OBSTETRICS & GYNECOLOGY OSCE

Condensed Comprehensive Study Guide

Contents

How to use this guide

SOURCE BOUNDARY: The original obstetrics and gynecology chapters remain source-grounded in the uploaded clinical checklists. Six previously absent syllabus topics have been added from the attached lecture-derived guides.

CONSTRUCTED STATIONS: Because these six topics did not include official OSCE checklists, each new chapter contains three original 20-mark stations. Patient scenarios and marks are constructed; every medical fact, threshold and lecture-specific statement comes from the attached source guide.

REVISION METHOD: Read the student scenario, answer the examiner questions aloud, then memorize the checklist domains and the station memory section. Source contradictions are displayed rather than silently corrected.

Study every chapter in this order

Station spine -> examiner domains -> values and thresholds -> management sequence -> exam traps.

PART I - OBSTETRICS

Checklist-derived chapters plus four constructed three-station topics.

A. Antenatal Booking, Pregnancy Confirmation and Routine Care

Source set: 8- BOOKING .pdf

STATION SPINE: Confirm pregnancy -> calculate dates -> complete booking history/examination -> baseline tests and ultrasound -> aneuploidy screening -> plan routine follow-up and preventive medication.

Pregnancy confirmation and dating

- Amenorrhea plus morning sickness, nausea or vomiting may suggest pregnancy. Confirm with serum/urine hCG and a transvaginal ultrasound showing a gestational sac.
- Calculate the EDD by Naegele rule: first day of LMP - 3 calendar months + 1 year + 7 days. Source worked example: LMP 27/6/2015 -> EDD 4/4/2016.
- Clarify cycle regularity, LMP, how pregnancy was confirmed, contraception before conception and symptoms/complaints such as bleeding, pain, nausea, vomiting, discharge or morning sickness.

Complete booking history

- Age, occupation and exposure to toxins/radiation/medications; gravida, para and outcomes; mode and complications of previous deliveries; previous cesarean/uterine surgery; gynecologic and contraceptive history.
- Medical, surgical, drug and allergy history; previous transfusion; immunization; family/genetic history; smoking and alcohol; current lactation status.
- Ask specifically about diabetes, hypertension and other chronic disease, and any previous pregnancy complication.

Examination and baseline tests

- Weight, height, BMI and blood pressure; general and obstetric examination.
- CBC; blood group and Rh; antibody screen/indirect Coombs; urinalysis plus culture; blood glucose/FBS or GTT; TSH/thyroid function; rubella immunity; hepatitis screening (B, and C where listed); source also lists a high-vaginal swab for GBS at booking.
- If Rh negative: determine husband blood group, ask whether anti-D was given in previous pregnancies, and order indirect Coombs. The notes state that if the antibody screen is negative, anti-D is given at 28 weeks.

Why ultrasound is required at booking

- Confirm pregnancy and location (intrauterine versus ectopic), viability/fetal heart and dating; determine number of fetuses; assess placenta and liquor; identify fetal abnormality, uterine fibroid or ovarian cyst.
- Source scan timetable: booking scan; nuchal translucency at 11-14 weeks; anomaly scan at 18-22 weeks.

Down syndrome screening and confirmation

- Screening options: nuchal translucency, first-trimester biochemistry, triple test and quadruple test. Source note places maternal-serum AFP at 15-20 weeks and gives a normal range of 0.75-2.5.
- Positive screening is confirmed invasively by chorionic-villus sampling or amniocentesis.

Routine antenatal follow-up

- At each visit ask about nausea/vomiting, bleeding, discharge and other problems; measure BP and weight; dip urine for protein, glucose and infection; perform obstetric assessment and ultrasound when indicated.
- Source visit frequency: before 28 weeks monthly; 28-36 weeks every 2-3 weeks; from 37 weeks weekly; at 41-42 weeks every 2-3 days for fetal testing. A separate 8-week station schedules the next visit after 4 weeks.
- Pregnancy trimesters in the notes: 0-13 weeks, 14-27 weeks and 28 weeks to birth.

Routine medication exactly as listed

- Folic acid 500 micrograms daily from day 1 to delivery; another booking station simply advises folic acid at 8 weeks.
- Iron from week 16 to delivery; vitamin D 5,000 IU every other day from day 1 to delivery; calcium 500 mg daily from week 26 to delivery.
- For nausea at 8 weeks: folic acid and an antiemetic are listed.

Source-specific wording retained: several screening, GBS and supplementation details reflect the uploaded notes and are presented without external correction.

Trimester-by-trimester antenatal schedule

- First trimester: establish personal/family and previous-pregnancy history; general examination, BP, weight/BMI; CBC/Hb, ABO/Rh and antibody screen, urine protein/culture, Pap smear, gonorrhea/chlamydia testing and infection screening for rubella, syphilis, HBV, HIV, TB/hepatitis; the annotations also add blood sugar and TSH.
- First-trimester ultrasound: location and viability, number of fetuses, gestational age by crown-rump length and cardiac activity, cervix/uterus/placenta, and nuchal translucency at about 11-14 weeks. The notes label NT <3 mm normal and >3 mm abnormal.
- Second trimester: 50-g 1-hour glucose challenge at 24-28 weeks; if positive, 100-g 3-hour OGTT; repeat Hb/CBC; triple screen (AFP, hCG, estriol) or quadruple screen adding inhibin A; detailed anomaly scan at 18-22 weeks including liquor and cervical length/funneling.
- Third trimester: CBC, 50-g glucose screen where not already performed, urine dipstick for PET, repeat Rh/atypical-antibody screen with anti-D at 28 weeks if negative, and group-B streptococcal vaginal/rectal swab at 36-37 weeks. Ultrasound assesses placental relation to internal os, presentation, BPP, postmaturity calcification, estimated weight and biometry.

Serum screening details retained from the notes

- Raised maternal-serum AFP (>2.5 MoM in the note): consider wrong dates, multiple pregnancy, neural-tube defect, ventral-wall defect such as gastroschisis/omphalocele, renal disease or sacrococcygeal teratoma.
- Low AFP (<0.75-0.85 in the note): consider wrong dates, trisomy or fetal demise.

Gestational-age examination and Leopold maneuvers

- Fundal-height landmarks: symphysis-to-umbilicus corresponds to about 20 weeks; from 20-36 weeks, centimeters above the umbilicus/symphysis are used to estimate gestation; after 36 weeks the fetus may descend. The note highlights fundal height as useful from 20-36 weeks.
- Leopold maneuvers: first identifies fundal contents/lie; second locates the fetal back; third identifies the presenting part and engagement; fourth assesses the fetal brow/head flexion and descent.

Early pregnancy ultrasound landmarks and signs

- The teaching slide lists gestational sac at 4.5-5 weeks, yolk sac at 5-6 weeks and fetal pole at 5.5-6 weeks.
- Pregnancy symptoms/signs listed: amenorrhea, nausea/vomiting, fatigue, breast enlargement and mild cramping; telangiectasia/palmar erythema/linea nigra, Goodell sign, Ladin sign and Chadwick sign.

Additional practical skills flagged in the CVS source

- Obstetric examination, clinical pelvimetry, engagement diameters and speculum examination are explicitly signposted. The same source also names IUCD insertion and Pap smear, which are addressed in the contraception and gynecological-examination sections of the complete guide.

B. Abdominal Pain in Pregnancy and Acute Pelvic Pain

Source set: 22 Abdominal pain.pdf

STATION SPINE: Stability and gestation -> characterize pain -> pregnancy/labor red flags -> urinary, gastrointestinal, gynecologic and obstetric differentials -> focused examination and investigations -> conservative or operative plan.

Acute pelvic pain in a newly married woman

- 19-year-old with left iliac-fossa colicky pain and nausea: ask LMP, urinary symptoms, contraception, abdominal/pelvic surgery, obstetric/gynecologic history and previous PID.
- Differentials: renal colic, torsion of ovarian/adnexal cyst or mass, acute pyelonephritis, ectopic pregnancy and bowel obstruction.
- Essential tests: urinalysis, CBC, pelvic/abdominal ultrasound, supine and erect abdominal X-rays, and pregnancy test.
- If ultrasound shows a simple 6 x 5 cm adnexal cyst: CA125, color-flow Doppler, laparoscopic or open ovarian cystectomy; if no torsion, conservative follow-up and re-evaluation.

Abdominal pain in late pregnancy - decide whether it is labor

- Ask booked/unbooked status, LMP and gestational age, fetal movements, medications and associated show or passage of liquor.
- Labor-pain criteria in the source: regular and painful; rhythmic; increasing intensity and duration; not relieved by analgesia; associated cervical change.
- Evaluate general condition/vital signs, abdominal/obstetric examination, contractions and fetal heart, then vaginal examination when appropriate.

