Why do you ask about previous pregnancies in detail?

Previous complications such as pre-eclampsia, preterm birth, caesarean section, PPH or stillbirth increase risk in this pregnancy and change the plan of care.

Q5. What is the fundal height at 12, 20 and 36 weeks?

At 12 weeks the fundus is just palpable above the symphysis; at 20 weeks it is at the umbilicus; at 36 weeks it is at the xiphisternum.

Q6. How do you measure symphysis–fundal height?

With a tape measure from the top of the symphysis pubis to the top of the fundus, blank side up to avoid bias. From 24 weeks, the measurement in cm should equal the gestation in weeks ±2 cm. It is plotted on a customised chart.

Q7. What are the Leopold manoeuvres?

Four steps of abdominal palpation: fundal grip (what is in the fundus), lateral grip (where the back lies), Pawlik's grip (presenting part), and pelvic grip (engagement).

Q8. What do you mean by lie, presentation and position?

Lie is the relation of the fetal long axis to the mother's long axis. Presentation is the part of the fetus over the pelvic inlet. Position is the relation of the denominator (e.g. occiput) to the maternal pelvis.

Q9. When is the head said to be engaged?

When the widest diameter (biparietal) has passed through the pelvic brim. Clinically, when 2/5 or less of the head is palpable above the brim.

Q10. What normal physical signs of pregnancy might you see?

Linea nigra, striae gravidarum, chloasma, darkened areolae, Montgomery's tubercles, mild ankle oedema and a soft ejection systolic murmur.

PHYSIOLOGY AND DIAGNOSIS

Q11. How is pregnancy confirmed?

> A urine or serum hCG test, confirmed by ultrasound showing an intrauterine gestational sac and fetal heart activity.

Q12. When can the fetal heart be seen on transvaginal ultrasound?

> From about 6 weeks of gestation.

Q13. What happens to cardiac output in pregnancy?

> It rises by about 40% by 20–24 weeks, due to higher stroke volume and heart rate.

Q14. Why does haemoglobin fall in pregnancy?

> Plasma volume rises by about 50% while red cell mass rises by only 20–30%, so there is haemodilution (physiological anaemia).

Q15. What is the cut-off for anaemia in pregnancy?

> Hb below 110 g/L in the first trimester, below 105 g/L in the second and third trimesters, and below 100 g/L after delivery.

Q16. Why is pregnancy a hypercoagulable state?

> Clotting factors (fibrinogen, VII, VIII, X) rise, fibrinolysis and protein S fall, and the uterus causes venous stasis. This protects against haemorrhage at delivery but raises VTE risk.

Q17. What is supine hypotensive syndrome?

> In late pregnancy, lying flat lets the uterus compress the inferior vena cava, lowering venous return and BP. Turning the woman to her left side relieves it.

Q18. When does a primigravida usually first feel fetal movements?

> At about 18–20 weeks; a multiparous woman may feel them from about 16 weeks.

INVESTIGATIONS AND SCREENING

Q19. What blood tests are done at booking?

> Full blood count, blood group and red cell antibodies, and screening for HIV, hepatitis B and syphilis. Sickle cell and thalassaemia screening is offered.

Q20. Why do we test urine at booking?

> Urine culture detects asymptomatic bacteriuria, which is treated to prevent pyelonephritis and preterm birth. Dipstick checks protein as a baseline.

Q21. When should booking take place?

> Ideally by 10 weeks, so screening and the dating scan can be arranged in time.

Q22. What are the two routine ultrasound scans in pregnancy?

> The dating scan at 11–14 weeks (with nuchal translucency if wanted) and the fetal anomaly scan at 18–20+6 weeks.

Q23. How is the pregnancy dated on ultrasound?

> By crown–rump length between 45 and 84 mm (about 11+2 to 14+1 weeks). After this, head circumference is used.

Q24. What is the combined test?

> A screening test for Down syndrome at 11–14 weeks using maternal age, nuchal translucency, PAPP-A and free beta-hCG.

Q25. What is the role of NIPT?

> Cell-free fetal DNA in maternal blood screens for trisomies 21, 18 and 13 with high accuracy. It is offered after a higher-chance screening result; a positive result needs confirmation by invasive testing.

Q26. What are the diagnostic invasive tests for chromosomal problems?

> Chorionic villus sampling from 11 weeks and amniocentesis from 15 weeks. Each carries a small miscarriage risk (about 0.5%).

Q27. Why is thalassaemia screening especially important in Pakistan?

> Beta-thalassaemia carrier rates are high. If the mother is a carrier, the partner is tested; if both are carriers, prenatal diagnosis is offered.

Q28. Which women need a glucose tolerance test?

> Women with risk factors such as BMI above 30, previous GDM, previous baby over 4.5 kg, family history of diabetes, or high-risk ethnic origin (South Asian). A 75 g OGTT is done at 24–28 weeks (earlier if previous GDM).

MANAGEMENT AND ADVICE

Q29. How many antenatal visits does a healthy first-time mother have?

> NICE advises about 10 appointments for a nulliparous woman with an uncomplicated pregnancy and about 7 for a parous woman.

Q30. What is checked at each routine antenatal visit?

> Blood pressure, urine for protein, fundal height from 24 weeks, fetal movements and heart rate where appropriate, and any symptoms or concerns.

Q31. What folic acid dose would you advise?

> 400 micrograms daily from before conception until 12 weeks. 5 mg is advised for higher-risk women (BMI 30 or more, diabetes, antiepileptic drugs, previous neural tube defect, sickle cell or thalassaemia).

Q32. Which other supplement is recommended for all pregnant women?

> Vitamin D 10 micrograms daily throughout pregnancy and breastfeeding.

Q33. What dietary advice do you give?

> Avoid unpasteurised milk, soft mould-ripened cheese, pâté, liver and raw or undercooked meat and eggs. Limit caffeine to 200 mg a day. Avoid alcohol completely.

Q34. Which women should take low-dose aspirin?

> Women at high risk of pre-eclampsia (one high-risk factor or two moderate risk factors) should take 75–150 mg aspirin daily from 12 weeks until birth.

Q35. Which vaccines are recommended in pregnancy?

> Pertussis (from 16 weeks), inactivated influenza (any trimester), COVID-19 and RSV (from 28 weeks) where available. Live vaccines such as MMR are avoided.

Q36. What advice do you give about smoking?

> Smoking raises the risk of miscarriage, growth restriction, preterm birth, stillbirth and sudden infant death. Refer to stop-smoking services; nicotine replacement can be used.

Q37. Is exercise safe in pregnancy?

> Yes. Moderate exercise such as walking and swimming is encouraged. Contact sports and scuba diving should be avoided.

Q38. Is air travel safe in pregnancy?

> Generally yes up to about 36 weeks in a singleton pregnancy. Advise mobility, fluids and compression stockings on long flights.

Q39. What is anti-D prophylaxis and who gets it?

> Routine antenatal anti-D is given to RhD-negative women without antibodies at about 28 weeks (or 28 and 34 weeks) to prevent rhesus sensitisation.

Q40. How do you manage reduced fetal movements after 24 weeks?

> Same-day assessment: check the fetal heart, do a CTG from 28 weeks, and arrange an ultrasound if risk factors or concerns.

Q41. When is a pregnancy considered post-term and what is offered?

> Beyond 42 weeks. NICE offers induction of labour from 41 weeks to reduce stillbirth risk.

Q42. Which minor symptoms of pregnancy are common?

> Nausea and vomiting, heartburn, constipation, backache, pelvic girdle pain, carpal tunnel syndrome, varicose veins and leg cramps.

Q43. How do you manage nausea and vomiting in early pregnancy?

> Reassurance, small frequent meals and ginger. If more severe, antiemetics such as cyclizine or promethazine; admit if there is dehydration or ketosis (hyperemesis gravidarum).

COUNSELLING AND RECENT ADVANCES

Q44. Why is domestic abuse screening done in pregnancy?

> Abuse often starts or worsens in pregnancy and is linked to miscarriage, preterm birth and maternal death. Every woman should be asked in private at least once.

Q45. What is continuity of carer?

> Care from the same midwife or small team throughout pregnancy, birth and after birth. It reduces preterm birth and improves satisfaction.

Q46. Why is a woman's mental health asked about at booking?

> Past or current mental illness, especially bipolar disorder or severe depression, raises the risk of relapse and postpartum psychosis. Early referral to perinatal mental health services is needed.

Q47. What is the role of carbon monoxide testing at booking?

> It detects smoking and exposure to carbon monoxide, and helps refer women to stop-smoking support.

Q48. What is the Saving Babies' Lives care bundle?

> A UK bundle to reduce stillbirth: reducing smoking, detecting growth restriction, raising awareness of reduced fetal movements, effective fetal monitoring in labour, and reducing preterm birth.

Q49. What is meant by personalised care planning?

> Care tailored to the woman's risks and wishes, recorded in her notes, including her birth preferences and place of birth.

Q50. What is the main message for a low-risk woman about place of birth?

> For a low-risk multiparous woman, home or a midwife-led unit is particularly suitable. For a low-risk first-time mother, a midwife-led unit is suitable; home birth carries a small extra risk to the baby.

CASE 2

Anaemia in Pregnancy

> **Typical exam case:** A 28-year-old G3 P2 at 30 weeks complains of tiredness and breathlessness on exertion. She looks pale. Her haemoglobin is 82 g/L.

HISTORY AND EXAMINATION

Q1. What symptoms would you ask about?

> Tiredness, breathlessness, palpitations, dizziness, headache, reduced exercise tolerance, and pica (craving for non-food items like ice or clay).

Q2. What risk factors for anaemia would you look for in the history?

> Short interval between pregnancies, multiple pregnancy, poor diet, heavy periods before pregnancy, worm infestation, chronic disease, bleeding, and a family history of thalassaemia or sickle cell.

Q3. Why is dietary history important?

> Iron, folate and vitamin B12 come from diet. Vegetarian diets may lack iron and B12; tea with meals reduces iron absorption.

Q4. What signs would you look for on examination?

> Pallor of conjunctiva, palms and nail beds, koilonychia, angular stomatitis, glossitis, tachycardia, a flow murmur, and signs of heart failure in severe cases.

Q5. Why do you examine the abdomen for an enlarged spleen?

> Splenomegaly suggests haemolytic anaemia, thalassaemia, chronic malaria or other haematological disease.

Q6. What obstetric findings would you check?

> Fundal height for growth restriction, fetal lie and presentation, and fetal heart.

Q7. Why is anaemia common in Pakistan?

> Poor nutrition, frequent pregnancies, worm infestation, malaria and high thalassaemia carrier rates.

Q8. How does anaemia affect the mother in labour?

> She tolerates blood loss poorly, so even a normal PPH can lead to shock, need for transfusion and death.

Q9. Which past obstetric events increase the risk now?

> A previous PPH, previous anaemia, or a short interval since the last delivery.

Q10. What social factors would you ask about?

> Income and access to food, family support, and any barriers to attending antenatal care.

DIAGNOSIS

Q11. How is anaemia defined in pregnancy?

> Hb below 110 g/L in the first trimester, below 105 g/L in the second and third trimesters, and below 100 g/L after delivery.

Q12. What is the most common cause of anaemia in pregnancy?

> Iron deficiency anaemia.

Q13. What is physiological anaemia of pregnancy?

> A fall in Hb because plasma volume rises more than red cell mass. Indices such as MCV and ferritin remain normal.

Q14. What are the types of anaemia by MCV?

> Microcytic (iron deficiency, thalassaemia trait), normocytic (chronic disease, acute blood loss, early iron deficiency) and macrocytic (folate or B12 deficiency).

Q15. How would you classify anaemia severity?

> Mild (Hb 90–104 g/L), moderate (70–89 g/L) and severe (below 70 g/L), using WHO-based grading.

Q16. How do you tell iron deficiency from thalassaemia trait?

> Both are microcytic. Iron deficiency has low ferritin; thalassaemia trait has normal or high ferritin, a raised red cell count and raised HbA2 on electrophoresis.

Q17. What causes folate deficiency in pregnancy?

> Increased demand, poor diet, multiple pregnancy, haemolytic anaemia and antiepileptic drugs such as phenytoin.

Q18. What other causes of anaemia must be excluded?

> Bleeding (e.g. haemorrhoids), haemoglobinopathies, malaria, hookworm, chronic kidney disease and bone marrow problems.

INVESTIGATIONS

Q19. What is the first investigation?

> Full blood count with red cell indices (MCV, MCH).

Q20. What is the best single test for iron deficiency?

> Serum ferritin. A level below 30 µg/L in pregnancy suggests iron deficiency.

Q21. Why can ferritin be misleading?

> It is an acute-phase protein and rises with infection or inflammation.

Q22. When would you do haemoglobin electrophoresis?

> When the MCV is low and ferritin is normal, or the woman is from a high-risk population, to look for thalassaemia or sickle cell.

Q23. What does a blood film show in iron deficiency?

> Microcytic hypochromic red cells, pencil cells and anisocytosis.

Q24. What tests would you do if MCV is high?

> Serum B12 and red cell folate, and thyroid function.

Q25. Why might you check stool?

> For ova and cysts (hookworm) and occult blood.