Broad differential at 19-28 weeks

- UTI, renal colic, biliary colic, cholecystitis, intestinal colic/obstruction, appendicitis, ovarian cyst/torsion/tumor, degenerating fibroid and placental abruption.
- Focused pain history: site, onset, character, radiation, severity, progression, aggravating/relieving factors and associated fever, nausea/vomiting, bowel/urinary symptoms, vaginal bleeding, fluid loss, contractions and fetal movement.
- One 28-week simulator describes gradual dull supraumbilical pain with nausea but no fever, GI/urinary symptoms, labor pain, fluid loss, bleeding, hypertension or proteinuria; the guide preserves this as a differential-building scenario rather than assigning an unsupported final diagnosis.

C. Cardiotocography and Intrapartum Fetal Monitoring

Source set: 1- CTG .pdf; 9- Fetal well being .pdf

STATION SPINE: Confirm patient/date/paper speed -> contractions -> baseline -> variability -> accelerations -> decelerations -> classify the pattern -> correct reversible causes -> examine and expedite delivery when persistent.

Intrapartum surveillance methods

- FHR monitoring, fetal-scalp blood pH, intrapartum fetal stimulation tests, fetal ECG, scalp lactate and continuous biochemical/pulse-oximetry approaches are listed.
- Intermittent auscultation: at least every 30 minutes in first stage and every 15 minutes in second stage; abnormal findings require continuous electronic monitoring.
- External EFM is noninvasive but may be difficult with obesity or polyhydramnios. Internal monitoring is used for inadequate external tracing; requires cervical dilation and amniotomy and may increase infection/fetal injury or viral transmission. Contraindications listed: placenta previa, face or unknown presentation, HIV and active genital herpes.

CTG components and contractions

- CTG records fetal heart rate and uterine contractions. Interpret patient ID, paper speed, date/time, baseline, variability, accelerations, decelerations and contractions.
- Normal resting uterine tone 5-10 mmHg, rising to 10-15 in labor; contraction pressure about 25-100 mmHg. Resting pressure above 20 mmHg reduces uterine perfusion.
- Hyperstimulation/tachysystole: more than 5 contractions in 10 minutes, risking hypoxia, acidosis and abnormal FHR.

Baseline, variability and sinusoidal pattern

- Normal baseline 110-160 bpm. Tachycardia causes: maternal pyrexia/chorioamnionitis, fetal anemia or hypoxia, beta-agonists and fetal SVT/arrhythmia.
- Bradycardia causes: maternal hypothermia, hypotension or hypoglycemia; beta-blockers; fetal AV block including SSA/Ro-associated disease; fetal metabolic acidosis.
- Normal variability 5-25 bpm. Reduced variability: fetal sleep, hypoxia/acidosis, tachycardia, opiates, benzodiazepines, methyldopa, magnesium sulfate, betamethasone, prematurity and congenital heart disease.
- Sinusoidal pattern: smooth regular waves, 2-5 cycles/min, stable baseline 120-160 with no beat-to-beat variability; associated with severe hypoxia/anemia, fetomaternal hemorrhage and ruptured vasa previa.

Accelerations and decelerations

Pattern	Definition/cause	Meaning and source action
Acceleration	Abrupt rise >15 bpm for >15 sec.	Reassuring and suggests a responsive fetus without hypoxia/acidosis.
Early deceleration	Abrupt fall >15 bpm for >15 sec, caused by head compression.	Not pathological in the source.
Late deceleration	Uteroplacental insufficiency; causes include tachysystole, maternal hypotension, regional anesthesia, IUGR, infection and abruption.	Left lateral position, IV fluids, oxygen, stop oxytocin, consider tocolysis, vaginal examination, scalp pH if persistent, then immediate delivery if unresolved.
Variable deceleration	Cord compression; abrupt V-shaped onset/recovery and variable timing/depth.	Position change, IV hydration, oxygen, reduce/stop oxytocin, consider amnioinfusion or tocolysis; digital elevation of head if cord pressure/prolapse. Severe source rule: below 60 bpm for >=60 sec (60 x 60) and persistent -> prompt delivery.
Prolonged deceleration	Usually >3 minutes.	With reduced variability indicates acute hypoxia/acidosis and needs emergency intervention. Rule of 3s: 3 min call for help, 6 min move to theatre, 9 min prepare delivery.

Sudden fetal bradycardia and station actions

- Non-correctable/major causes listed: uterine hyperstimulation from oxytocin/prostaglandin, abruption, cord prolapse, uterine rupture and vasa previa. Correctable causes: epidural/spinal or paracervical block and supine-hypotension-related maternal hypotension.
- 41-week induction station with reduced variability plus late decelerations: stop Syntocinon, IV hydration, correct maternal position and perform vaginal examination. If cervix 4 cm, 60% effaced, vertex at -2: urgent cesarean section.
- Variable-deceleration station: stop Syntocinon, left lateral position, hydration and vaginal examination to exclude cord prolapse and determine delivery method.

NST interpretation and indications

- Reactive NST in the source: baseline 110-160, variability 5-25, accelerations during a 20-minute observation and no decelerations.
- Indications listed: advanced maternal age, diabetes, hypertension, post-dates, reduced fetal movements and reduced amniotic fluid.

NST, CST and BPP interpretation

- Reactive NST: at least two accelerations of >=15 bpm lasting >=15 seconds in 20 minutes, normal baseline/variability and no decelerations. Before 32 weeks, the slide uses two accelerations of >=10 bpm for >=10 seconds in 20 minutes.
- Nonreactive NST: continue up to 40 minutes to allow for fetal sleep; use vibroacoustic stimulation, then BPP if still nonreactive.
- CST stimulates contractions with low-dose oxytocin until about three palpable contractions lasting >=40 seconds occur in 10 minutes. Negative/reactive CST has no late decelerations; positive/nonreactive CST has late decelerations and is treated in the source as an indication for delivery.
- BPP components: NST, AFI, fetal breathing, body movement and tone, each scored 0 or 2. Breathing earns 2 for >=1 episode lasting >=20 seconds; movement for >=2 movements in <=30 minutes; tone for >=1 episode. Score 8-10 reassuring; 4-6 concerning (>=36 weeks deliver; <36 repeat in 12-24 h or CST); 0-2 ominous/prompt delivery. Modified BPP uses NST plus AFI.

Additional deceleration and bradycardia details

- Occasional mild/moderate variable decelerations are common; severe variable decelerations fall below 60 bpm for at least 60 seconds (the source “60 x 60” rule) and persistent non-remediable cases require prompt delivery.
- Acute bradycardia causes listed as not readily correctable: uterine hyperstimulation from oxytocin/prostaglandin, abruption, cord prolapse, uterine rupture and vasa previa. Correctable causes: epidural/spinal or paracervical block and maternal supine hypotension.
- Prolonged deceleration lasts >3 minutes. “Rule of 3s”: at 3 minutes call for help, at 6 minutes move to theatre, at 9 minutes prepare for delivery.
- For variable decelerations the vaginal examination aims to exclude cord prolapse and determine delivery route.

Fetal scalp sampling and stimulation

- Fetal-scalp pH/lactate table: normal pH >=7.25 and lactate <4.2 mmol/L; repeat in 30 minutes for pH 7.21-7.24 or lactate 4.2-4.8; expedite birth for pH <=7.20 or lactate >4.8; urgent delivery for pH <7.15 or lactate >5.0.
- Fetal stimulation methods listed: digital scalp stimulation, vibroacoustic stimulation, fetal scalp puncture and transabdominal light stimulation. An acceleration after stimulation is reassuring.

D. Contraception and Family Planning

Source set: 10- contraception .pdf

STATION SPINE: Identify patient goals and contraindications -> explain mechanism/use/effectiveness -> manage missed pills -> compare combined, progestogen-only, IUD and permanent methods -> counsel return to fertility and complications.

Combined hormonal contraception

- Forms: combined oral pills, transdermal patch and vaginal ring; other routes listed include injectable. Contains ethinyl estradiol plus progestogen.
- Mechanism: estrogen suppresses the midcycle gonadotropin surge so ovulation does not occur. Pack description: 21 active pills, or 28-pill packs with 21 active and 7 placebo pills.
- Start first day of cycle with no backup; otherwise may start any day after excluding pregnancy and use backup for 7 days. Take at the same time daily; source station describes 21 days then 7-day rest and starting the next pack on day 5 of the cycle.
- Perfect-use pregnancy rate <1%; usual-use rate about 8% (source range 5-10%). Main failure reason: noncompliance. Return to fertility is immediate; no routine pill-free “rest” is required; source states no proven weight gain or teratogenic effect and no serious adverse effect on sexual relationship.