Q26. What is a trial of iron?

> Starting oral iron and checking the Hb in 2–3 weeks. A rise confirms iron deficiency and avoids delay in treatment.

Q27. What would you check if the Hb does not rise on iron?

> Compliance, side effects, ongoing blood loss, malabsorption, wrong diagnosis (e.g. thalassaemia) and coexisting folate deficiency.

Q28. Which fetal investigations may be useful?

> A growth scan if the anaemia is severe or fundal height is small, as severe anaemia is linked to growth restriction.

MANAGEMENT

Q29. How do you treat iron deficiency anaemia in pregnancy?

> Oral iron (e.g. ferrous sulfate 200 mg once or twice daily, or alternate days), with advice on diet. Recheck Hb in 2–3 weeks.

Q30. How should oral iron be taken?

> On an empty stomach or with vitamin C (orange juice). Avoid taking it with tea, coffee, milk or antacids.

Q31. What are the side effects of oral iron?

> Nausea, abdominal pain, constipation, diarrhoea and black stools.

Q32. When is intravenous iron indicated?

> When oral iron is not tolerated or does not work, when compliance is poor, or when the woman presents late in pregnancy with significant anaemia. It is given after the first trimester.

Q33. Name an intravenous iron preparation.

> Ferric carboxymaltose or iron sucrose.

Q34. When is blood transfusion considered in pregnancy?

> For severe symptomatic anaemia, heart failure, ongoing bleeding, or very low Hb close to delivery. It is not first line for iron deficiency.

Q35. How long should oral iron be continued?

> For 3 months after the Hb is normal to build up stores, and at least 6 weeks after delivery.

Q36. What would you do with Hb 82 g/L at 36 weeks?

> Consider intravenous iron, as there is not enough time for oral iron to correct it before delivery.

Q37. What precautions would you take for her delivery?

> Deliver in a hospital with blood available, group and save, IV access, and active management of the third stage.

Q38. How do you prevent PPH in an anaemic woman?

> Active management of the third stage with oxytocin, consider tranexamic acid, and avoid prolonged labour.

Q39. How do you treat folate deficiency?

> Folic acid 5 mg daily.

Q40. How do you treat vitamin B12 deficiency?

> Intramuscular hydroxocobalamin, then oral or IM maintenance.

Q41. How is a woman with beta-thalassaemia trait managed?

> Do not give iron unless ferritin is low. Give 5 mg folic acid. Test the partner and offer prenatal diagnosis if both are carriers.

Q42. How is hookworm treated in pregnancy?

> Mebendazole or albendazole is usually avoided in the first trimester; treatment can be given in the second or third trimester.

Q43. What is routine iron supplementation policy in the UK?

> Not routine for all women; given if Hb is low. In countries with high anaemia rates, WHO recommends routine iron and folic acid.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal risks of anaemia?

> Fatigue, poor tolerance of blood loss, infection, heart failure, need for transfusion, postnatal depression and maternal death.

Q45. What are the fetal risks of severe anaemia?

> Preterm birth, low birth weight, growth restriction and perinatal death.

Q46. Why is iron deficiency in the mother bad for the baby later?

> Babies are born with lower iron stores and are at risk of anaemia and poor neurodevelopment.

Q47. What advice would you give about the next pregnancy?

> Space pregnancies by at least 18 months, correct anaemia before conception, take folic acid and eat an iron-rich diet.

Q48. Which foods are rich in iron?

> Red meat, liver (avoided in pregnancy), green leafy vegetables, lentils, beans, fortified cereals and dried fruit.

Q49. What is the WHO 'anaemia in pregnancy' message for low-income countries?

> Daily iron and folic acid for all pregnant women, deworming, malaria prevention and better nutrition.

Q50. What is a recent advance in treating anaemia in pregnancy?

> Single-dose intravenous iron such as ferric carboxymaltose is now used earlier in pregnancy in the second and third trimesters, giving faster correction than oral iron with fewer transfusions.

CASE 3

Pre-eclampsia and Eclampsia

> **Typical exam case:** A 22-year-old primigravida at 34 weeks has a BP of 160/105 mmHg, 2+ protein on dipstick, headache and swelling of the face and hands.

HISTORY AND EXAMINATION

Q1. What symptoms of severe pre-eclampsia would you ask about?

> Severe headache, visual disturbance (flashing lights, blurring), epigastric or right upper quadrant pain, vomiting, sudden swelling and reduced fetal movements.

Q2. Which risk factors would you ask about?

> First pregnancy, previous pre-eclampsia, chronic hypertension, kidney disease, diabetes, autoimmune disease (SLE, antiphospholipid syndrome), age 40 or more, BMI 35 or more, multiple pregnancy, family history and long pregnancy interval.

Q3. How should BP be measured in pregnancy?

> Seated, with the correct cuff size, arm at heart level, using Korotkoff V for diastolic.

Q4. What signs would you look for on general examination?

> BP, oedema of the face and hands, brisk reflexes and clonus, epigastric tenderness, and signs of pulmonary oedema.

Q5. What does clonus suggest?

> Cerebral irritability and an increased risk of eclampsia.

Q6. What would you check on abdominal examination?

> Fundal height (for growth restriction), uterine tenderness (for abruption), liquor volume clinically, and the fetal heart.

Q7. Why is right upper quadrant pain important?

> It suggests liver involvement, such as HELLP syndrome or liver capsule distension, which may precede rupture.

Q8. What is the significance of visual symptoms?

> They suggest cerebral involvement and impending eclampsia, or rarely cortical blindness or retinal detachment.

Q9. Why is urine output monitored?

> Pre-eclampsia can cause acute kidney injury; oliguria suggests worsening disease.

Q10. Why is fundoscopy useful?

> To look for retinal oedema, haemorrhages or papilloedema in severe hypertension.

DIAGNOSIS

Q11. How is pre-eclampsia defined?

> New hypertension (BP 140/90 or more) after 20 weeks with significant proteinuria, or with other maternal organ dysfunction (kidney, liver, neurological, haematological) or uteroplacental dysfunction (FGR).

Q12. What is significant proteinuria?

> A urine protein:creatinine ratio of 30 mg/mmol or more, or albumin:creatinine ratio of 8 mg/mmol or more.

Q13. What is gestational hypertension?

> New hypertension after 20 weeks without proteinuria or other features of pre-eclampsia.

Q14. What is chronic hypertension?

> Hypertension present before pregnancy or before 20 weeks.

Q15. What BP defines severe hypertension in pregnancy?

> Systolic 160 mmHg or more, or diastolic 110 mmHg or more.

Q16. What is HELLP syndrome?

> Haemolysis, Elevated Liver enzymes and Low Platelets, a severe form of pre-eclampsia.

Q17. What is eclampsia?

> Generalised tonic-clonic seizures in a woman with pre-eclampsia, with no other cause.

Q18. What is the basic pathophysiology of pre-eclampsia?

> Poor trophoblast invasion of the spiral arteries leads to placental ischaemia. The placenta releases factors (high sFlt-1, low PlGF) that damage the maternal endothelium, causing widespread organ dysfunction.

INVESTIGATIONS

Q19. What blood tests would you send?

> Full blood count (platelets, Hb), urea, creatinine and electrolytes, liver enzymes (ALT/AST), and LDH and blood film if HELLP is suspected.

Q20. How would you quantify proteinuria?

> Urine protein:creatinine ratio on a spot sample.

Q21. When would you check clotting?

> If platelets are below 100 × 10^9/L, in suspected abruption, or before regional anaesthesia when platelets are low.

Q22. What is the role of placental growth factor (PlGF) testing?

> A low PlGF between 20 and 35 weeks helps diagnose or rule out pre-eclampsia in suspected cases and helps decide on admission.

Q23. What is the sFlt-1/PlGF ratio used for?

> A ratio below 38 has a high negative predictive value, so pre-eclampsia within a week is unlikely.

Q24. What fetal assessment is needed?

> Ultrasound for growth and liquor volume, umbilical artery Doppler, and CTG.

Q25. How often are bloods repeated in admitted pre-eclampsia?

> At least twice a week, or more often if severe or changing.

Q26. What does a rising creatinine suggest?

> Worsening kidney involvement; creatinine above 90 µmol/L in pregnancy is abnormal.

Q27. What does a rising LDH and falling platelets suggest?

> Developing HELLP syndrome.

Q28. What imaging is needed if she has a seizure with focal signs?

> CT or MRI of the brain to exclude haemorrhage or other causes; PRES (posterior reversible encephalopathy) may be seen.

MANAGEMENT

Q29. Where should a woman with severe pre-eclampsia be managed?

> In hospital, on the labour ward or a high-dependency area, with senior obstetric, anaesthetic and midwifery input.

Q30. What is the first-line antihypertensive in pregnancy?

> Labetalol, unless the woman has asthma. Nifedipine or methyldopa are alternatives.

Q31. How is severe hypertension treated acutely?

> Oral labetalol or nifedipine, or IV labetalol or hydralazine, aiming for BP below 150/80–100 mmHg.

Q32. What is the target BP?

> 135/85 mmHg or below in treated women (NICE).

Q33. Why is magnesium sulfate given?

> To prevent and treat eclamptic seizures. It is more effective than diazepam or phenytoin (Collaborative Eclampsia Trial, Magpie trial).

Q34. What is the regimen for magnesium sulfate?

> 4 g IV loading dose over 5–15 minutes, then 1 g/hour infusion for 24 hours after the last seizure or delivery.

Q35. How do you monitor for magnesium toxicity?

> Check patellar reflexes, respiratory rate (above 12/min), urine output and oxygen saturation. Check serum magnesium if oliguric.

Q36. What is the antidote to magnesium toxicity?

> 10 mL of 10% calcium gluconate IV over 10 minutes.

Q37. Why is fluid restricted in severe pre-eclampsia?

> Leaky capillaries make pulmonary oedema likely. Fluids are limited to about 80 mL/hour.

Q38. What is the definitive treatment of pre-eclampsia?

> Delivery of the placenta.

Q39. When is delivery recommended?

> At 37 weeks for pre-eclampsia, or earlier for uncontrolled BP, worsening bloods, eclampsia, HELLP, pulmonary oedema, abruption or fetal compromise.

Q40. Why give corticosteroids before preterm delivery?

> To improve fetal lung maturity and reduce respiratory distress syndrome.

Q41. What is the first aid for an eclamptic fit?

> Call for help, left lateral position, protect the airway, give oxygen, then give magnesium sulfate. Control BP and plan delivery once she is stable.

Q42. Which uterotonic must be avoided in the third stage?

> Ergometrine (and Syntometrine), as it raises BP. Oxytocin alone is used.

Q43. Which antihypertensive is preferred after delivery in breastfeeding women?

> Enalapril (or nifedipine or amlodipine). Labetalol can also be continued.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal complications?

> Eclampsia, stroke, HELLP, DIC, acute kidney injury, pulmonary oedema, liver rupture, placental abruption and death.

Q45. What are the fetal complications?

> Growth restriction, oligohydramnios, preterm birth (often iatrogenic), placental abruption and stillbirth.

Q46. How can pre-eclampsia be prevented in the next pregnancy?

> Low-dose aspirin 75–150 mg from 12 weeks until birth, and calcium if dietary intake is low.

Q47. What is her risk of pre-eclampsia in a future pregnancy?

> About 1 in 6 overall, and up to 1 in 4 if she had severe or early-onset disease.

Q48. What long-term risks does she face?

> Higher lifelong risk of hypertension, heart disease, stroke and kidney disease. She needs regular BP checks.

Q49. When can postnatal antihypertensives be stopped?

> When BP is consistently below 140/90; review at 2 weeks and at the 6–8 week postnatal check.

Q50. What is a recent advance in pre-eclampsia care?

> PlGF-based testing (and sFlt-1/PlGF ratio) to diagnose or rule out pre-eclampsia, and first-trimester screening combining maternal factors, BP, uterine artery Doppler and PlGF to target aspirin.

CASE 4

Diabetes in Pregnancy

Typical exam case: A 32-year-old G2 P1 of South Asian origin at 28 weeks has a 75 g OGTT with fasting glucose 6.0 mmol/L and 2-hour value 8.9 mmol/L. Her last baby weighed 4.3 kg.

HISTORY AND EXAMINATION

Q1. What are the risk factors for gestational diabetes?

BMI above 30, previous GDM, previous baby weighing 4.5 kg or more, first-degree relative with diabetes, and high-risk ethnic origin (South Asian, Black Caribbean, Middle Eastern).

Q2. What symptoms of hyperglycaemia would you ask about?

Thirst, polyuria, tiredness, recurrent thrush and urinary infections. Most women with GDM have no symptoms.

Q3. Which past obstetric events suggest undiagnosed diabetes?

Previous macrosomic baby, shoulder dystocia, unexplained stillbirth, polyhydramnios or congenital anomaly.

Q4. What would you look for on abdominal examination?

A fundal height larger than dates, polyhydramnios and fetal size.

Q5. Why ask about family history?

A first-degree relative with type 2 diabetes increases her risk.

Q6. What should you ask in a woman with pre-existing diabetes?

Type and duration, current treatment, HbA1c, hypoglycaemia awareness, and complications such as retinopathy, nephropathy and heart disease.

Q7. What are the features of diabetic ketoacidosis in pregnancy?

Vomiting, abdominal pain, dehydration, rapid deep breathing and drowsiness. It can occur at lower glucose levels in pregnancy.

Q8. Why is BMI important?

Obesity increases insulin resistance and the risk of GDM, pre-eclampsia, macrosomia and caesarean section.

Q9. Why ask about her diet and activity?

Lifestyle changes are the first step of treatment.

Q10. Why ask about steroid use?

Corticosteroids, including those given for fetal lung maturity, raise blood glucose.

DIAGNOSIS

Q11. What are the NICE diagnostic thresholds for GDM?

Fasting plasma glucose 5.6 mmol/L or more, or 2-hour glucose 7.8 mmol/L or more on a 75 g OGTT (the 5-6-7-8 rule).

Q12. Does this woman have GDM?

Yes. Both her fasting (6.0) and 2-hour (8.9) values are above the thresholds.

Q13. When is the OGTT done?

> At 24–28 weeks for women with risk factors. Women with previous GDM have early self-monitoring or an OGTT soon after booking, repeated at 24–28 weeks if normal.

Q14. Why does GDM develop in the second half of pregnancy?

> Placental hormones (human placental lactogen, progesterone, cortisol) cause insulin resistance. Women who cannot increase insulin output enough become hyperglycaemic.

Q15. What is pre-existing diabetes?

> Type 1 or type 2 diabetes present before pregnancy.

Q16. What does a fasting glucose of 7.0 mmol/L or more at diagnosis suggest?

> Possible undiagnosed pre-existing diabetes; it also means insulin should be started at once.

Q17. Is glycosuria diagnostic of diabetes in pregnancy?

> No. The renal threshold for glucose falls in pregnancy, so glycosuria is common. Persistent glycosuria needs an OGTT.

Q18. What is MODY?

> Maturity-onset diabetes of the young, a monogenic form; suspect it with strong family history, young age and no obesity.

INVESTIGATIONS

Q19. How is blood glucose monitored in GDM?

> Capillary glucose testing: fasting and 1 hour after each meal (more often if on insulin).

Q20. What are the NICE glucose targets in pregnancy?

> Fasting below 5.3 mmol/L, 1 hour after meals below 7.8 mmol/L, or 2 hours after meals below 6.4 mmol/L.

Q21. What fetal scans are done?

> Growth and liquor scans every 4 weeks from 28 to 36 weeks in pre-existing diabetes, and as needed in GDM.

Q22. What extra scans are needed in pre-existing diabetes?

> A detailed anomaly scan including fetal heart views at 20 weeks, and retinal screening at booking and 28 weeks.

Q23. Why check HbA1c in pre-existing diabetes?

> It reflects control around conception; levels above 48 mmol/mol raise the risk of anomalies, and above 86 mmol/mol pregnancy should be avoided.

Q24. Why check renal function?

> Diabetic nephropathy worsens in pregnancy and increases pre-eclampsia risk.

Q25. What does polyhydramnios suggest in a diabetic pregnancy?

> Poor glucose control with fetal polyuria, or a fetal anomaly.

Q26. Why check ketones?

> If glucose is high or she is unwell, ketones help diagnose DKA early.

Q27. What is the test after delivery for women with GDM?

> Fasting plasma glucose or HbA1c at 6–13 weeks after birth, then HbA1c every year.

Q28. Why is fetal growth assessed by ultrasound and not fundal height alone?

> Fundal height is unreliable with polyhydramnios and obesity; macrosomia may be missed.

MANAGEMENT

Q29. What is the first step in treating GDM?

> Diet and exercise advice. If targets are not met in 1–2 weeks, start metformin.

Q30. What dietary advice is given?

> Low glycaemic index foods, regular meals, reduced simple sugars and portion control.

Q31. When is insulin started?

> If metformin is not tolerated or does not achieve targets, or at once if fasting glucose is 7.0 mmol/L or more, or 6.0–6.9 with complications such as macrosomia or polyhydramnios.

Q32. Is metformin safe in pregnancy?

> Yes. It crosses the placenta but no major harms are shown; it reduces weight gain and hypoglycaemia compared with insulin.

Q33. Which oral drug other than metformin may be used?

> Glibenclamide, if metformin is not suitable and insulin is declined.

Q34. When should a woman with GDM on diet only be delivered?

> No later than 40+6 weeks.

Q35. When should a woman with GDM on medication be delivered?

> Offer delivery between 37+0 and 38+6 weeks.

Q36. When should a woman with pre-existing diabetes be delivered?

> Offer delivery between 37+0 and 38+6 weeks, earlier if complications.

Q37. When is caesarean section considered?

> For obstetric reasons, or an estimated fetal weight above 4.5 kg due to shoulder dystocia risk.

Q38. How is glucose managed in labour?

> Capillary glucose hourly, aiming for 4–7 mmol/L. A variable-rate insulin infusion with dextrose is used if needed, especially in type 1 diabetes.

Q39. What happens to treatment for GDM after delivery?

> Stop all glucose-lowering medicines straight after birth.

Q40. What happens to insulin in pre-existing diabetes after delivery?

> Reduce insulin to pre-pregnancy doses straight away, because insulin needs fall sharply, with risk of hypoglycaemia.

Q41. How is the baby cared for after birth?

> Early feeding within 30 minutes, then every 2–3 hours, with blood glucose checks at 2–4 hours to detect hypoglycaemia.

Q42. What pre-conception advice is given to women with pre-existing diabetes?

> Aim for HbA1c below 48 mmol/mol, take 5 mg folic acid, stop ACE inhibitors and statins, and review retinopathy and kidney function.

Q43. What is the role of aspirin in diabetic pregnancy?

> Pre-existing diabetes is a high-risk factor for pre-eclampsia, so aspirin 75–150 mg is given from 12 weeks.

COMPLICATIONS AND COUNSELLING

Q44. What are the fetal risks of pre-existing diabetes?

> Miscarriage, congenital anomalies (heart and neural tube, caudal regression), macrosomia, polyhydramnios, stillbirth and preterm birth.

Q45. What are the neonatal risks?

> Hypoglycaemia, respiratory distress, jaundice, polycythaemia, hypocalcaemia and shoulder dystocia injuries.

Q46. Why do babies of diabetic mothers become hypoglycaemic?

> Fetal hyperinsulinaemia from high maternal glucose continues after birth when the glucose supply stops.

Q47. What are the maternal risks?

> Pre-eclampsia, infection, caesarean section, worsening retinopathy and nephropathy, DKA and hypoglycaemia.

Q48. What is her risk of type 2 diabetes after GDM?

> About 50% within 5–10 years. She needs annual HbA1c, weight control and a healthy lifestyle.

Q49. What is her risk of GDM in the next pregnancy?

> High, about 30–50%. She should have early testing next time.

Q50. What are recent advances in diabetic pregnancy care?

> Continuous glucose monitoring for women with type 1 diabetes (improves neonatal outcomes) and hybrid closed-loop insulin systems.

CASE 5

Antepartum Haemorrhage: Placenta Praevia and Abruption

> **Typical exam case:** A 30-year-old G3 P2 at 34 weeks presents with sudden painless bright red vaginal bleeding of about 300 mL. She has had two previous caesarean sections. The uterus is soft and the fetus is in a transverse lie.

HISTORY AND EXAMINATION

Q1. How do you define antepartum haemorrhage?

Bleeding from or into the genital tract from 24 weeks of pregnancy until the birth of the baby.

Q2. What are the main causes of APH?

Placenta praevia, placental abruption, and local causes (cervical ectropion, polyps, infection, cancer). Rare causes are vasa praevia and uterine rupture. Some cases are unexplained.

Q3. What features of the bleeding would you ask about?

Amount, colour, onset, pain, contractions, trigger (sex, trauma), previous episodes, fetal movements and leakage of fluid.

Q4. How does praevia bleeding differ from abruption?

Praevia: painless, bright red, recurrent, soft uterus, malpresentation. Abruption: painful, dark blood, tense tender uterus, fetal distress, and shock out of proportion to visible loss.

Q5. What is the first step on examination?

Assess maternal condition: pulse, BP, respiratory rate, colour and level of consciousness, and resuscitate if needed.

Q6. What would you find on abdominal examination in praevia?

Soft, non-tender uterus, high presenting part or malpresentation, and usually a normal fetal heart.

Q7. Which examination must be avoided?

Digital vaginal examination, until placenta praevia has been excluded by ultrasound, as it can cause massive bleeding.

Q8. Is speculum examination allowed?

Yes, gently, to assess bleeding and look for local causes or ruptured membranes.

Q9. Why ask about previous caesarean sections?

They increase the risk of placenta praevia and placenta accreta spectrum.

Q10. What is the blood group concern in APH?

RhD-negative women need anti-D and a Kleihauer test after bleeding from 20 weeks.

DIAGNOSIS

Q11. What is placenta praevia?

A placenta implanted partly or fully in the lower uterine segment.

Q12. How is placenta praevia now classified?

As placenta praevia (covering the internal os) or low-lying placenta (edge within 20 mm of the internal os).

Q13. What are the risk factors for placenta praevia?

Previous caesarean section, previous praevia, multiparity, older age, smoking, multiple pregnancy and assisted conception.

Q14. What is placental abruption?

> Separation of a normally situated placenta from the uterine wall before birth.

Q15. What are the risk factors for abruption?

> Pre-eclampsia and hypertension, previous abruption, smoking, cocaine, trauma, PPROM, multiple pregnancy and polyhydramnios.

Q16. What is a concealed abruption?

> Bleeding stays behind the placenta, so there is little or no visible bleeding but severe pain, a tense uterus and shock.

Q17. What is a Couvelaire uterus?

> Blood tracks into the myometrium in severe abruption, giving a bruised purple uterus that may contract poorly.

Q18. What is placenta accreta spectrum?

> Abnormal adherence of the placenta: accreta (attached to myometrium), increta (into myometrium) and percreta (through the serosa, possibly into bladder).

INVESTIGATIONS

Q19. What bloods would you send?

> Full blood count, group and cross-match (4–6 units for major bleeding), clotting screen and fibrinogen, urea and electrolytes, and Kleihauer if RhD negative.

Q20. How is placenta praevia diagnosed?

> By ultrasound, ideally transvaginal, which is safe and more accurate.

Q21. What follow-up scan is done if the placenta is low at 20 weeks?

> A repeat (transvaginal) scan at 32 weeks; if still low, another at 36 weeks to plan delivery.

Q22. How is placenta accreta diagnosed?

> Specialist ultrasound (loss of the clear zone, lacunae, bladder wall interruption, increased vascularity), with MRI if needed.

Q23. Is ultrasound good for diagnosing abruption?

> No. It misses most cases. Abruption is a clinical diagnosis.

Q24. What is the role of the CTG?

> To assess fetal wellbeing and look for contractions or fetal compromise.

Q25. Why is a Kleihauer test done?

> To measure the size of fetomaternal haemorrhage and calculate the dose of anti-D in RhD-negative women.

Q26. Which test detects DIC?

> Platelets, PT, APTT and fibrinogen; low fibrinogen is an early warning sign.

Q27. What is the role of a urinary catheter?

> To monitor urine output in major haemorrhage and detect kidney injury.

Q28. Why is a growth scan done after recurrent or unexplained APH?

> Because unexplained APH is linked to growth restriction.

MANAGEMENT

Q29. What is the immediate management of major APH?

> Call for help, ABC, two large-bore cannulas, bloods and cross-match, IV fluids and blood, left lateral tilt, and continuous fetal monitoring.

Q30. How is placenta praevia managed if bleeding stops and she is stable at 34 weeks?

> Admit, give steroids for fetal lungs, cross-match blood, and observe. Delivery planned by caesarean.

Q31. When is planned caesarean done for uncomplicated praevia?

> Between 36+0 and 37+6 weeks.

Q32. When is earlier delivery considered in praevia?

> At 34+0 to 35+6 weeks for women with recurrent bleeding or other risk factors.

Q33. Who should be present at caesarean for praevia?

> A consultant obstetrician and consultant anaesthetist, especially for anterior praevia or suspected accreta.

Q34. How is suspected accreta spectrum managed?

> Planned delivery at 35+0 to 36+6 weeks in a specialist centre, with blood products, cell salvage, interventional radiology and possible caesarean hysterectomy.

Q35. What is the management of abruption with a live fetus and fetal distress?

> Resuscitate the mother and perform an emergency caesarean section.

Q36. What is the management of abruption with fetal death?

> Resuscitate, correct clotting, and aim for vaginal birth with amniotomy, as caesarean adds risk if coagulopathy is present.

Q37. How is mild abruption at 32 weeks managed if both are stable?

> Admit, give steroids, monitor closely, and plan delivery if bleeding or fetal concerns develop.

Q38. How is mild abruption at term managed?

> Delivery, usually by induction of labour.

Q39. What drug is given for bleeding in obstetric haemorrhage?

> Tranexamic acid 1 g IV.

Q40. What is the target fibrinogen in obstetric haemorrhage?