Missed-pill instructions exactly as listed

- One missed pill: take as soon as remembered and continue the pack/on-time pill.
- Two missed pills: source examiner says the same approach.
- Three or more missed pills or a 3-day interruption: use emergency/backup contraception and start a new pack after withdrawal bleeding; another station says backup plus start a new pack.
- Vomiting within 2 hours: take another pill from the pack as soon as possible and continue regularly.

Benefits, adverse effects and contraindications

- Noncontraceptive benefits listed: dysmenorrhea, menorrhagia/DUB, endometriosis, simple ovarian cysts, acne/hirsutism; reduced PID/ectopic pregnancy and reduced ovarian, endometrial and colon cancer; less osteoporosis and benign breast disease.
- Estrogen-related effects: VTE/PE, CVA/MI, nausea/vomiting, migraine/headache, tiredness, fluid retention/bloating, breast changes and libido/cervical changes. Progestogen-related: breakthrough bleeding, acne, alopecia, weight gain, irritability/depression, hypertension, cholestasis and breast tenderness. Patch may irritate skin; ring may cause discomfort, discharge or recurrent vaginitis.
- Absolute/source contraindications: breastfeeding, known/suspected pregnancy, smoker >15 cigarettes/day and age >35, migraine with aura, severe/uncontrolled hypertension (>160/100 or vascular disease), current IHD/stroke/VTE/DVT/PE or thrombophilia, abnormal LFT, breast/endometrial cancer, SLE and unexplained vaginal bleeding.

Progestogen-only methods and IUD

- Progestogen-only/minipill is listed for women in whom estrogen is unsuitable, especially lactation; source effectiveness figure is 92%. Contraindications shown include suspected pregnancy and undiagnosed bleeding.
- IUD mechanism: sterile inflammatory reaction, prevention of implantation, reduced tubal motility and thicker cervical mucus. Paragard duration 10 years (notes say up to 14); Mirena 5 years (up to 7).
- IUD complications/figures in the notes: dysmenorrhea/menorrhagia for 3-6 months; 15% discontinuation; perforation 2/1000; expulsion 1/20; rare pregnancy with risk of early/mid-trimester loss and preterm delivery; ectopic absolute risk 1.5/1000 years of use.

Tubal ligation counseling

- Approaches named: laparoscopy, laparotomy and hysteroscopic route. Failure <0.5 per hundred woman-years in the source.
- Failure may reflect pregnancy before procedure, tubal recanalization or ligation of another structure. It does not itself increase ectopic risk according to the marked answer.
- Benefits: effective and no hormonal side effects. Disadvantages: surgical risk and intended irreversibility. Source answers state no menstrual-pattern effect and that reversal is not easy.

Source conflict retained: missed-pill and pack-start instructions vary across uploaded stations; each source version is recorded rather than replaced by a modern external algorithm.

Contraceptive goals, emergency contraception and efficacy

- Benefits listed: fewer unintended pregnancies/abortions, health and social benefits for mother/children, reduced postpartum depression and therapeutic effects for heavy menses, acne, hirsutism and endometriosis; reduced ovarian/endometrial cancer risk is also stated.
- Emergency contraception options in the source: progesterone-receptor-modulator pill within 5 days, progestogen pill within 3 days, or copper IUD within 5 days of intercourse.
- Efficacy is expressed as pregnancies per 100 women per year.

Natural methods and barrier methods

- Periodic abstinence/fertility awareness: calendar/rhythm method (shortly before and after ovulation), ovulation-prediction kits detecting the LH surge, basal-body temperature and cervical-mucus assessment; source effectiveness 50-80%.
- Coitus interruptus: withdrawal before ejaculation; source effectiveness 27%, with high failure and dependence on self-control.
- Lactational amenorrhea: prolactin suppresses GnRH/ovulation; criteria are exclusive breastfeeding, infant <=6 months and feeding at least every 4 hours by day and 6 hours at night. No cost/no effect on nursing, but low actual efficacy in the notes.
- Barrier methods/spermicides are the only methods stated to protect against STDs. Male condom source effectiveness 85-90% and female condom about 80%; latex rupture/hypersensitivity and reduced sensation are noted. Diaphragm and cervical cap are placed before intercourse and left 6-8 hours after; source effectiveness about 80%. Diaphragm risks include vaginal injury, staphylococcal colonization/toxic shock and latex allergy.

Progestogen-only injections and implants

- Minipill: lower progestin dose, taken daily for 28 days; mechanism includes cervical-mucus thickening, ovulation suppression and endometrial atrophy.
- Depo-Provera: IM every 3 months; source efficacy 99.7%. Disadvantages include amenorrhea, weight gain/bone-mineral-density concern and delayed return of ovulation about 6-8 months after stopping.
- Implanon: subcutaneous, effective after 24 hours and provides about 3 years of contraception; rapid return to fertility after removal, but requires clinician insertion/removal and has progestin effects.

IUD indications, contraindications and permanent male sterilization

- IUD advantages: long-term, cost-effective, early reversibility and possible immediate insertion after spontaneous first-trimester abortion. Indications include estrogen contraindication, long-term protection, low STI risk and treatment of menorrhagia/dysmenorrhea.
- Absolute/source contraindications include pregnancy, unexplained bleeding, pelvic infection and copper/Wilson disease for copper IUD. Relative/source items include recent STI, uterine anomaly/fibroid/nulliparity; annotations also list endometrial/cervical cancer and active liver/breast disease for LNG-IUD.
- Vasectomy: office ligation of vas deferens under local anesthesia; not immediately effective. Continue another method for 6-8 weeks until azoospermia is confirmed by semen analyses (three in the notes). It is described as safe, simple, inexpensive and more effective.

1. Positive Pregnancy Test, Empty Uterus and Pregnancy of Unknown Location

Source set: 23 Positive pregnancy test .pdf

STATION SPINE: Confirm stability -> identify pregnancy of unknown location -> use quantitative beta-hCG and transvaginal ultrasound -> repeat in 48 hours -> interpret rise/fall and hCG ratio -> rescan or manage according to the source algorithm.

Original examiner station

- Stable 24-year-old with mild abdominal pain, vaginal bleeding, 7 weeks of amenorrhea, positive pregnancy test and an empty uterus on ultrasound.
- Three checklist differentials: early pregnancy, complete abortion and ectopic pregnancy.
- Differentiating test: quantitative beta-hCG. Given value 800 mIU/mL -> repeat after 48 hours.
- Checklist interpretation: decreased = most likely complete abortion; doubling = most likely early pregnancy; abnormal increase = think of ectopic pregnancy.

Pregnancy testing and beta-hCG facts in the teaching pages

- hCG is detected with antibodies directed at its beta subunit. Serum testing is the most sensitive method, detects about 1-2 mIU/mL and can become positive within one week of conception. Urine testing has a source threshold of 20-50 mIU/mL and may not become positive until two weeks or more.
- Beta-hCG is a glycoprotein with a source-stated half-life of 24 hours. Values <5 IU/mL are labeled not pregnant and >25 IU/mL pregnant.
- About 85% of early pregnancies are stated to double every 48 hours. The notes also state a doubling time of about 1.4 days before 5 weeks and 2.4 days up to 7 weeks.
- The teaching slide says beta-hCG should rise at least 60% over 48 hours; another source statement uses >63%, while the PUL algorithm uses >66%. These source thresholds are retained separately.
- Beta-hCG peaks at about 100,000 mIU/mL at 8-10 weeks, declines to about 12,000 mIU/mL at 20 weeks, has a wide range of peak/plateau values, cannot be used alone to date pregnancy and is used clinically for diagnosis rather than routine late-pregnancy measurement.

Ultrasound and clinical evidence of pregnancy

- Transvaginal ultrasound follows a positive beta-hCG test, confirms an intrauterine pregnancy and is described as the most accurate method of first-trimester dating.
- Source ultrasound landmarks: gestational sac 4.5-5 weeks, yolk sac 5-6 weeks and fetal pole 5.5-6 weeks.
- Clinical symptoms listed: amenorrhea, nausea with or without vomiting, general fatigue, breast enlargement and mild uterine cramping.
- Examination findings listed: telangiectasias, palmar erythema, linea nigra, cervical softening (Goodell sign), uterine softening (Ladin sign) and blue discoloration of vagina/cervix (Chadwick sign).

48-hour interpretation and PUL ratios

- If beta-hCG rises >63% after 48 hours, the source labels an intrauterine pregnancy likely and advises repeat scan in one week.
- PUL hCG ratio = beta-hCG at 48 hours divided by beta-hCG at 0 hours: <0.8 is labeled missed miscarriage; 0.8-1.66 is labeled ectopic pregnancy or persistent PUL; >1.66 is labeled intrauterine pregnancy. The slide then lists expectant or medical treatment.
- In the larger PUL algorithm: >66% increase/ratio >1.66 with progesterone >16-80 nmol/L suggests IUP; <66% increase/ratio <1.66 or <15% decrease/ratio >0.87 suggests ectopic pregnancy while stable; >15% decrease/ratio <0.87 with progesterone <16 nmol/L suggests failing PUL.
- A single beta-hCG has little value when ultrasound is confusing; serial beta-hCG is required. The side note states that when hCG is <2,000 IU/L, doubling time helps predict viable versus nonviable pregnancy; a rise <66% in 48 hours suggests ectopic or nonviable intrauterine pregnancy. The source defines biochemical pregnancy as two beta-hCG values >10 IU/L.

PUL pathway exactly as depicted

- Positive urine pregnancy test/suspected ectopic -> transvaginal ultrasound -> PUL. Pain-free patients enter outpatient expectant management; patients with pain enter inpatient management. Measure serum beta-hCG at 0 and 48 hours plus serum progesterone.
- For a likely IUP or stable possible ectopic, rescan after one week. If an early intrauterine pregnancy is visualized, rescan in two weeks to confirm viability. If ectopic pregnancy is visualized, manage as clinically indicated. If location remains unknown, repeat beta-hCG again 48 hours later.

- For a failing PUL, repeat serum beta-hCG in one week, consider weekly monitoring until <20 IU/L, and if no pregnancy is seen on repeat scan with a suboptimal beta-hCG rise, consider methotrexate.

Diagnostic-curettage branch shown in the source

- If chorionic villi are present: continue serial quantitative beta-hCG; rising levels prompt a search for coexisting ectopic pregnancy, whereas a decline of at least 15% is labeled incomplete abortion in the diagram.
- If villi are absent: continue serial beta-hCG and transvaginal ultrasound. Declining levels are followed to <20 IU/L. Rising/plateaued beta-hCG with ectopic mass <3.5 cm leads to medical treatment; a mass >=3.5 cm leads to laparoscopy or laparotomy in the source diagram.

Source table: change in beta-hCG over 48 hours

Clinical outcome	Change pattern	Percentage of cases
Intrauterine pregnancy	Appropriate increase >=50%	99%
Intrauterine pregnancy	Inappropriate increase <50%	1%
Ectopic pregnancy	Inappropriate increase or decrease	71%
Ectopic pregnancy	Increase similar to viable IUP >=50%	21%
Ectopic pregnancy	Decrease similar to spontaneous abortion <50%	8%
Spontaneous abortion	Appropriate decrease >=35%	90%
Spontaneous abortion	Inappropriate decrease <35%	10%

- The table footnote states that the decline after spontaneous abortion depends on the initial beta-hCG. Average decline in the first 48 hours is usually >70%; a decline <21-35% (depending on initial level) is inappropriate and ectopic pregnancy should be suspected.
- The source also includes illustrative curves showing beta-hCG peaking in the first trimester and then falling, and falling after miscarriage/abortion; these illustrations are retained as qualitative trends rather than converted into new thresholds.

Source-threshold warning: the uploaded pages contain several different “appropriate rise” cutoffs (50%, 60%, 63% and 66%) and two ratio schemes. They are presented as source-specific statements rather than silently reconciled.

2. Ectopic Pregnancy

Source set: 1 Ectopic.pdf

STATION SPINE: Stability first → confirm pregnancy location with serial β-hCG and ultrasound → screen for rupture → choose observation, methotrexate, laparoscopy, or laparotomy using the source criteria.

Definition, frequency, implantation and sites

- Ectopic pregnancy is implantation outside the uterine cavity. The source describes it as a leading cause of maternal mortality and separately gives an incidence of 2% in one checklist and a 6% mortality-related figure in the teaching notes.
- Implantation is stated to occur around day 6 after fertilization.
- Tubal sites account for about 95%: ampulla is most common (about 5–6 mm and symptoms commonly at 6–7 weeks), followed by isthmus, fimbrial, and cornual/interstitial sites. The cornual site may accommodate pregnancy to about 10 weeks and is described as the most dangerous when rupture occurs.
- Other source-listed sites: ovary about 3%, abdominal cavity about 1%, and cervix as a rare site.

Station presentations and immediate safety question

- Station variant: 32-year-old with left lower-quadrant pain, vaginal spotting, and a positive pregnancy test. Before attending, ask: Is she hemodynamically stable?
- Mrs. XX: 33 years, G7P2+4, two previous ectopic pregnancies, two surgical deliveries, IUCD for 2 years, successful IVF; pain, dizziness, heavy creamy discharge and spotting; vaginal ultrasound shows marked endometrial thickness with no definite intrauterine sac; pregnancy hormone 647 mIU/mL.
- Another source case gives nausea, abdominal pain, spotting, a thick endometrium, left adnexal mass, and β-hCG 871 IU/mL.

Focused history

- Establish gravida, parity, abortions, prior ectopic pregnancies, LNMP/LMP, cycle regularity, pregnancy-test result, and any ultrasound or β-hCG result.
- Pain: onset, location, character, severity, duration, progression, aggravating/relieving factors, back pain, and shoulder-tip pain.
- Bleeding: spotting versus bleeding, quantity, color, clots, passage of tissue, and associated discharge.
- Associated features: dizziness or faintness, nausea/vomiting, diarrhea, urinary symptoms, and collapse symptoms.
- Complete medical, surgical, gynecologic, obstetric, drug and allergy histories, with explicit ectopic risk-factor questions.

Risk factors and source pathophysiology

Risk-factor group	Source-listed details
Tubal damage/infection	PID, sexually transmitted infection, tuberculosis, damaged tubal cilia, intratubal/peritubal adhesions, and partial obstruction.
Previous events/procedures	Previous ectopic pregnancy (the source gives recurrence up to 30%), pelvic/tubal/abdominal surgery, tubal ligation or reconstructive surgery.
Fertility/contraception	Infertility treatment, fertility drugs, IVF/ART, and an IUCD.
Other	Smoking, DES exposure, congenital uterine malformation, and endometriosis.

- Source pathophysiology sequence: infection damages tubal cilia → egg transport is disrupted; pocket-like pools may engulf the fertilized egg; infection-related or bleeding-related scarring causes partial tubal blockage.

Clinical features and rupture screen

- Classic source triad 4–6 weeks after the LMP: amenorrhea, acute pelvic/lower-abdominal pain (which may radiate to the ipsilateral shoulder), and vaginal spotting.
- Examination: vital signs/hemodynamic status, abdominal examination, pelvic/bimanual examination for adnexal mass or tenderness; cervical-motion tenderness is also listed and is especially associated with PID in the source notes.
- Features suggesting rupture: sudden severe sharp pain, shoulder-tip pain, faintness/dizziness, hemorrhagic shock, diarrhea, intraperitoneal bleeding and collapse.

Investigations and source thresholds

- Quantitative β-hCG and transvaginal ultrasound are central. The source states that a normal intrauterine pregnancy doubles β-hCG after approximately 72 hours; an increase without doubling suggests ectopic pregnancy.

- Ultrasound clues: empty uterine cavity with thick endometrium, free fluid in the pouch of Douglas, tubal ring/blob, or an adnexal mass.
- Source discriminatory zones: 1,500–2,000 mIU/mL for transvaginal ultrasound, up to 2,300 with multiple gestation; 6,000–6,500 mIU/mL for transabdominal ultrasound. Above the zone with no visible intrauterine pregnancy is interpreted by the source as ectopic pregnancy or recent abortion.
- Laparoscopy is listed for diagnosis and treatment; the notes state laparotomy/urgent operative assessment for an unstable patient.

Differential diagnosis

- Appendicitis; salpingitis/PID or tubo-ovarian abscess; ruptured corpus-luteum cyst or ovarian follicle; spontaneous, complete, or threatened abortion; ovarian torsion; urinary-tract disease; normal early intrauterine pregnancy; molar pregnancy; and a degenerating fibroid.

Management options and applications

Option	Source application/criteria
Observation	Stable vital signs, acceptable pain, low hormonal level; one source uses β-hCG below 1,000 mIU/mL and notes that some resolve without intervention.
Methotrexate	Stable, unruptured, low hormone level, no fetal cardiac activity, and mass no more than 4 cm. One teaching page uses β-hCG <1,500; an older checklist simply says low level/stable.
Methotrexate contraindications	Hemodynamic instability or rupture; leukopenia or platelets <100,000; active renal/hepatic disease; active peptic-ulcer disease; breastfeeding; positive fetal heart activity.
Laparoscopy	Diagnosis and management in a stable patient; salpingectomy versus salpingostomy is listed.
Laparotomy	Urgent surgery for life-threatening bleeding or instability.
Surgical triggers	Failure of medical treatment, instability, positive fetal heart activity, mass >4 cm, or source β-hCG >1,500.