> Above 2 g/L; give cryoprecipitate or fibrinogen concentrate if lower.

Q41. What dose of anti-D is given after APH beyond 20 weeks?

> At least 500 IU within 72 hours, with more if the Kleihauer shows a large bleed.

Q42. Why is the lower segment prone to bleed after delivery of a praevia?

> It has less muscle and contracts poorly, so the placental bed keeps bleeding.

Q43. What surgical measures help with praevia bleeding at caesarean?

> Uterotonics, balloon tamponade, haemostatic sutures, uterine artery ligation, and caesarean hysterectomy if needed.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal complications of APH?

> Haemorrhagic shock, DIC, acute kidney injury, PPH, need for hysterectomy, Sheehan syndrome and death.

Q45. What are the fetal complications?

> Prematurity, hypoxia, growth restriction and stillbirth.

Q46. What advice is given to a woman with praevia managed at home?

> Live close to the hospital, have an adult with her, avoid intercourse, and attend at once with any bleeding or pain.

Q47. What is the recurrence risk of abruption?

> About 10% after one abruption, higher after two.

Q48. What advice for future pregnancy after praevia or caesarean?

> Higher risk of praevia and accreta; early scan to locate the placenta and deliver in a unit with full support.

Q49. Why must a Jehovah's Witness with praevia be planned for in advance?

> She may refuse blood, so haemoglobin is optimised early, cell salvage is discussed and a senior team plans delivery.

Q50. What is a recent advance in managing praevia and accreta?

> Specialist multidisciplinary accreta centres, standardised ultrasound reporting, intraoperative cell salvage and interventional radiology have reduced blood loss and maternal deaths.

CASE 6

Preterm Labour and PPROM

> **Typical exam case:** A 26-year-old G2 P1 at 31 weeks presents with a gush of clear fluid from the vagina 6 hours ago, followed by irregular tightenings. Her first baby was born at 33 weeks.

HISTORY AND EXAMINATION

Q1. How is preterm birth defined?

> Birth before 37+0 weeks. Extremely preterm is before 28 weeks, very preterm 28–31+6 weeks, and moderate to late preterm 32–36+6 weeks.

Q2. What is PPROM?

> Preterm prelabour rupture of membranes: rupture of membranes before 37 weeks and before labour starts.

Q3. What would you ask about the fluid loss?

> Time, amount, colour (clear, blood-stained, meconium), smell, ongoing leakage, and any fever or contractions.

Q4. What risk factors for preterm birth would you ask about?

> Previous preterm birth or mid-trimester loss, cervical surgery (LLETZ, cone), short cervix, multiple pregnancy, infection, smoking, polyhydramnios and uterine anomalies.

Q5. What is the strongest risk factor?

> A previous spontaneous preterm birth.

Q6. What signs of infection would you look for?

> Maternal fever, tachycardia, uterine tenderness, offensive discharge and fetal tachycardia.

Q7. What examination confirms PPROM?

> A sterile speculum examination showing pooling of amniotic fluid in the posterior fornix.

Q8. Why should digital vaginal examination be avoided in PPROM?

> It increases the risk of infection and shortens the time to labour. It is only done if active labour is suspected.

Q9. What would you check on abdominal palpation?

> Fundal height, presentation, contractions, uterine tenderness and the fetal heart.

Q10. What maternal observations are needed?

> Temperature, pulse, BP, respiratory rate, recorded on an obstetric early warning chart.

DIAGNOSIS

Q11. How is PPROM confirmed if pooling is not seen?

> A test of vaginal fluid for IGFBP-1 or PAMG-1 (e.g. Actim PROM, AmniSure).

Q12. Why is the nitrazine test not recommended?

> It has high false positive rates with blood, semen and infection.

Q13. How is preterm labour diagnosed?

> Regular painful contractions with cervical change.

Q14. What is the role of transvaginal cervical length in suspected preterm labour?

> From 30 weeks, a cervical length of 15 mm or less supports preterm labour; above 15 mm makes it unlikely.

Q15. What is the role of fetal fibronectin?

> If cervical length scanning is not available, a negative fetal fibronectin (below 50 ng/mL) makes delivery within 1–2 weeks unlikely.

Q16. What are the main causes of PPROM?

> Infection, cervical weakness, overdistension (twins, polyhydramnios), smoking and bleeding.

Q17. What is chorioamnionitis?

> Infection of the membranes and amniotic fluid, with fever, uterine tenderness, offensive discharge and fetal tachycardia.

Q18. What is the differential diagnosis of watery discharge?

> Urinary incontinence, vaginal discharge, and semen.

INVESTIGATIONS

Q19. Which swabs are taken?

> A high vaginal swab for culture, including group B streptococcus.

Q20. Which bloods are taken?

> Full blood count and CRP as a baseline for infection monitoring.

Q21. What fetal assessment is done?

> CTG and ultrasound for presentation, growth and liquor volume.

Q22. Is a single CRP diagnostic of chorioamnionitis?

> No. A rising trend helps, but diagnosis is mainly clinical.

Q23. Why is urine culture sent?

> Urinary infection can trigger preterm labour and should be treated.

Q24. What is the role of ultrasound for presentation?

> Breech presentation is more common in preterm labour and may change the mode of delivery.

Q25. How often are observations done in PPROM?

> At least every 4 hours, with temperature, pulse and CTG as part of infection surveillance.

Q26. When would you do an amniocentesis?

> Rarely; it may be used in research or selected cases to diagnose intra-amniotic infection.

Q27. What is the role of GBS status?

> If GBS positive, intrapartum antibiotics are needed, and from 34 weeks induction is offered.

Q28. How do you assess risk of preterm birth in a future pregnancy?

> Serial transvaginal cervical length scans from 16 to 24 weeks.

MANAGEMENT

Q29. Which antibiotic is given after PPROM?

> Erythromycin 250 mg four times a day for 10 days, or until labour.

Q30. Which antibiotic should be avoided in PPROM?

> Co-amoxiclav, because of a link with necrotising enterocolitis.

Q31. Why are corticosteroids given?

> To reduce respiratory distress syndrome, intraventricular haemorrhage and neonatal death.

Q32. What is the steroid regimen?

> Betamethasone 12 mg IM two doses 24 hours apart (or dexamethasone).

Q33. What gestation are steroids offered?

> Offered at 24+0 to 33+6 weeks; considered at 34+0 to 35+6 weeks.

Q34. Why is magnesium sulfate given?

> For fetal neuroprotection, to reduce cerebral palsy, when birth is expected within 24 hours from 24 to 29+6 weeks (considered up to 33+6).

Q35. Is tocolysis used in PPROM?

> No. It is not recommended in PPROM.

Q36. Which tocolytic is used in preterm labour with intact membranes?

> Nifedipine first line; atosiban if nifedipine is contraindicated. The aim is to delay birth for steroids or transfer.

Q37. What are the contraindications to tocolysis?

> Infection, abruption, fetal death or compromise, lethal anomaly and severe pre-eclampsia.

Q38. When should a woman with stable PPROM be delivered?

> Expectant management until 37 weeks if there is no infection or fetal compromise (RCOG 2019).

Q39. What if GBS is positive with PPROM at 34 weeks or later?

> Offer immediate induction with intrapartum antibiotics.

Q40. How is chorioamnionitis managed?

> IV broad-spectrum antibiotics and delivery, regardless of gestation.

Q41. Which intrapartum antibiotic is given in preterm labour?

> IV benzylpenicillin for GBS prophylaxis.

Q42. What is the preferred mode of delivery for a cephalic preterm baby?

> Vaginal birth. Avoid vacuum delivery before 34 weeks; forceps can be used.

Q43. How long should the cord be clamped after preterm birth?

> Delay clamping for at least 60 seconds if the baby does not need resuscitation.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal complications of PPROM?

> Chorioamnionitis, sepsis, abruption, retained placenta and endometritis.

Q45. What are the neonatal complications of prematurity?

> Respiratory distress syndrome, intraventricular haemorrhage, necrotising enterocolitis, sepsis, retinopathy and long-term neurodevelopmental problems.

Q46. What is the main risk of very early PPROM (before 22 weeks)?

> Pulmonary hypoplasia and limb contractures from low fluid.

Q47. How can preterm birth be prevented in her next pregnancy?

> Transvaginal cervical length scans; vaginal progesterone or cervical cerclage if the cervix is 25 mm or less.

Q48. What is a history-indicated cerclage?

> A stitch at 12–14 weeks for women with three or more previous preterm births or mid-trimester losses.

Q49. What advice is given to women with PPROM at home?

> Check temperature, avoid sex and tampons, and return at once with fever, pain, bleeding or reduced fetal movements.

Q50. What is a recent advance in preterm birth prevention?

> Preterm birth clinics with cervical length screening and the use of quantitative fetal fibronectin with the QUiPP app to predict birth risk.

CASE 7

Breech Presentation

> **Typical exam case:** A 29-year-old primigravida at 36 weeks is found on abdominal palpation to have a hard round mass at the fundus and a soft broad presenting part. Ultrasound confirms an extended breech.

HISTORY AND EXAMINATION

Q1. What is breech presentation?

> A longitudinal lie with the fetal buttocks or feet presenting over the pelvic inlet.

Q2. How common is breech?

> About 20% at 28 weeks but only 3–4% at term, as most babies turn.

Q3. What are the types of breech?

> Extended (frank): hips flexed, knees extended. Flexed (complete): hips and knees flexed. Footling: one or both feet below the buttocks.

Q4. Which type is most common at term?

> Extended (frank) breech, especially in first-time mothers.

Q5. What are the causes or associations of breech?

> Prematurity, placenta praevia, uterine anomalies, fibroids, polyhydramnios or oligohydramnios, multiple pregnancy, fetal anomalies and a short cord.

Q6. What would you feel on abdominal palpation?

> A hard, round, ballotable head at the fundus and a softer, broader presenting part; the fetal heart is heard above the umbilicus.

Q7. What would you feel on vaginal examination in labour?

> Soft buttocks, the anus and ischial tuberosities in a line, and possibly feet; meconium is common.

Q8. Why ask about previous caesarean section?

> It is a relative contraindication to ECV and vaginal breech birth.

Q9. What is the denominator in breech presentation?

> The sacrum.

Q10. What risk of malformation is linked with breech?

> Fetal anomalies such as neuromuscular conditions and hip dysplasia are more common.

DIAGNOSIS AND INVESTIGATIONS

Q11. How is breech confirmed?

> By ultrasound.

Q12. What else is checked on the ultrasound?

> Type of breech, fetal weight, attitude of the head (flexion or hyperextension), placental site, liquor volume and anomalies.

Q13. Why is hyperextension of the fetal neck important?

> It increases the risk of injury in vaginal breech birth; caesarean is advised.

Q14. Why must placenta praevia be excluded?

> It is a cause of breech and a contraindication to ECV and vaginal birth.

Q15. What blood test is needed before ECV?

> Blood group and antibody status, to give anti-D if RhD negative.

Q16. What monitoring is done before and after ECV?

> A CTG before and after the procedure.

Q17. Why do babies born breech need hip screening?

> Breech is a risk factor for developmental dysplasia of the hip; a hip ultrasound is done by 6 weeks.

Q18. What is an unstable lie?

> A lie that keeps changing after 36 weeks; it risks cord prolapse.

EXTERNAL CEPHALIC VERSION

Q19. What is ECV?

> Turning the baby from breech to cephalic by pressure on the mother's abdomen.

Q20. When is ECV offered?

> From 36 weeks in nulliparous women and from 37 weeks in multiparous women.

Q21. What is the success rate of ECV?

> About 50% (around 40% in first-time mothers and 60% in multiparous women).

Q22. What improves the success of ECV?

> Tocolysis with a beta-agonist such as terbutaline, multiparity, an unengaged breech, a relaxed uterus and adequate liquor.

Q23. What are absolute contraindications to ECV?

> Caesarean needed for another reason, APH in the last 7 days, abnormal CTG, major uterine anomaly, ruptured membranes and multiple pregnancy (except the second twin).

Q24. What are relative contraindications to ECV?

> Previous caesarean section, small baby, oligohydramnios, hyperextended head and unstable lie.

Q25. What are the risks of ECV?

> Transient CTG changes, placental abruption, fetomaternal haemorrhage, cord accident and emergency caesarean in about 1 in 200.

Q26. What is the chance of the baby turning back after a successful ECV?

> About 3%.

Q27. What happens to the risk of caesarean after successful ECV?

> It is still slightly higher than for a baby that was always cephalic, but much lower than for breech.

Q28. What is given to an RhD-negative woman after ECV?

> Anti-D and a Kleihauer test.

MODE OF DELIVERY

Q29. What did the Term Breech Trial show?

> Planned caesarean reduced perinatal death and serious early neonatal morbidity compared with planned vaginal breech birth. Long-term outcomes were similar.

Q30. When is planned caesarean for breech done?

> At 39 weeks if the baby is still breech.

Q31. Who might be suitable for vaginal breech birth?

> Women with an extended or flexed breech, estimated weight 2–3.8 kg, normal pelvis, no hyperextended neck, and a skilled attendant, after informed consent.

Q32. What are contraindications to vaginal breech birth?