Source conflict retained: the uploaded pages use more than one β-hCG threshold for observation/methotrexate. These values are recorded as written rather than silently reconciled.

Examiner checklist traps

- Answer the stability question before taking a detailed history.
- Link each management modality to its application; merely naming four options is incomplete.
- State every differential and risk factor independently. One checklist asks for any five risk factors but lists six.

3. Miscarriage, Recurrent Miscarriage and Cervical Incompetence

Source set: 11- miscarriage.pdf

STATION SPINE: Classify the loss -> assess stability, bleeding and pain -> confirm with hCG/ultrasound -> identify recurrent-loss cause -> recognize painless mid-trimester cervical weakness -> apply the source cerclage plan.

Miscarriage classification and diagnosis

- Threatened; inevitable; incomplete (source note: retained products >15 mm); complete (<15 mm); missed miscarriage/early fetal demise with an intact gestational sac.
- History: LMP/cycle regularity and contraception, quantitative hCG and ultrasound, bleeding, pain, diarrhea, urinary symptoms, passage of tissue or vesicles, obstetric/gynecologic risk factors, diabetes and medications.
- Assess hemodynamic stability and examination findings before selecting expectant, medical or surgical treatment; detailed treatment thresholds were not fully visible in the uploaded pages and are not invented here.

Recurrent miscarriage history and cause-test pairs

- Medical history: diabetes, thyroid disease and thrombosis. For every loss: number, timing, type, pain, early ROM, need for evacuation and whether fetal heart was documented.
- Gynecologic cycle/contraception, drugs, occupational hazards, smoking, consanguinity and family history of thrombosis/recurrent pregnancy loss.
- Uterine anomalies -> HSG and hysteroscopy; chromosomal -> karyotypes; endocrine -> TSH, prolactin and FBS; acquired thrombophilia -> lupus anticoagulant and anticardiolipin antibodies; inherited thrombophilia -> protein S/C, antithrombin III, factor V Leiden, factor II and MTHFR. Immunologic causes and inadequate luteal phase are listed without paired tests.

Cervical incompetence recognition

- Painless/recurrent second-trimester loss, sudden ROM/gush with passage of products, cervical dilation/effacement or bulging membranes; may present with heaviness, bleeding, discharge or an incidental finding.
- Risk factors: previous cervical incompetence/late loss/preterm birth or family history; D&C;, cone biopsy/colposcopy and other cervical surgery; large baby, instrumental/traumatic delivery and cervical injury; uterine anomalies/DES exposure and collagen disease.
- Pregnancy assessment: ultrasound cervical length and funneling. Nonpregnant tests listed: HSG showing funneling and serial Hegar dilators.

Source management

- Mrs Reem: HSG, Hegar test, cervical stitch and ultrasound follow-up of cervical length in the next pregnancy.
- Acute 20-week case with cervix 4 cm and bulging membranes: admission/bed rest and emergency cerclage.
- Extended notes: elective cerclage at 12-14 weeks after excluding chromosomal abnormality, remove at 36-38 weeks. Previable current case: expectant care, emergency cerclage or termination; viable case: bed rest, betamethasone and tocolysis if contracting, with cerclage considered.

Source wording retained: Hegar testing and some cerclage timing/management statements are reproduced from the uploaded notes without external reconciliation.

Definition, ultrasound milestones and miscarriage risk factors

- Miscarriage is defined in the source as spontaneous pregnancy loss at or before 24 weeks; most occurs in the first trimester. Pregnancy of unknown location means no pregnancy is visible inside or outside the uterus.
- Ultrasound milestones shown: gestational sac around week 5, yolk sac week 6 and fetal pole thereafter.
- Risk factors/causes listed: increasing maternal and paternal age (the note quotes risk 20% at age 20 and 93% at 45), previous losses, poorly controlled diabetes, thyroid disease, PCOS, smoking, thrombophilia/antiphospholipid syndrome, trisomy/monosomy/triploidy or parental balanced translocation, bacterial vaginosis/varicella/rubella, alcohol, methotrexate/antiepileptics, uterine anomalies/fibroids/intrauterine adhesions and cervical incompetence.

Clinical types and source treatment

- Threatened: bleeding with viable pregnancy, little/no pain, closed os and fetal heart present; 50-75% continue. Reassure/observe; progesterone is listed.
- Inevitable: bleeding, pain and increased contractions with open os and tissue still present; evacuate products.
- Incomplete: bleeding/pain with partial expulsion and retained tissue >15 mm; evacuation or misoprostol. Source complications: renal/hepatic failure, DIC and death in severe retained-product/septic cases.

- Complete: complete expulsion, cessation of bleeding, closed cervix, empty uterus or tissue <15 mm and falling hCG; reassure.
- Missed/early fetal demise: minimal symptoms, closed cervix, falling hCG; criteria displayed include mean sac >25 mm with no yolk sac/fetal pole or CRL >7 mm without cardiac activity. Management: expectant, misoprostol or surgical.
- Septic miscarriage: fever, purulent offensive discharge, tachycardia and tender uterus; organisms listed E. coli, Streptococcus faecalis and Klebsiella. Give antibiotics for 12 hours then evacuate.

Detailed assessment and evacuation

- Examination: vital signs/consciousness, abdominal palpation for mass/distension/pain, vaginal examination of cervical os, and send passed tissue for histopathology to distinguish miscarriage, ectopic or GTD.
- If ultrasound and dates disagree, rescan after 7 days. Additional diagnostic slide thresholds: mean sac 8 mm without yolk sac or 16 mm without embryo, and CRL 5 mm without fetal cardiac activity; these conflict with later criteria and are retained as source versions.
- Misoprostol is described as a prostaglandin analogue that ripens/dilates cervix and produces myometrial contractions; mifepristone is annotated.
- Surgical ERPC/D&C; indications: persistent excessive bleeding, hemodynamic compromise, infected retained products, suspicion of GTD or patient preference. Steps shown: lithotomy, grasp/dilate cervix with Hegar 8-10 mm, suction curette, oxytocin, histopathology and psychological support. Complications: perforation, transfusion, repeated evacuation, infection and rare cervical trauma.
- Anti-D: source says give to all nonsensitized Rh-negative women after >12-week loss and after instrumentation regardless of gestation.

Cerclage types, complications and removal

- Transvaginal McDonald stitch is at the cervicovaginal junction; Shirodkar is submucosal at the internal os. Transabdominal cerclage is permanent, considered after failed vaginal stitches, and requires cesarean delivery.
- Cerclage complications: infection/discharge, membrane rupture, preterm labor and cervical laceration/trauma. Remove urgently for labor, ROM, infection or intrauterine fetal death.

4. Gestational Trophoblastic Disease and Molar Pregnancy

Source set: 2 Molar pregnancy.pdf

STATION SPINE: Recognize high-hCG/abnormal early pregnancy → distinguish complete and partial mole → evacuate and stage → follow serial hCG → identify persistent GTN and select chemotherapy by FIGO score.

Disease spectrum and source classification

- Gestational trophoblastic disease (GTD) is abnormal trophoblastic proliferation associated with β-hCG. The source emphasizes marked chemosensitivity, high curability and possible fertility preservation.
- Hydatidiform mole is listed as about 80% of GTD and usually benign: complete/classic mole about 90% and partial/incomplete mole about 10%.
- Persistent or invasive mole is listed as 10–15%; choriocarcinoma 2–5%; placental-site trophoblastic tumor (PSTT) is rare and malignant.
- Malignant trophoblastic disease commonly metastasizes, especially to the lungs.

Risk factors

- Previous GTD; maternal age under 20 or over 35; nulliparity (the source gives 70%); Asian ethnicity; blood group A; infertility; smoking/lifestyle factors; low β-carotene, folate or animal-fat intake; and oral-contraceptive use.

Complete versus partial mole

Feature	Complete mole	Partial mole
Genetics/mechanism	Usually 46XX paternal; empty ovum fertilized by one haploid sperm that duplicates (75–80%) or by two sperm (20–25%).	Usually triploid, commonly 69XXY, following dispermic fertilization of an ovum.
Fetal tissue	No fetal tissue or fetal red cells.	Fetal tissue or a defective triploid fetus may coexist.
Pathology	Diffuse hydropic “grape-like” villi with marked trophoblastic hyperplasia.	Focal changes with less trophoblastic proliferation.
Typical presentation	Bleeding, uterus larger than dates, very high hCG (>100,000 in the source), hyperemesis, hyperthyroidism, preeclampsia before 20–24 weeks, bilateral theca-lutein cysts, absent fetal parts/FHT, expulsion of swollen villi.	Often resembles missed abortion; uterus may equal dates; hCG is only slightly elevated; bleeding/tenderness may occur.
Ultrasound	Snowstorm/honeycomb/bunch-of-grapes pattern, no fetus or FHT.	Fetal parts/FHT may be visible, fluid is present and the placenta may be thickened.
Malignant potential	Invasive mole 5–25%; choriocarcinoma about 4% in one source table.	Invasive disease 2–4%; source states no choriocarcinoma.
hCG normalization note	Source teaching note says about 14 weeks.	Source teaching note says about 8 weeks.