> Footling breech, large (over 3.8 kg) or small (under 2 kg) baby, hyperextended neck, previous caesarean, and lack of a skilled attendant.

Q33. What is the key principle in vaginal breech delivery?

> 'Hands off the breech' and avoid traction, to prevent extension of the arms and head.

Q34. Which manoeuvre is used for extended arms?

> The Lovset manoeuvre.

Q35. Which manoeuvres help deliver the aftercoming head?

> The Mauriceau-Smellie-Veit manoeuvre, the Burns-Marshall method, or forceps for the aftercoming head.

Q36. What position is used for vaginal breech birth?

> Lithotomy or an upright all-fours position, depending on the unit and the attendant's training.

Q37. What is the main risk of a preterm breech in labour?

> Head entrapment through a partly dilated cervix, because the head is larger than the body.

Q38. What is the advice for preterm breech in labour?

> Discuss and consider caesarean section between 26 and 36+6 weeks.

Q39. What should be done if a woman presents in advanced labour with a breech?

> Counsel; caesarean may not be better if birth is imminent. A skilled attendant should conduct the vaginal birth.

Q40. What is the main complication at vaginal breech birth?

> Head entrapment, leading to hypoxia, and birth trauma (brachial plexus injury, fractures).

Q41. Is induction or augmentation of labour allowed for breech?

> Induction may be considered in selected cases; augmentation is generally not advised because delay may mean disproportion.

COUNSELLING AND RECENT ADVANCES

Q42. What postural methods to turn breech have been tried?

> Knee-chest positions and moxibustion; evidence of benefit is weak.

Q43. What should a woman know if ECV fails?

> She can choose planned caesarean or, if suitable, planned vaginal breech birth after counselling.

Q44. Is breech likely to recur?

> Recurrence is higher, especially if there is a uterine cause.

Q45. What is the neonatal follow-up for breech?

> Hip examination and hip ultrasound by 6 weeks.

Q46. Why is vaginal breech skills training important?

> Some women still deliver breech vaginally (undiagnosed or rapid labour), so staff must keep their skills up.

Q47. What is a recent trend in breech care?

> Renewed interest in upright vaginal breech birth and dedicated breech clinics offering ECV and counselling.

Q48. What is the perinatal mortality associated with breech in general?

> It is higher than for cephalic babies, partly because of associated prematurity and anomalies rather than the mode of birth alone.

Q49. What should be recorded after a breech birth?

> Time of birth of the body and head, manoeuvres used, cord gases, neonatal condition and any injuries.

Q50. Why is ECV offered rather than routine caesarean?

> It halves the chance of breech at birth and reduces caesarean rates without harm.

CASE 8

Twin Pregnancy

> **Typical exam case:** A 31-year-old G2 P1 at 20 weeks has a uterus larger than dates. Ultrasound shows a monochorionic diamniotic twin pregnancy.

HISTORY AND EXAMINATION

Q1. What clinical features suggest twins?

> Uterus larger than dates, severe nausea and vomiting, many fetal parts on palpation, two fetal heart sounds and anaemia.

Q2. What are the risk factors for twins?

> Assisted conception, older maternal age, family history (dizygotic), higher parity and African ethnicity.

Q3. Which minor symptoms are worse in twin pregnancy?

> Nausea and vomiting, heartburn, backache, breathlessness, oedema and varicose veins.

Q4. What would you find on abdominal examination?

> Fundal height larger than dates, two heads or many limbs palpable, and two fetal hearts heard at different places with different rates.

Q5. Why is it important to check BP and urine at each visit?

> Twins increase the risk of pre-eclampsia.

Q6. Why is fundal height not used to monitor growth in twins?

> It cannot assess two babies separately; ultrasound is used instead.

Q7. What is the incidence of twins?

> About 1 in 60–80 pregnancies in the UK; higher with fertility treatment.

Q8. What would you ask about fertility treatment?

> IVF and ovulation induction increase dizygotic twins; IVF slightly increases monozygotic twins.

Q9. What are the two types of twins by zygosity?

> Monozygotic (one egg splits; identical) and dizygotic (two eggs fertilised; non-identical).

Q10. Which type of twinning is more common?

> Dizygotic, about two-thirds of twins.

CHORIONICITY AND DIAGNOSIS

Q11. Why is chorionicity more important than zygosity?

> Chorionicity decides the risks; monochorionic twins share a placenta and face extra complications.

Q12. When is chorionicity best determined?

> By ultrasound at 11–14 weeks.

Q13. What is the lambda (twin peak) sign?

> A triangle of placental tissue between the membranes; it shows dichorionic twins.

Q14. What is the T sign?

> A thin membrane meeting the placenta at a right angle; it shows monochorionic diamniotic twins.

Q15. How does the timing of embryo split decide chorionicity in monozygotic twins?

> Before day 4: DCDA; days 4–8: MCDA; days 8–12: MCMA; after day 12: conjoined.

Q16. Are dizygotic twins ever monochorionic?

> No. Dizygotic twins are always dichorionic diamniotic.

Q17. Which twins can be of different sex?

> Only dizygotic twins, which are always dichorionic.

Q18. Why should twins be labelled at the first scan?

> To follow each twin reliably for screening and growth, e.g. by position and placental site.

INVESTIGATIONS AND SURVEILLANCE

Q19. How is Down syndrome screening done in twins?

> The combined test at 11–14 weeks, with a risk for each fetus in DC twins and per pregnancy in MC twins.

Q20. How often are uncomplicated DCDA twins scanned?

> Every 4 weeks from 20 weeks.

Q21. How often are uncomplicated MCDA twins scanned?

> Every 2 weeks from 16 weeks.

Q22. What is checked on the fortnightly scans in MC twins?

> Deepest vertical pool of liquor, bladders, growth and, from 20 weeks, MCA Doppler for TAPS.

Q23. How is weight discordance calculated?

> (Larger weight − smaller weight) ÷ larger weight × 100. A difference of 25% or more needs referral.

Q24. What is twin-to-twin transfusion syndrome?

> In MC twins, unbalanced placental connections send blood from the donor to the recipient. The recipient has polyhydramnios and the donor oligohydramnios.

Q25. What are the diagnostic criteria for TTTS?

> Recipient deepest pool 8 cm or more and donor deepest pool 2 cm or less.

Q26. What is the Quintero staging?

> I: fluid discordance with donor bladder visible; II: donor bladder not seen; III: abnormal Dopplers; IV: hydrops; V: death of one or both.

Q27. What is TAPS?

> Twin anaemia-polycythaemia sequence: slow transfusion through small connections, with one twin anaemic and the other polycythaemic, shown by MCA Doppler, with normal fluid.

Q28. When is the FBC checked in twin pregnancy?

> At booking and at 20–24 weeks, as anaemia is more common.

MANAGEMENT

Q29. Why is low-dose aspirin considered in twins?

> Twins are a moderate risk factor for pre-eclampsia; aspirin is given if there is another moderate risk factor.

Q30. What is the treatment of TTTS stage II or above?

> Fetoscopic laser ablation of placental connections between 16 and 26 weeks.

Q31. When should uncomplicated DCDA twins be delivered?

> At 37 weeks.

Q32. When should uncomplicated MCDA twins be delivered?

> At 36 weeks, after considering steroids.

Q33. When and how should MCMA twins be delivered?

> By caesarean section at 32+0 to 33+6 weeks, because of cord entanglement.

Q34. What decides the mode of delivery in twins?

> Mainly the presentation of the first twin. If twin 1 is cephalic, vaginal birth is reasonable; if not, caesarean is advised.

Q35. Where should twins be delivered?

> In an obstetric unit with an obstetrician, anaesthetist and neonatal team present.

Q36. Which analgesia is recommended in twin labour?

> Epidural, as it allows quick manipulation or operative delivery of twin 2.

Q37. How are twins monitored in labour?

> Continuous CTG of both twins, often with a scalp electrode on twin 1.

Q38. What is done after twin 1 is born?

> Check the lie and heart rate of twin 2 by palpation or ultrasound; if longitudinal, restart contractions with oxytocin and rupture membranes once the presenting part is engaged.

Q39. What is done if twin 2 is transverse?

> External cephalic version, or internal podalic version and breech extraction if membranes are intact.

Q40. What is the aim for the interval between twins?

> Deliver twin 2 within about 30 minutes, with continuous monitoring.

Q41. How is the third stage managed in twins?

> Actively, with oxytocin and often an oxytocin infusion, because of the high PPH risk.

Q42. What is done if one twin dies in an MC pregnancy?

> Expectant management with fetal medicine review; the survivor may have brain injury, so an MRI is done about 4 weeks later.

Q43. Why does single twin death matter more in MC twins?

> The survivor can bleed into the dead twin's circulation through shared vessels, causing brain injury or death.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal risks of twin pregnancy?

> Hyperemesis, anaemia, pre-eclampsia, GDM, APH, PPH, caesarean section and VTE.

Q45. What are the fetal risks?

> Preterm birth, growth restriction, congenital anomalies, stillbirth, cord accidents and complications specific to monochorionic twins.

Q46. What is the main cause of perinatal death in twins?

> Preterm birth.

Q47. Do bed rest, progesterone or cerclage prevent preterm birth in twins?

> No. NICE does not recommend them routinely.

Q48. What is interlocking (locked) twins?

> Twin 1 breech and twin 2 cephalic, with the chins locking; it is one reason for caesarean if twin 1 is breech.

Q49. What is TRAP sequence?

> Twin reversed arterial perfusion: a normal 'pump' twin perfuses an acardiac twin, risking heart failure in the pump twin.

Q50. What is a recent advance in twin care?

> Specialist multiple pregnancy clinics with standardised surveillance (NICE 2019) and fetoscopic laser for TTTS, which has greatly improved survival.

CASE 9

Previous Caesarean Section

> **Typical exam case:** A 30-year-old G2 P1 at 34 weeks had a lower segment caesarean section 3 years ago for fetal distress. She wants to know whether she can have a normal delivery this time.

HISTORY AND EXAMINATION

Q1. What would you ask about the previous caesarean?

Indication, gestation, type of uterine incision, whether in labour, complications (infection, PPH, extension of the incision), and baby's outcome.

Q2. Why is the indication for the previous caesarean important?

A non-recurrent cause (fetal distress, breech) gives better VBAC success than a recurrent one (failure to progress).

Q3. How do you find out the type of uterine incision?

From the operation notes; a low transverse incision allows VBAC, a classical incision does not.

Q4. Why ask about the interval since the last delivery?

A delivery interval under 12–18 months increases the risk of uterine rupture.

Q5. Why ask about previous vaginal births?

A previous vaginal birth greatly increases VBAC success (85–90%).

Q6. What examination findings are relevant?

Scar on the abdomen, scar tenderness, fundal height, presentation and engagement, and BMI.

Q7. Why check placental site in this pregnancy?

A low anterior placenta over the scar suggests placenta praevia and possible accreta.

Q8. What features of the current pregnancy affect VBAC?

Malpresentation, macrosomia, need for induction, BMI over 30 and pregnancy beyond 40 weeks.

Q9. What is the woman's own preference?

It is central; she should be supported in an informed choice.

Q10. What social factors matter?

Distance from hospital, transport and support at home, as labour should be in a hospital.

OPTIONS AND COUNSELLING

Q11. What are the two options for birth after one caesarean?

Planned vaginal birth after caesarean (VBAC) or elective repeat caesarean section (ERCS).

Q12. What is the success rate of planned VBAC?

About 72–75%.

Q13. What is the risk of uterine rupture in VBAC?

About 0.5% (1 in 200).

Q14. How does induction change the rupture risk?

It increases it two to three times, especially with prostaglandins.

Q15. What are the benefits of successful VBAC?

> Shorter recovery, fewer complications, and lower risk of placenta praevia and accreta in future pregnancies.

Q16. What are the risks of planned VBAC?

> Uterine rupture, emergency caesarean (about 25%), and a small increase in perinatal death and hypoxic injury compared with ERCS.

Q17. What are the risks of ERCS?

> Surgical complications, longer recovery, neonatal breathing problems, and higher risk of praevia and accreta in future pregnancies.

Q18. What are contraindications to VBAC?

> Previous uterine rupture, classical or inverted T incision, and the usual contraindications to vaginal birth such as placenta praevia.

INVESTIGATIONS

Q19. Which scans are relevant?

> Placental localisation at the anomaly scan; if low anterior, further imaging to exclude accreta.

Q20. What blood tests are needed?

> FBC and group and save on admission in labour.

Q21. Is measuring lower segment thickness useful?

> It is being studied; thin segments may increase rupture risk but it is not routine.

Q22. Why monitor the CTG continuously in VBAC labour?

> An abnormal CTG is often the first sign of uterine rupture.

Q23. What is the commonest first sign of uterine rupture?

> Fetal heart rate abnormality, especially prolonged bradycardia.

Q24. Why is pain over the scar important?

> It may suggest impending rupture, though it is not reliable alone.

Q25. What is the role of a partogram?

> To detect slow progress early, as dystocia in VBAC increases rupture risk.

Q26. What tests would you do if rupture is suspected?

> None that delay delivery; it is a clinical diagnosis needing immediate laparotomy.

Q27. Why is IV access needed in VBAC labour?

> For quick resuscitation and surgery if needed.

Q28. Why is maternal pulse checked?

> Tachycardia can be an early sign of bleeding from rupture.

MANAGEMENT

Q29. Where should VBAC labour take place?

> In a hospital with continuous monitoring and facilities for immediate caesarean and blood transfusion.

Q30. When is ERCS performed?

> At 39 weeks to reduce neonatal respiratory problems.

Q31. How is labour induced in a woman planning VBAC?

> With a balloon catheter or amniotomy and oxytocin, after discussion; prostaglandins carry a higher rupture risk.

Q32. How is oxytocin used in VBAC?

> Only after senior review, at a careful rate, with close monitoring of contractions.

Q33. Is epidural allowed in VBAC?

> Yes. It does not hide signs of rupture and is not a contraindication.

Q34. What are the signs of uterine rupture?

> Fetal distress, severe abdominal pain or scar pain, vaginal bleeding, haematuria, loss of contractions, loss of station of the presenting part, and maternal shock.

Q35. How is uterine rupture managed?

> Immediate resuscitation and category 1 laparotomy, delivery of the baby, and uterine repair or hysterectomy.

Q36. What is the decision-to-delivery time for category 1 caesarean?

> Within 30 minutes.

Q37. What happens if she goes into labour before her planned ERCS?

> Assess; she may continue to VBAC if suitable or have an emergency caesarean.

Q38. What is the advice if she has had two previous caesareans?

> VBAC may be offered after counselling; success is similar but rupture risk is slightly higher.

Q39. What is the main surgical risk of repeat caesarean?

> Adhesions and bladder injury.

Q40. What prophylactic antibiotics are given at caesarean?

> IV antibiotics before skin incision.

Q41. What VTE prophylaxis is given after emergency caesarean?

> LMWH for 10 days, or 6 weeks if additional risk factors.

Q42. When is a classical caesarean needed?

> Poorly formed lower segment (very preterm), transverse lie with ruptured membranes, large lower segment fibroid, or anterior praevia/accreta in some cases.

Q43. What is the next birth plan after a classical caesarean?

> Planned repeat caesarean at about 37 weeks, as rupture risk is high.

COMPLICATIONS AND COUNSELLING

Q44. What is the risk of placenta praevia after one caesarean?

> About doubled compared with no previous caesarean.

Q45. What is the risk of placenta accreta with praevia after multiple caesareans?

> It rises steeply, to about 40% after three caesareans with praevia.

Q46. What is a caesarean scar ectopic pregnancy?

> A pregnancy implanted in the caesarean scar; it can cause rupture and bleeding and must be diagnosed early by scan.

Q47. What is a caesarean scar niche (isthmocele)?

> A defect at the scar that can cause spotting, pain and subfertility.

Q48. What advice is given about future pregnancies after a caesarean?

> Wait at least 18 months, book early, and have an early scan to locate the pregnancy and placenta.

Q49. What is the success rate of VBAC after a previous vaginal birth?

> About 85–90%.

Q50. What is a recent advance in VBAC care?

> Use of VBAC prediction calculators and dedicated birth-options clinics for informed choice.

CASE 10

Rhesus Negative Pregnancy

> **Typical exam case:** A 27-year-old G2 P1 at 16 weeks has blood group O RhD negative. Her first baby was RhD positive. She did not receive anti-D after her first delivery, which took place at home.

HISTORY AND EXAMINATION

Q1. Why is it important to know the blood group in pregnancy?

To detect RhD-negative women, give anti-D prophylaxis, and screen for red cell antibodies that can cause haemolytic disease of the fetus and newborn.

Q2. What history would you take from an RhD-negative woman?

Previous pregnancies and their outcomes, anti-D doses received, sensitising events, blood transfusions, previous affected babies (jaundice, exchange transfusion, hydrops, stillbirth).

Q3. What is the partner's role?

If the partner is RhD negative, the baby will also be RhD negative (if paternity is certain).

Q4. What are sensitising events in pregnancy?

Miscarriage with surgery, ectopic pregnancy, termination, APH, abdominal trauma, ECV, amniocentesis, CVS, intrauterine death and delivery.

Q5. Why is this woman at risk?

She had an RhD-positive baby and did not receive anti-D, so she may have become sensitised.

Q6. What signs of fetal anaemia might be found clinically?

Few; polyhydramnios or a large uterus may suggest hydrops. Reduced fetal movements may occur.

Q7. What history suggests a previous affected baby?

Early jaundice, phototherapy, exchange transfusion, intrauterine transfusion, hydrops or stillbirth.

Q8. Which other red cell antibodies can cause HDFN?

Anti-c, anti-Kell and anti-E, among others.

Q9. What is special about anti-Kell?

It suppresses fetal red cell production as well as causing haemolysis, so anaemia can occur at low titres.

Q10. Why ask about blood transfusions?

Transfusion can sensitise a woman to red cell antigens such as Kell.

PATHOPHYSIOLOGY AND DIAGNOSIS

Q11. How does rhesus sensitisation occur?

Fetal RhD-positive red cells enter the circulation of an RhD-negative mother, and she makes anti-D antibodies.

Q12. Why is the first baby usually not affected?

The first response produces IgM, which does not cross the placenta; the IgG response comes in later pregnancies.

Q13. How do antibodies harm the fetus?

Maternal IgG anti-D crosses the placenta and destroys fetal RhD-positive red cells, causing anaemia, hydrops and death.

Q14. What is hydrops fetalis?

> Fluid in two or more fetal compartments (ascites, pleural or pericardial effusion, skin oedema).

Q15. What is the neonatal consequence of haemolysis?

> Jaundice in the first 24 hours, anaemia, kernicterus and hepatosplenomegaly.

Q16. What does the direct Coombs test show in the newborn?

> Antibody coating fetal red cells; it is positive in haemolytic disease.

Q17. What is the indirect Coombs test?

> A test for antibodies in the mother's serum (antibody screen).

Q18. How common is RhD negativity?

> About 15% in white populations; lower in South Asian populations (about 5–7%).

INVESTIGATIONS

Q19. What is the first test for this woman?

> An antibody screen to look for anti-D.

Q20. How is fetal RhD type found without invasive testing?

> Cell-free fetal DNA testing of maternal blood.

Q21. When is anti-D measured as a level?

> If anti-D is detected, it is quantified in IU/mL every 4 weeks until 28 weeks, then every 2 weeks.

Q22. What anti-D level needs referral to fetal medicine?

> Above 4 IU/mL; above 15 IU/mL carries a high risk of severe disease.

Q23. How is fetal anaemia detected?

> Middle cerebral artery peak systolic velocity on Doppler ultrasound; a value above 1.5 multiples of the median suggests anaemia.

Q24. Why is MCA Doppler preferred to amniocentesis?

> It is non-invasive and accurate, and avoids further sensitisation.

Q25. How is fetal anaemia confirmed?

> By fetal blood sampling (cordocentesis), usually at the time of intrauterine transfusion.

Q26. What does a Kleihauer test measure?

> The amount of fetal red cells in maternal blood after a sensitising event.

Q27. What test is done on cord blood at birth?

> Blood group, direct Coombs test, haemoglobin and bilirubin.

Q28. What can be confused with immune anti-D in a recently treated woman?

> Passive anti-D from recent prophylaxis; it is usually at a low level.

MANAGEMENT

Q29. What is routine antenatal anti-D prophylaxis?

> Anti-D given to non-sensitised RhD-negative women at 28 weeks (1500 IU single dose) or at 28 and 34 weeks (two doses).

Q30. What dose of anti-D is given for a sensitising event before 12 weeks?

> Only for surgical management of miscarriage, ectopic pregnancy or termination; 250 IU.

Q31. What dose is given for a sensitising event between 12 and 20 weeks?

> 250 IU within 72 hours; no Kleihauer needed.

Q32. What dose is given after 20 weeks?

> At least 500 IU within 72 hours, with a Kleihauer test to decide on more.

Q33. How much fetal blood does 500 IU of anti-D cover?

> About 4 mL of fetal red cells.

Q34. What if anti-D was not given within 72 hours?

> It may still give some protection up to 10 days.

Q35. What is done after delivery for an RhD-negative woman?

> Check cord blood group; if baby is RhD positive, give at least 500 IU anti-D within 72 hours and do a Kleihauer.

Q36. Does a sensitised woman need anti-D prophylaxis?

> No. It does not help once she has immune anti-D.

Q37. How is a sensitised pregnancy managed?

> Early referral to fetal medicine, regular anti-D levels and MCA Dopplers, and treatment of fetal anaemia.

Q38. What is the treatment of severe fetal anaemia?

> Intrauterine transfusion of O RhD-negative, CMV-negative, irradiated red cells into the umbilical vein.

Q39. When is a sensitised pregnancy delivered?

> Usually at 37–38 weeks, or earlier (about 34–36 weeks) if transfusions are ongoing.

Q40. What is the newborn treatment for haemolytic disease?

> Phototherapy, top-up transfusion, IV immunoglobulin in some cases and exchange transfusion for severe jaundice.

Q41. Why are cffDNA tests now used routinely in some countries?

> They avoid giving anti-D to the 40% of RhD-negative women carrying RhD-negative babies.

Q42. What is the role of anti-D after abdominal trauma at 30 weeks?

> Give 500 IU within 72 hours and do a Kleihauer, even if she has had routine prophylaxis.

Q43. What is IV immunoglobulin used for in severe alloimmunisation?

> In very early severe disease, to delay the need for intrauterine transfusion; this is specialist care.

COMPLICATIONS AND COUNSELLING

Q44. What are the fetal outcomes of untreated severe disease?

> Hydrops, stillbirth and neonatal death.

Q45. What are the risks of intrauterine transfusion?

> Fetal bradycardia, infection, preterm labour, fetal loss (1–2% per procedure) and further sensitisation.

Q46. What is kernicterus?

> Bilirubin deposition in the brain, causing deafness, cerebral palsy and learning problems.

Q47. What is the recurrence risk in a sensitised woman?

> Disease usually gets earlier and more severe in each affected pregnancy.

Q48. What should the woman carry with her?

> A card or record showing her blood group and antibody status.

Q49. What advice is given for future pregnancies?

> Early booking, early antibody testing and referral to fetal medicine.

Q50. What is a recent advance in rhesus disease prevention?

> Routine high-throughput cffDNA testing for fetal RhD to target anti-D prophylaxis.

CASE 11

Fetal Growth Restriction

> **Typical exam case:** A 34-year-old G2 P1 at 32 weeks has a symphysis-fundal height of 27 cm. She smokes 10 cigarettes a day. Ultrasound shows an estimated fetal weight below the 3rd centile with reduced liquor.

HISTORY AND EXAMINATION

Q1. What is the difference between SGA and FGR?

SGA is a baby below the 10th centile, which may be constitutionally small and healthy. FGR is a baby that has not reached its growth potential, usually due to placental dysfunction.

Q2. What maternal risk factors for FGR would you ask about?

Smoking, drug misuse, age over 40, previous SGA baby or stillbirth, hypertension, kidney disease, diabetes with vascular disease, antiphospholipid syndrome and poor nutrition.

Q3. What current pregnancy factors increase FGR risk?

Pre-eclampsia, APH, low PAPP-A, multiple pregnancy, and heavy bleeding in early pregnancy.

Q4. What fetal causes of FGR are there?

Chromosomal problems (trisomy 18, 13), structural anomalies and congenital infection (CMV, toxoplasmosis, rubella).

Q5. Why is fundal height plotted on a customised chart?

Customised charts adjust for maternal height, weight, ethnicity and parity, improving detection of growth problems.

Q6. What does a fundal height 3 cm or more below gestation mean?

It may indicate growth restriction or oligohydramnios and needs an ultrasound scan.

Q7. Why is BP important here?

Pre-eclampsia is a common cause of FGR.

Q8. What would you ask about fetal movements?

Reduced fetal movements can be a sign of fetal compromise in FGR.

Q9. Why is smoking important?

It doubles the risk of FGR; stopping at any stage helps.

Q10. What abdominal findings suggest oligohydramnios?

Small uterus for dates and easily palpable fetal parts.

DIAGNOSIS

Q11. How is FGR defined (Delphi consensus)?

Early FGR (before 32 weeks): AC or EFW below the 3rd centile, or absent umbilical end-diastolic flow, or AC/EFW below the 10th centile with abnormal Dopplers. Late FGR uses similar criteria plus fall in growth across centiles.

Q12. What is symmetrical FGR?

Head and body both small; often due to early causes such as chromosomal problems or infection.

Q13. What is asymmetrical FGR?

Head growth spared with a small abdomen; typical of placental insufficiency.

Q14. Why is the abdominal circumference small in placental FGR?

> Reduced liver glycogen stores due to poor nutrition, while blood is redistributed to the brain (brain-sparing).

Q15. What is the most common cause of FGR?

> Placental insufficiency.

Q16. What is early-onset versus late-onset FGR?

> Early-onset is before 32 weeks, often severe and linked with pre-eclampsia. Late-onset is after 32 weeks, more common and harder to detect.

Q17. How does FGR differ from a constitutionally small baby?