Station presentation and additional signs

- 40-year-old at 9 weeks with vaginal bleeding and an ultrasound image; expected diagnosis is molar pregnancy.
- Another case: 20-year-old, married 6 months, 8 weeks amenorrhea, excessive vomiting for 3 weeks, slight bleeding for 2 days, β-hCG 150,000 IU, fundus 2 cm above the symphysis.
- Additional source signs: lower-abdominal pain, ovarian enlargement, absent fetal heart and fetal parts, toxemia before 24 weeks, hyperthyroidism and passage of swollen villi.

Investigation and preparation

- Quantitative β-hCG; pelvic ultrasound; CBC and platelets; blood group/Rh and crossmatch; coagulation profile; thyroid function; liver and kidney function; chest X-ray.
- The extended notes also list CT or other assessment of brain, kidney, liver and lung/metastatic sites when indicated, plus histopathology after evacuation.
- Ultrasound suggests the diagnosis, while uterine evacuation with histopathology is the definitive source confirmation.

Immediate management and surveillance

- Admit and perform suction evacuation followed by sharp curettage, regardless of duration of pregnancy in the source notes.
- One source warns against oxytocin “at present” during the initial management.

- Serial β-hCG: one checklist says weekly until normal for two values, then monthly for 6–12 months; another teaching page says weekly until three consecutive normals, then monthly until three consecutive normals.
- Use effective contraception during surveillance. Source counseling includes contraception for one year and the possibility of chemotherapy.

Persistent GTN after non-molar pregnancy

- Any woman with persistent vaginal bleeding after a pregnancy event is at risk; perform a urine pregnancy test and assess symptoms of metastatic disease.
- Vaginal bleeding is the most common presenting symptom. Prognosis after non-molar pregnancy may be worse because of delayed diagnosis.

Optimum follow-up and treatment in the uploaded notes

- If hCG returns to normal within 56 days of the pregnancy event: follow for 6 months from uterine evacuation.
- If hCG has not normalized within 56 days: follow for 6 months from hCG normalization.
- Chemotherapy is required in about 15% after complete mole and 0.5% after partial mole.
- FIGO score ≤6: low-risk disease → single-agent chemotherapy. FIGO score ≥7: multi-agent chemotherapy. PSTT → surgery only in the source.

FIGO scoring components

- Age; antecedent pregnancy; interval in months from end of index pregnancy to treatment; pretreatment serum hCG; largest tumor size including uterus; site of metastases; number of metastases; previous failed chemotherapy.

Long-term counseling

- After chemotherapy, avoid conception for 1 year. The source notes an earlier menopause and increased risk of secondary cancers: myeloid leukemia, colon cancer, melanoma and breast cancer.
- Use barrier contraception until hCG is normal; once normal, the combined oral contraceptive pill may be used. Hormone replacement therapy is stated to be safe after hCG normalization.
- Source survival notes: 95–100% in good-prognosis disease and 50–70% in poor-prognosis disease; deaths are often associated with brain or liver metastases.

> Source conflict retained: the files contain different serial-hCG schedules and slightly different normalization durations. Both versions are preserved as written.

5. Multiple Gestation

Source set: 15-multiple gestation .pdf

STATION SPINE: Suspect a large-for-dates uterus -> define chorionicity/amnionicity -> anticipate maternal, fetal and placental complications -> plan monitored delivery and protect the second twin.

Large-for-dates differential and membrane timing

- Multiple gestation, polyhydramnios, macrosomic fetus, wrong dates and fibroid or ovarian cyst.
- Cleavage 0-72 hours -> dichorionic diamniotic; 4-8 days -> monochorionic diamniotic; 9-12 days -> monochorionic monoamniotic.

Placental/fetal abnormalities and complications

- Conjoined twins, twin-twin transfusion, fetal malformations, cord abnormalities, retained-dead-fetus syndrome and the source phrase “fetal growth restoration.”
- Maternal: anemia, hydramnios, APH (previa/abruption), hypertension/PET, premature/preterm labor, cesarean delivery, uterine atony and PPH.
- Fetal: malpresentation, PROM, prematurity, cord prolapse, IUGR and increased perinatal morbidity/mortality.

Presentation, intrapartum prerequisites and second twin

- Order at term: vertex-vertex, vertex-breech, breech-vertex, breech-breech.
- Delivery room capable of immediate CS; large-bore IV; simultaneous continuous FHR monitoring; anesthesiologist; two pediatricians; ultrasound for presentation.
- Second-twin risks: cord prolapse, placental abruption and malpresentation. Main causes of perinatal morbidity/mortality: prematurity, RDS, birth asphyxia, birth trauma and cerebral hemorrhage.

Definition, zygosity, causes and diagnosis

- Multiple pregnancy is development of more than one fetus; increased assisted reproduction is emphasized. About one-third are monozygotic/identical and two-thirds dizygotic. Dizygotic twins are always dichorionic-diamniotic; monozygotic membrane pattern depends on division timing; division after day 12 may produce conjoined twins.
- Causes/risk factors: race (more common in Black women), increasing age/parity, tall/high-BMI women as annotated, family history, ovulation induction/ART and conception after stopping contraception.
- Symptoms/signs: exaggerated nausea/vomiting/fatigue, increased fetal activity and uterus larger than dates. Labs may show high beta-hCG and maternal AFP. Ultrasound can diagnose by about 6 weeks; lambda sign/thick septum suggests dichorionicity, T-sign/thin membrane monochorionicity.

Additional complications and management

- Maternal additions: hyperemesis, iron/folate-deficiency anemia, gestational diabetes, cervical incompetence and increased abortion. Fetal additions: small-for-gestational-age, congenital anomalies and TTTS in monochorionic pregnancy.
- Monochorionic-specific problems listed: interplacental vascular anastomoses, TTTS, twin reversed arterial perfusion (TRAP), cord abnormalities and retained-fetus syndrome.
- Antepartum: from 16-22 weeks, review every 2 weeks for cervical-length ultrasound; increase iron/folate/vitamins; later prevent prematurity, monitor uterine activity/hospitalize as required and screen closely for PET.
- Intrapartum: vaginal birth if both cephalic; do not give oxytocin until the second twin is delivered. CS indications shown include >2 fetuses, first-twin malpresentation, CPD/conjoined twins and relative indication for MCMA at 34-36 weeks because of cord risk. Average gestations in the note: twins 35 weeks, triplets 32-33 and quadruplets 28-29. Postpartum use active third-stage management and watch for atony/PPH.

6. Polyhydramnios

Station snapshots

- **Poly 1:** 24-year-old primigravida at 32 weeks, singleton pregnancy, abdominal distension and shortness of breath, fundal height 37 cm.
- **Poly 2:** Mrs. Kadeer, 26, first pregnancy, weight 95 kg, height 1.62 m; fundal size about 38 weeks. Ultrasound: cephalic, longitudinal lie, fundal placenta, AFI 45, measurements corresponding to 34 weeks.
- **Poly 3:** Mrs. Abeer, 26, first pregnancy, completed 34 weeks, weight 95 kg, height 1.62 m; fundal size about 38 weeks, cephalic longitudinal lie.

Recognition and differential

- Uterus or fundal height greater than dates.
- Abdominal distension and dyspnea may be present.
- Differential: uterine fibroids, twin pregnancy, polyhydramnios, and wrong dates.
- Next step in the large-for-dates station: detailed ultrasound.

Causes

- Idiopathic.
- Maternal diabetes.
- Intestinal obstruction.
- Impaired fetal swallowing, with anencephaly as the example.
- Fetal malformations and genetic anomalies.
- Infection: parvovirus B19, rubella, and cytomegalovirus.
- Fetal anemia.

Symptoms and physical findings

- Abdominal distension and shortness of breath.
- Distended abdomen, shiny skin, superficial dilated veins, and transmitted thrill.
- Abnormal lie and presentation.
- Fetal parts difficult to feel.
- Fundal height greater than dates.

Investigations

- Detailed ultrasound or anomaly scan to confirm the condition and assess fetal abnormalities.
- GTT.
- CBC.
- Blood group.
- Viral screening when infection is suspected.
- Source ultrasound details: AFI 45, fundal placenta, cephalic presentation, longitudinal lie, and 34-week fetal measurements.