> A healthy small baby grows along its centile with normal Dopplers and liquor.

Q18. What is the main danger of FGR?

> Stillbirth.

INVESTIGATIONS

Q19. What does the ultrasound assess in suspected FGR?

> Biometry (HC, AC, FL, EFW), liquor volume and Dopplers.

Q20. What is the main surveillance tool in FGR?

> Umbilical artery Doppler.

Q21. What does absent or reversed end-diastolic flow in the umbilical artery mean?

> High placental resistance and a high risk of fetal compromise and death.

Q22. What is the role of middle cerebral artery Doppler?

> A low MCA pulsatility index shows brain-sparing, especially useful in late FGR.

Q23. What does an abnormal ductus venosus Doppler indicate?

> Fetal heart strain and acidaemia; it is a late sign before fetal death.

Q24. What is computerised CTG used for?

> To measure short-term variability; a fall suggests fetal compromise.

Q25. What maternal tests are done?

> BP and urine for pre-eclampsia, PlGF testing, and screening for antiphospholipid syndrome when relevant.

Q26. When is karyotyping offered?

> In severe early FGR, especially with anomalies or normal Dopplers, to exclude chromosomal problems.

Q27. Which infections are tested for in severe FGR?

> CMV and toxoplasmosis (and syphilis), especially with symmetrical growth restriction.

Q28. How often are scans repeated in FGR?

> Growth every 2 weeks at the most; Dopplers more often (often weekly or twice weekly) depending on severity.

MANAGEMENT

Q29. What is the main principle of FGR management?

> Monitoring and timely delivery; no drug treatment improves growth.

Q30. What should be given if delivery is expected before 34–35 weeks?

> Antenatal corticosteroids.

Q31. When is magnesium sulfate given?

> For neuroprotection if birth is expected before 32 weeks (considered to 33+6).

Q32. When is an SGA baby with normal Dopplers delivered?

> Usually from 37 weeks, after discussion.

Q33. When is delivery advised with absent end-diastolic flow?

> By 32–34 weeks, or earlier if ductus venosus or CTG becomes abnormal.

Q34. When is delivery advised with reversed end-diastolic flow?

> By about 30–32 weeks, sooner if worsening.

Q35. What is the usual mode of delivery for severe FGR with abnormal Dopplers?

> Caesarean section.

Q36. Can a growth-restricted baby with normal Dopplers be delivered vaginally?

> Yes, with induction and continuous CTG monitoring.

Q37. Why is continuous CTG advised in labour?

> Growth-restricted babies tolerate labour poorly.

Q38. Where should a very preterm FGR baby be delivered?

> In a unit with a neonatal intensive care unit.

Q39. Does bed rest help FGR?

> No.

Q40. Does aspirin treat established FGR?

> No; it is used for prevention in high-risk women from 12 weeks.

Q41. What smoking advice should be given now?

> Stopping smoking at any point improves outcomes; refer to stop-smoking services.

Q42. What should be done if pre-eclampsia develops?

> Manage BP and plan delivery according to maternal and fetal condition.

Q43. What follow-up is needed after delivery?

> Placental histology, maternal BP checks and planning for the next pregnancy.

COMPLICATIONS AND COUNSELLING

Q44. What are the neonatal complications of FGR?

> Hypoglycaemia, hypothermia, polycythaemia, NEC, respiratory problems and meconium aspiration.

Q45. What are the long-term effects?

> Higher risk of neurodevelopmental delay and, in adult life, hypertension, heart disease and type 2 diabetes (Barker hypothesis).

Q46. What is the recurrence risk?

> About 20% in the next pregnancy.

Q47. How is FGR prevented in the next pregnancy?

> Aspirin 150 mg from 12 weeks if placental dysfunction caused it, stop smoking, and serial growth scans from 26–28 weeks.

Q48. What is the Saving Babies' Lives approach to FGR?

> Risk assessment at booking, customised growth charts, and serial scans for high-risk women.

Q49. Why is maternal awareness of fetal movements important?

> Reduced movements may precede stillbirth in growth-restricted babies.

Q50. What is a recent advance in FGR?

> The international Delphi definition of FGR, and the use of PlGF and the sFlt-1/PlGF ratio to identify placental dysfunction.

CASE 12

Prolonged Pregnancy and Induction of Labour

> **Typical exam case:** A 24-year-old primigravida is 41+2 weeks pregnant by a first-trimester dating scan. Her pregnancy has been uncomplicated and she is not in labour.

HISTORY AND EXAMINATION

Q1. How is prolonged (post-term) pregnancy defined?

> A pregnancy lasting 42+0 weeks or more.

Q2. How common is post-term pregnancy?

> About 5–10% of pregnancies reach 42 weeks; fewer when dating is by early ultrasound.

Q3. Why is accurate dating important?

> Wrong dates are the most common reason for an apparent post-term pregnancy; early ultrasound dating reduces unnecessary induction.

Q4. What risk factors are linked with post-term pregnancy?

> First pregnancy, previous post-term pregnancy, obesity, male fetus and, rarely, anencephaly or placental sulphatase deficiency.

Q5. What would you ask about at 41 weeks?

> Fetal movements, show, contractions, leakage of fluid, and her wishes about induction.

Q6. What would you check on abdominal examination?

> Fundal height, presentation, engagement and the fetal heart.

Q7. What is the Bishop score?

> A score of cervical dilatation, length (effacement), consistency, position and station of the presenting part, used to judge readiness for induction.

Q8. What Bishop score is favourable?

> A score of more than 6 (NICE uses 7 or more as favourable).

Q9. Why must presentation be checked before induction?

> Breech or unstable lie changes the plan.

Q10. What maternal conditions often need earlier induction?

> Pre-eclampsia, diabetes, obstetric cholestasis, FGR and PPROM.

RISKS AND COUNSELLING

Q11. What are the fetal risks of prolonged pregnancy?

> Rising stillbirth risk, meconium aspiration, macrosomia, oligohydramnios, shoulder dystocia and neonatal admission.

Q12. What are the maternal risks?

> More instrumental birth, caesarean section, perineal trauma and PPH (often due to a large baby).

Q13. What does NICE recommend about induction?

> Offer induction from 41+0 weeks, explaining that stillbirth risk rises after this.

Q14. What if the woman declines induction?

> Offer increased monitoring, such as twice-weekly CTG and ultrasound for liquor volume.

Q15. What is a membrane sweep?

> Separating the membranes from the cervix with a finger to release prostaglandins and start labour.

Q16. When is a membrane sweep offered?

> At 39–40 weeks onwards; it reduces the need for formal induction.

Q17. What are the risks of membrane sweep?

> Discomfort, spotting and irregular contractions; no increase in infection.

Q18. What is post-maturity syndrome in the baby?

> Dry, peeling skin, long nails, loss of subcutaneous fat and meconium staining.

METHODS OF INDUCTION

Q19. What are the main methods of induction?

> Membrane sweep, vaginal prostaglandins (dinoprostone) or oral misoprostol, mechanical methods (balloon catheter), amniotomy and oxytocin.

Q20. How do prostaglandins work?

> They soften and ripen the cervix and may start contractions.

Q21. Which prostaglandin preparations are used?

> Dinoprostone vaginal gel, tablet or pessary (Propess), or low-dose oral misoprostol.

Q22. What is a balloon catheter induction?

> A Foley or double balloon catheter placed through the cervix to ripen it mechanically; it causes less hyperstimulation.

Q23. When is a balloon preferred?

> In women with a previous caesarean section and in outpatient induction programmes.

Q24. When is amniotomy used?

> When the cervix is favourable, followed by oxytocin if labour does not start.

Q25. What is the role of oxytocin?

> To start or strengthen contractions after the membranes are ruptured.

Q26. How is oxytocin given?

> As an IV infusion, increased every 30 minutes to achieve 4–5 contractions in 10 minutes.

Q27. What is outpatient induction?

> Cervical ripening at home with a balloon or prostaglandin in low-risk women, returning when labour starts.

Q28. What monitoring is needed after prostaglandins?

> CTG before and after insertion, and monitoring for contractions and hyperstimulation.

COMPLICATIONS OF INDUCTION AND THEIR MANAGEMENT

Q29. What is uterine hyperstimulation?

> Excessive contractions (more than 5 in 10 minutes, or contractions lasting over 2 minutes) with fetal heart changes.

Q30. How is hyperstimulation managed?

> Remove the prostaglandin, stop oxytocin, change position, and consider terbutaline 0.25 mg SC.

Q31. What is failed induction?

> Labour does not start after one cycle of treatment; options include further induction or caesarean.

Q32. What is the risk of cord prolapse with amniotomy?

> It is higher if the head is high; amniotomy should be done when the head is well applied.

Q33. Why must oxytocin be used carefully in multiparous women?

> Higher risk of uterine rupture.

Q34. What is a side effect of prolonged high-dose oxytocin?

> Water intoxication and hyponatraemia, because of its antidiuretic effect.

Q35. Does induction at 41 weeks increase caesarean rates?

> No. Studies show induction at 41 weeks does not increase, and may reduce, caesarean rates.

Q36. What did the ARRIVE trial show?

> Induction at 39 weeks in low-risk first-time mothers slightly reduced caesarean rates without harming babies.

Q37. What did the SWEPIS trial show?

> Induction at 41 weeks reduced perinatal deaths compared with waiting until 42 weeks.

Q38. Is uterine rupture a risk of induction?

> Yes, mainly in women with a uterine scar or high parity.

Q39. Is induction contraindicated with a previous caesarean?

> No, but risks are higher; balloon or amniotomy with oxytocin is preferred over prostaglandins.

Q40. What are contraindications to induction?

> Placenta praevia, vasa praevia, transverse lie, cord presentation, active genital herpes and previous classical caesarean.

Q41. What is the role of the obstetric team in long inductions?

> Regular review of progress and wellbeing, and senior input before escalating oxytocin.

Q42. What should women be told about the induction process?

> It can take 24–48 hours, may be uncomfortable, and they will have monitoring and choices of pain relief.

Q43. Which women should not have outpatient induction?

> Women with high-risk pregnancies such as FGR, pre-eclampsia or previous caesarean (in most units).

RECENT ADVANCES AND KEY POINTS

Q44. What is the main stillbirth benefit of offering induction from 41 weeks?

> It reduces perinatal deaths with no increase in caesarean section.

Q45. What is low-dose oral misoprostol?

> A tablet-based induction method recommended by WHO and NICE (2021) with effectiveness similar to dinoprostone.

Q46. Why is shared decision making important in induction?

> Induction is an intervention; women should understand the benefits, risks and alternatives.

Q47. What is the role of cervical length ultrasound before induction?

> It is being studied as a predictor of success but is not routine.

Q48. What are the newborn checks after a post-term birth?

> Assessment for meconium aspiration, hypoglycaemia and birth injury.

Q49. What is the most important factor in the success of induction?

> A favourable cervix (high Bishop score) and previous vaginal birth.

Q50. What is the key take-home message for post-term pregnancy?

> Accurate early dating, offer a membrane sweep, offer induction from 41 weeks, and monitor if she declines.

CASE 13

Postpartum Haemorrhage

Typical exam case: A 35-year-old G5 P4 had a vaginal birth of a 4.2 kg baby after a 16-hour labour. Ten minutes after delivery of the placenta she is bleeding heavily, with an estimated loss of 1200 mL. Her pulse is 120/min and BP 90/55 mmHg.

DEFINITION AND RISK FACTORS

Q1. How is primary PPH defined?

Blood loss of 500 mL or more from the genital tract within 24 hours of birth.

Q2. How is PPH graded?

Minor: 500–1000 mL. Major: more than 1000 mL (moderate 1000–2000 mL, severe over 2000 mL or ongoing bleeding with shock).

Q3. What is secondary PPH?

Abnormal or excessive bleeding from 24 hours to 12 weeks after birth.

Q4. What are the 4 Ts of PPH?

Tone (uterine atony), Trauma (tears, rupture, inversion), Tissue (retained placenta or membranes) and Thrombin (clotting problems).

Q5. Which is the most common cause?

Uterine atony (Tone), about 70% of cases.

Q6. What risk factors does this woman have?

Grand multiparity, a large baby and a prolonged labour.

Q7. What other antenatal risk factors are there?

Previous PPH, multiple pregnancy, polyhydramnios, placenta praevia, abruption, pre-eclampsia, anaemia, obesity, fibroids and age over 40.

Q8. What intrapartum risk factors are there?

Induction, prolonged labour, oxytocin augmentation, instrumental birth, caesarean section, retained placenta and general anaesthesia.

Q9. Why is blood loss often underestimated?

Blood mixes with liquor and soaks into drapes and pads; measuring by weighing pads is more accurate.

Q10. What signs show significant blood loss?

Tachycardia, low BP, pallor, cold peripheries, low urine output and confusion. Young women may keep BP until late.

IMMEDIATE MANAGEMENT

Q11. What is the first step in managing major PPH?

Call for help (senior obstetrician, anaesthetist, midwives), and start resuscitation with ABC.

Q12. What immediate resuscitation steps are taken?

High-flow oxygen, lay flat, two large-bore cannulas, bloods (FBC, cross-match, clotting, fibrinogen), warm IV fluids and blood.

Q13. How do you assess the cause quickly?

Feel the uterus for tone, check the placenta is complete, inspect for tears, and check clotting.

Q14. What is the first step for an atonic uterus?

> Rub up the uterus (fundal massage) and empty the bladder with a catheter.

Q15. What is bimanual compression?

> Compressing the uterus between a fist in the anterior fornix and a hand on the abdomen to reduce bleeding.

Q16. What drugs are used for uterine atony?

> Oxytocin 5 IU IV then infusion, ergometrine 500 micrograms IM/IV, carboprost 250 micrograms IM every 15 minutes (max 8 doses), and misoprostol 800 micrograms.

Q17. When is ergometrine avoided?

> In hypertension and pre-eclampsia.

Q18. When is carboprost used with care?

> In asthma, as it can cause bronchospasm.

Q19. What is the role of tranexamic acid?

> 1 g IV given early (within 3 hours) reduces death from bleeding (WOMAN trial).

Q20. When is the massive haemorrhage protocol activated?

> With ongoing bleeding over 1500 mL, or with signs of shock; blood products are given in a set ratio.

FURTHER MANAGEMENT

Q21. What fibrinogen level should be maintained?

> Above 2 g/L, using cryoprecipitate or fibrinogen concentrate.

Q22. What is the target platelet count during PPH?

> Above 75 × 10^9/L.

Q23. What is done if the uterus is contracted but bleeding continues?

> Examine for genital tract trauma under good light, often in theatre, and repair.

Q24. How is a retained placenta managed?

> Manual removal in theatre with regional or general anaesthesia, with antibiotics.

Q25. What is the first surgical step for atony not responding to drugs?

> Intrauterine balloon tamponade (e.g. Bakri balloon).

Q26. What surgical options follow if bleeding continues?

> Laparotomy with B-Lynch compression suture, uterine or internal iliac artery ligation, interventional radiology embolisation, and hysterectomy.

Q27. When should hysterectomy be done?

> Early rather than late if other measures fail, by a senior surgeon, to save the mother's life.

Q28. What is cell salvage?

> Collecting the woman's own blood during surgery, washing it and returning it; useful in caesarean and for Jehovah's Witnesses.

Q29. Why keep the woman warm?

> Hypothermia worsens clotting.

Q30. Where should she be monitored after a major PPH?

> In a high-dependency unit with fluid balance, urine output and observations.

Q31. What is the mnemonic HAEMOSTASIS used for?

> A stepwise approach to PPH: Help, Assess, Establish aetiology, Massage, Oxytocin, Shift to theatre, Tamponade, Apply compression sutures, Systematic devascularisation, Interventional radiology, Subtotal or total hysterectomy.

Q32. What is the dose of oxytocin infusion?

> Commonly 40 IU in 500 mL given over 4 hours.

Q33. What postnatal care is needed after PPH?

> Check Hb, give iron (oral or IV), VTE prophylaxis once bleeding stops, debriefing and documentation.

Q34. What is uterine inversion and how is it managed?

> The uterus turns inside out after delivery; it causes shock and bleeding. Replace it at once by hand (Johnson manoeuvre) or by hydrostatic pressure.

Q35. What is the shock index?

> Heart rate divided by systolic BP; above 1 suggests significant blood loss. Here it is 120/90 = 1.3.

PREVENTION

Q36. What is the best way to prevent PPH?

> Active management of the third stage of labour.

Q37. What does active management include?

> Oxytocin 10 IU IM, delayed cord clamping (after 1 minute, before 5 minutes), and controlled cord traction.

Q38. Why should anaemia be treated before delivery?

> Women with good Hb tolerate blood loss better.

Q39. Which women should deliver in a hospital with blood bank facilities?

> Women with risk factors such as previous PPH, praevia, accreta, grand multiparity and anaemia.

Q40. What is the role of carbetocin?

> A long-acting oxytocin analogue used for PPH prevention, especially at caesarean section.

Q41. What is the role of tranexamic acid in prevention?

> It may be given at caesarean section in high-risk women; routine use is still being studied.

Q42. Why is the third stage of labour observed for at least an hour after birth?

> Most primary PPH happens soon after delivery; early checks of uterine tone, bleeding and observations allow fast action.

Q43. What drills help teams manage PPH?

> Regular multidisciplinary skills training (e.g. PROMPT).

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of major PPH?

> Hypovolaemic shock, DIC, acute kidney injury, ARDS, transfusion reactions, infection, Sheehan syndrome, hysterectomy and death.

Q45. What is Sheehan syndrome?

> Pituitary necrosis after severe PPH; the first sign is often failure of lactation, followed by hypothyroidism and adrenal insufficiency.

Q46. What is the recurrence risk of PPH?

> About 10–15% in a future pregnancy.

Q47. What advice is given for the next pregnancy?

> Deliver in hospital, treat anaemia, IV access in labour, active third stage and possibly prophylactic tranexamic acid.

Q48. Why is debriefing important?

> PPH can be traumatic; it helps understanding and reduces post-traumatic stress.

Q49. What is the global importance of PPH?

> It is the leading cause of maternal death worldwide, especially in low-income countries.

Q50. What is a recent advance in PPH care?

> The WHO E-MOTIVE bundle: early detection with a calibrated drape plus a bundle of massage, oxytocin, tranexamic acid, IV fluids, examination and escalation, which reduced severe PPH.

CASE 14

Heart Disease in Pregnancy

> **Typical exam case:** A 26-year-old G2 P1 at 24 weeks has known rheumatic mitral stenosis. She complains of increasing breathlessness on mild exertion and palpitations. There is a mid-diastolic murmur at the apex.

HISTORY AND EXAMINATION

Q1. What is the most common cause of heart disease in pregnant women in Pakistan?

> Rheumatic heart disease, especially mitral stenosis.

Q2. What symptoms would you ask about?

> Breathlessness, orthopnoea, paroxysmal nocturnal dyspnoea, palpitations, chest pain, syncope, cough with frothy or blood-stained sputum, and ankle swelling.

Q3. Which normal pregnancy symptoms can mimic heart disease?

> Mild breathlessness, tiredness, ankle swelling and a soft ejection systolic murmur.

Q4. Which symptoms in pregnancy suggest real heart disease?

> Orthopnoea, paroxysmal nocturnal dyspnoea, haemoptysis, syncope, chest pain and breathlessness at rest.

Q5. What past history would you ask about?

> Rheumatic fever, previous valve surgery or balloon valvotomy, congenital heart disease, and current medicines (warfarin, penicillin prophylaxis).

Q6. How is functional status graded?

> By the NYHA classification: class I (no symptoms) to class IV (symptoms at rest).

Q7. What signs of mitral stenosis would you find?

> Malar flush, irregular pulse if AF, tapping apex beat, loud first heart sound, opening snap and a mid-diastolic rumbling murmur at the apex.

Q8. What signs of heart failure would you look for?

> Tachycardia, raised JVP, basal crackles, liver enlargement and ankle oedema.

Q9. Why does mitral stenosis worsen in pregnancy?

> Increased blood volume and heart rate shorten diastolic filling time, raising left atrial pressure and causing pulmonary oedema.

Q10. When is the danger greatest?

> At 28–32 weeks (peak volume), in labour, and immediately after delivery when blood returns from the uterus to the circulation.

PHYSIOLOGY AND RISK

Q11. How much does cardiac output rise in pregnancy?

> By about 40% by mid-pregnancy, and further in labour and straight after delivery.

Q12. How is maternal cardiac risk assessed?

> Using the modified WHO classification (mWHO I–IV), with CARPREG or ZAHARA scores.

Q13. Which conditions are mWHO class IV, where pregnancy is not advised?

> Pulmonary arterial hypertension, severe left ventricular dysfunction (EF below 30%), severe symptomatic mitral stenosis, severe aortic stenosis, Marfan with aorta over 45 mm and previous peripartum cardiomyopathy with residual dysfunction.

Q14. Why is pulmonary hypertension so dangerous in pregnancy?

> Maternal mortality is high (up to 30–50%) because the right ventricle cannot cope with the volume load.

Q15. Why is heart disease important in maternal mortality?

> Cardiac disease is the leading indirect cause of maternal death in the UK.

Q16. What are the fetal risks of maternal heart disease?

> Growth restriction, preterm birth, and congenital heart disease if the mother has congenital heart disease.

Q17. What is peripartum cardiomyopathy?

> Heart failure with reduced ejection fraction in late pregnancy or up to 5 months after birth, with no other cause.

Q18. What is the recurrence risk of congenital heart disease in the baby if the mother has it?

> About 3–5%, higher for some lesions.

INVESTIGATIONS

Q19. What is the key investigation?

> Echocardiography, to assess valve area, gradients, pulmonary pressures and ventricular function.

Q20. What is severe mitral stenosis on echo?

> A valve area below 1.0–1.5 cm^2.

Q21. What does the ECG show in mitral stenosis?

> P mitrale (broad notched P waves), atrial fibrillation and right ventricular hypertrophy.

Q22. Is a chest X-ray safe in pregnancy?

> Yes, the fetal dose is negligible. It may show left atrial enlargement and pulmonary congestion.

Q23. What blood tests are useful?

> FBC (anaemia worsens heart failure), thyroid function, renal function and BNP.

Q24. What is BNP used for?

> A raised BNP or NT-proBNP supports heart failure; a normal level makes it unlikely.

Q25. What fetal monitoring is needed?

> Serial growth scans and a fetal echocardiogram if the mother has congenital heart disease.

Q26. Why is INR monitored?

> Women on warfarin for mechanical valves need tight INR control.

Q27. Why are infections searched for?

> Infection, anaemia and arrhythmia can trigger heart failure.

Q28. Who should look after these women?

> A joint cardiac-obstetric team, including a cardiologist, obstetrician, anaesthetist and neonatologist.

MANAGEMENT

Q29. How is symptomatic mitral stenosis treated in pregnancy?

> Beta-blockers (e.g. metoprolol) to slow the heart rate and diuretics for congestion; rest and treat anaemia.

Q30. What is done if symptoms persist despite medicines?

> Percutaneous balloon mitral valvotomy, ideally in the second trimester.

Q31. How is atrial fibrillation managed?

> Rate control (beta-blockers, digoxin) and anticoagulation with LMWH.

Q32. Which anticoagulant is used for mechanical heart valves in pregnancy?

> Specialist decision: warfarin (lowest valve thrombosis risk) or LMWH with anti-Xa monitoring; warfarin is avoided at 6–12 weeks and near delivery.

Q33. What are the risks of warfarin in pregnancy?

> Embryopathy if used at 6–12 weeks, and fetal intracranial bleeding.

Q34. Which drugs are avoided in pregnancy?

> ACE inhibitors, angiotensin receptor blockers, amiodarone if possible, and statins.

Q35. Why is penicillin prophylaxis continued?

> To prevent recurrence of rheumatic fever.

Q36. What is the preferred mode of delivery?

> Vaginal birth in most cases; caesarean is for obstetric reasons or specific cardiac conditions (e.g. aortic dilatation, severe pulmonary hypertension in some cases).

Q37. How is the second stage managed?

> Shortened with an assisted delivery if pushing is not advised, and with good epidural analgesia.

Q38. Why is epidural analgesia useful?

> It reduces pain and tachycardia, lowering cardiac workload; it is given slowly to avoid hypotension.

Q39. How is the third stage managed?

> With oxytocin given slowly or as a low-dose infusion; ergometrine is avoided as it causes sudden vasoconstriction.

Q40. Why is fluid balance important in labour?

> To avoid fluid overload and pulmonary oedema.

Q41. What position is advised in labour?

> Upright or left lateral; avoid lying flat.

Q42. Where should she be monitored after delivery?

> In a high-dependency unit for at least 24–48 hours, as fluid shifts after delivery can cause pulmonary oedema.

Q43. Is endocarditis prophylaxis routinely needed in delivery?

> Not routinely for vaginal birth in UK guidance; it is considered for high-risk lesions according to local cardiology advice.

COMPLICATIONS AND COUNSELLING

Q44. What are the maternal complications?

> Heart failure, pulmonary oedema, arrhythmia, thromboembolism, valve thrombosis, endocarditis and death.

Q45. What contraception is suitable for women with heart disease?

> Progestogen-only methods and the copper IUD are safe; combined hormonal contraception is avoided in many cardiac conditions.

Q46. What pre-pregnancy advice is given?

> Specialist counselling, optimising the heart (e.g. valve surgery before pregnancy), changing teratogenic drugs, and planning pregnancy timing.

Q47. Is breastfeeding allowed?

> Yes, with most cardiac medicines, including warfarin and beta-blockers.

Q48. What is the risk of recurrence of peripartum cardiomyopathy?

> High, especially if heart function has not recovered; further pregnancy may be advised against.

Q49. What is the take-home message for heart disease in pregnancy?

> Assess risk before pregnancy, joint specialist care, plan delivery, and watch closely after birth.

Q50. What is a recent advance in cardiac disease in pregnancy?

> The ESC 2018 guidelines with the modified WHO classification, and the use of bromocriptine as an added treatment in peripartum cardiomyopathy.