Obstetric complications

- Maternal dyspnea.
- Preterm labor.
- PROM.
- Placental abruption.
- Abnormal fetal presentation.
- Cord prolapse.
- Postpartum hemorrhage.

Management options

- Expectant management if not severe.
- Amnioreduction.
- Indomethacin.
- Dexamethasone for lung maturity.
- Consider earlier delivery.

Exam trap

- The checklists provide AFI 45 but no general AFI cutoff; do not add an external threshold.

7. Diabetes in Pregnancy

Source set: 3- DM .pdf

STATION SPINE: Optimize control before conception -> screen end-organ disease and medication safety -> explain maternal/fetal risk -> specialist antenatal surveillance -> maintain intrapartum glucose and plan delivery/newborn care.

Preconception counseling and targets

- Explain that tight control reduces fetal abnormality; monitor blood glucose and HbA1c and delay conception until control is optimal. Source target HbA1c <6.5%; if HbA1c is 10, use effective contraception until controlled.
- Folic acid 5 mg before conception and through the first 12 weeks; dietitian plus combined obstetric/diabetes team. Review medication and stop source-listed ACE inhibitors, ARBs and statins.
- Assess hypertension, nephropathy, neuropathy and retinopathy.

Baseline assessment

- HbA1c, fasting/postprandial profile, KFT, CBC, urine routine, blood group, thyroid screen, ophthalmoscopy and 24-hour urine collection.
- Consult ophthalmology, nephrology and cardiology where indicated.

Maternal and fetal complications

- Maternal: miscarriage/recurrent abortion, PET/superimposed PET/gestational hypertension, UTI/Candida and other infection, worsening retinopathy/nephropathy, preterm labor, obstructed labor, operative delivery and maternal trauma.
- Fetal/neonatal: cardiac, skeletal and neural-tube defects; macrosomia, polyhydramnios, prematurity/RDS, IUFD/stillbirth and perinatal/neonatal mortality, shoulder dystocia/birth trauma, neonatal hypoglycemia and jaundice.

Pregnancy, labor and delivery

- Specialist obstetric unit, tighter glucose control, more frequent visits, detailed ultrasound and fetal surveillance.
- Timing/mode depends on fetal assessment; vaginal delivery is possible. Maintain optimal glucose in labor and anticipate a large baby and shoulder dystocia; arrange special newborn care.
- One source specifies planned delivery at 38-39 weeks to reduce stillbirth and diabetes-related fetal complications.

Definitions, diabetogenic hormones and GDM risk factors

- Pregestational diabetes is type 1 or 2 present before pregnancy. The source defines gestational diabetes as glucose intolerance beginning after 20 weeks.
- Diabetogenic hormones listed: placental hPL (second half), placental insulinase, cortisol, progesterone and prolactin.
- Risk factors: obesity, family history of GDM/DM, PCOS, recurrent infections, hypertension/PET, previous GDM/macrosomia/polyhydramnios, current polyhydramnios/macrosomia and prior unexplained fetal/neonatal death, congenital anomaly or IUGR.

GDM screening and diagnostic values in the source

- Fasting glucose at booking: >126 mg/dL is labeled pre-existing diabetes; 92-126 mg/dL GDM; <92 mg/dL leads to 75-g OGTT at 24-28 weeks.
- All pregnancies are screened at 24-28 weeks. Initial source test: 50-g 1-hour OGCT with target <135 mg/dL. Confirmation: 100-g 3-hour OGTT, with the slide showing fasting 95, 1 h 180, 2 h 155 and 3 h 140 mg/dL; another annotation abbreviates >140 mg/dL.
- HbA1c note: normal 4-5.7%, prediabetic 5.7-6.4%, diabetic >6.4%.

Additional pathophysiology, complications and GDM management

- Maternal glucose crosses by facilitated diffusion, causing fetal hyperglycemia and hyperinsulinemia. Congenital anomalies are emphasized for pregestational disease; cardiac defects and caudal regression are highlighted.
- Additional maternal complications: recurrent hypo-/hyperglycemia admissions, later type 2 diabetes/metabolic syndrome/obesity/CVD and acceleration of vascular/end-organ disease. Neonatal additions: hypocalcemia and hypomagnesemia; later child risk of DM/HTN/CVD is stated.
- Management notes: optimize glucose for 3 months before conception, diet/exercise, insulin (metformin annotated for 2nd/3rd trimester), ophthalmology/cardiac/endocrine/nephrology review. Well-controlled GDM without complications may await spontaneous labor; intervene earlier if complications or control fails; consider CS when estimated weight >4,500 g, otherwise vaginal birth if no CS indication.

Fetal cardiac-defect list added from the CVS teaching pages

- The cardiovascular source states congenital heart defects in 3-9% of babies with diabetes in pregnancy and lists transposition of the great arteries, VSD, truncus arteriosus, tricuspid atresia and PDA. Its annotation links the malformation risk to poor control during organogenesis at 3-8 weeks rather than later-onset gestational diabetes.

8. Cardiovascular Disease and Physiological Cardiovascular Changes in Pregnancy

Source set: 2 - CVS .pdf

STATION SPINE: Recognize severe maternal cardiac disease -> reproduce the source counseling decision -> assess pregnancy-related hemodynamic changes -> identify poorly tolerated lesions and teratogenic/cardiac-risk medication notes.

High-risk congenital heart disease station

- 37-year-old G3P2 at 18 weeks attends for fetal echocardiography because of a family history of congenital heart disease. Routine examination identifies uncorrected Tetralogy of Fallot with several episodes of supraventricular tachycardia, maternal oxygen saturation 72% and a ventricular ejection fraction of 20%.
- The student page labels the 20% value as right-ventricular ejection fraction, whereas the examiner answer refers to left ejection fraction <50%. Both source versions are retained.
- Checklist counseling: terminate the pregnancy; source-stated maternal mortality risk 25-50%; decision/care in combination between the high-risk obstetric team and cardiology team.

Other cyanotic congenital heart disorders named by the examiner

- Complete transposition of the great arteries, Ebstein anomaly, truncus arteriosus and tricuspid valve atresia.

Cardiac output and structural changes during pregnancy

- Changes begin early in gestation; the handwritten note attributes the early vasodilation to progesterone rather than fetal size.
- Peripheral vasodilation causes a fall in systemic vascular resistance; the notes also state a fall in pulmonary vascular resistance and afterload.
- Cardiac output increases about 40% during pregnancy and is already about 20% higher by 8 weeks. Both stroke volume and heart rate increase; maternal heart rate rises by about 10 beats/minute.
- Maximum cardiac output is at about 20-28 weeks, falls minimally at term, and returns to normal by about 2 weeks after delivery.
- The heart is physiologically dilated and myocardial contractility increases. Plasma volume and red-cell mass both rise, but plasma rises more, producing dilutional anemia in the annotation.
- Labor causes a further cardiac-output rise: about 15% in first stage and 50% in second stage.

Blood-pressure pattern and measurement

- Blood pressure is described as directly proportional to systemic vascular resistance and cardiac output. Vasodilation is the primary vascular change.
- BP falls from the beginning of pregnancy, reaches its lowest level around 22-24 weeks and returns toward the prepregnancy level at term.
- Use Korotkoff phase V/disappearance rather than phase IV/muffling for diastolic BP, and measure sitting or at about a 30-degree tilt. The annotation warns that supine measurement may cause supine hypotension from vena-caval compression and reduced venous return.
- BP may rise transiently during labor because uterine contractions autotransfuse blood into the circulation. It falls immediately after delivery and then rises to a peak around postpartum days 3-6.
- Source annotation: a BP of 90/50 may be normal around 20-24 weeks, whereas 125/85 at 20 weeks may be considered high relative to the pregnancy nadir. Another annotation says BP medication may sometimes be stopped shortly after labor because BP falls physiologically; this is retained as source wording, not a universal prescription.

Normal cardiovascular findings and lesion tolerance

- S3 and an early ejection systolic murmur may be normal in pregnancy. The S3 slide describes S3 as a marker of volume loading/increased left-atrial pressure but a normal finding in children and pregnant women.
- Rise in blood volume increases preload. Aortic/mitral regurgitation is labeled well tolerated because afterload falls; aortic/mitral stenosis is labeled poorly tolerated with the increased flow/volume load.

Medication and teratogen notes included in the CVS source

- NSAIDs are generally avoided in pregnancy and may close the ductus arteriosus, especially in the late second or third trimester. Low-dose aspirin 81 mg is listed for prevention of preeclampsia.
- Antacids are generally safe. Avoid sodium bicarbonate because of fluid retention/alkalosis and avoid magnesium antacids because they inhibit uterine contractions. If antacids fail, the slide lists sucralfate, then H2-receptor antagonists or proton-pump inhibitors.

- Lithium is labeled a pregnancy-class-D mood stabilizer whose teratogenic effects primarily involve the heart; Ebstein anomaly is the most common listed defect, with apical displacement of the tricuspid valve in the annotation.
- Alcohol-associated heart defects listed: atrial septal defect, ventricular septal defect and Tetralogy of Fallot.

Diabetes-related fetal cardiac defects shown in the source

- Congenital heart defects are stated to affect 3-9% of babies in diabetes in pregnancy. Listed defects: transposition of the great arteries, ventricular septal defects, truncus arteriosus, tricuspid atresia and patent ductus arteriosus.
- The handwritten note emphasizes that malformation risk relates to pregestational/poorly controlled diabetes during organ formation at about 3-8 weeks, whereas gestational diabetes usually develops later.

Practical skills signposted by the source

- The source separately lists obstetric examination, clinical pelvimetry, engagement diameters, speculum examination, IUCD insertion and Pap smear as practical skills. Their detailed content remains in the antenatal/labor, contraception and gynecology-examination chapters.

Source-specific counseling: the termination recommendation and mortality estimate are reproduced from the uploaded examiner sheet and are not replaced with outside guideline advice.

9. Hypertensive Disorders of Pregnancy and Preeclampsia

Source set: 4 - PET .pdf

STATION SPINE: Diagnose after 20 weeks -> recognize organ involvement and severe features -> investigate maternal/fetal status -> stabilize BP and prevent seizures -> deliver and continue postpartum surveillance.

Placental basis, diagnostic criteria and epidemiology

- Normal trophoblast invasion of spiral arteries begins in the first 12 weeks and is normally complete by 20 weeks; maternal placental flow rises from about 50 mL/min in first trimester to 500-750 mL/min at term.
- Mild/source criteria: BP >=140/90 on two occasions >=4 hours apart, >=300 mg protein/24 h, de novo after 20 weeks in a previously normotensive woman and complete resolution by 6 postpartum weeks.
- Severe source threshold: 160/110 with proteinuria >5 g, or symptoms/laboratory organ involvement. More common in primigravidas; recurrence about 20%, higher after very early severe disease; first-degree relatives have a 3-4-fold higher incidence.

Clinical features and severe organ involvement

- Many are asymptomatic. Classic symptoms: frontal headache, visual disturbance and epigastric/RUQ pain; epigastric tenderness suggests liver involvement.
- Rapid face/hand/general edema, oliguria/glomerular endotheliosis, dyspnea/pulmonary edema, retinal hemorrhage/exudate/papilledema, hyperreflexia/clonus, thrombocytopenia and HELLP.
- Severe criteria in the station: creatinine >1.1 mg/dL or double baseline, pulmonary edema, RUQ/epigastric pain, AST/ALT >=2 x normal and platelets <100,000/microliter.

Investigations and management

- CBC/platelets, urinalysis/protein, fibrinogen/PT/PTT, KFT/uric acid, LFT and ultrasound/fetal surveillance.
- Admit, control BP, give magnesium sulfate, monitor fetus and plan timing/mode of delivery; continue postpartum magnesium/medication as listed.
- Emergency agents listed: hydralazine, labetalol and nifedipine. Monitor MgSO4 with reflexes, respiratory rate, urine output and serum level if toxicity suspected.

Magnesium levels and complications

- Therapeutic 4.8-9.6 mg/dL; loss of patellar reflex 8-12; warmth/flushing 9-12; somnolence/slurred speech 10-12; paralysis/respiratory difficulty 15-17; cardiac arrest 30-35.
- Complications: placental insufficiency with IUGR/oligohydramnios/hypoxia, abruption, eclampsia, intracranial hemorrhage/stroke, heart or renal failure/dialysis, pulmonary edema/respiratory failure, HELLP/liver failure, DIC, maternal death and fetal death.

Prevention and delivery statements in the source

- Low-dose aspirin 75 mg; calcium only for women with low dietary intake; vitamins C/E do not reduce risk.
- Source note states cesarean for <34 weeks and contraindicates ergometrine; these are retained as source-specific statements rather than generalized externally.

Risk factors, screening and medication details

- Risk factors listed: previous PET, first-degree family history, first pregnancy, multiple pregnancy, >=10 years since last birth, age >=40, BMI >=35, pre-existing hypertension/renal disease/diabetes, antiphospholipid antibodies, booking diastolic BP >=80 and repeated/quantified booking proteinuria.
- Ultrasound assesses growth/oligohydramnios; Doppler placental perfusion; fundal-height examination growth. CBC note describes hemoconcentration/high RBC/Hct, hemolysis/low Hb and thrombocytopenia.
- Methyldopa is centrally acting, orally administered and slow (>24 h), with sedation/depression. Labetalol has alpha- and beta-blocking effects and can be oral/IV. Nifedipine has rapid onset but may cause severe headache. IV hydralazine/labetalol are listed for acute control.
- Lasix is marked contraindicated because of hemoconcentration/thrombosis and ergometrine because of vasoconstriction.

Delivery timing, eclampsia and magnesium toxicity

- Delivery is the definitive cure; vaginal birth unless another CS indication is stated. Mild/no fetal compromise: <36 weeks expectant, >37 deliver. Severe: >34 weeks deliver after stabilization; <34 weeks stabilize/monitor, give corticosteroid and delay if possible to 34 weeks. Another source line says “<34 weeks, C-section”; both are source-specific statements.
- Eclampsia: generalized tonic-clonic seizures in a patient with preeclampsia, potentially leading to coma/death and complicated by DIC/respiratory failure. Magnesium sulfate is the anticonvulsant of choice; delivery is definitive. It is contraindicated in myasthenia gravis in the slide.
- Monitor magnesium clinically every 1-2 hours with reflexes, respiration, ECG/cardiac conduction and urine output; serum level especially with renal insufficiency. Calcium gluconate is the antidote.

Additional prevention note from the CVS source

- Low-dose aspirin 81 mg is listed for prevention of preeclampsia.

10. Intrauterine Growth Restriction

Source set: 13 IUGR.pdf

STATION SPINE: Confirm dating -> identify maternal/placental/fetal risks -> measure mother and uterus -> assess biometry, fluid, BPP and Doppler -> CTG -> steroids and delivery if indicated.

History and examination

- Correct dating; hypertension/diabetes; smoking, alcohol or drugs; previous IUGR; beta-blockers/anticonvulsants; personal/family thrombosis; infection in pregnancy; second-trimester loss.
- BP, proteinuria, weight/BMI/weight gain, fundal height and presentation.

Investigations and why

- Ultrasound for fetal biometry, congenital anomalies, liquor/AFI, biophysical profile and Doppler.
- Doppler vessels named: umbilical artery, middle cerebral artery and ductus venosus. Add CTG/NST.
- Thrombophilia screen for recurrent IUGR/other adverse outcomes; karyotype only for symmetrical IUGR or associated abnormality.

Delivery

- Administer antenatal steroid and consider delivery before term if indicated. The checklist does not supply a complete timing/mode algorithm; no external thresholds are added.

SGA, IUGR classification and causes

- SGA is estimated fetal weight below the 10th percentile; the source says most/familial cases are healthy (85%). Constitutional SGA has no structural abnormality, normal liquor and normal umbilical Doppler.
- Symmetric IUGR follows an early insult with equally reduced head/body potential; causes highlighted genetic/chromosomal and TORCH. Asymmetric IUGR is common, occurs later from maternal/placental substrate deficiency, with small abdominal circumference and preserved head circumference.
- Fetal causes: trisomies/Turner, TORCH/syphilis/malaria/varicella, gastroschisis/cardiac defects, abnormal liquor and uterine overcrowding. Maternal: age, HTN/PET/DM, CVD/renal/SLE, sickle disease, malnutrition BMI <19, infection, smoking/alcohol and chemo/radiotherapy/antiepileptic/beta-blocker drugs. Placental: mosaicism, abnormal vascular development, previa/abruption, thrombosis/calcification and TTTS.

Complications and expanded surveillance

- Complications listed: meconium aspiration/asphyxia; polycythemia, hyperbilirubinemia, hypocalcemia and hypoglycemia; impaired thermoregulation, fetal distress, neurodevelopmental disability and IUFD.
- Serial ultrasound is the most effective detection method. Umbilical arterial low resistance is reassuring; high resistance, absent or reversed end-diastolic flow indicates poor placentation. Clinical surveillance includes maternal weight, fundal height, serial BPD/head/abdominal circumference/femur length, liquor, NST, BPP and UA/MCA/ductus venosus Doppler.
- Management notes: nutrition/rest/stop smoking, baby aspirin for prevention, weekly NST/biometry/BPP/liquor. Reversed uterine/umbilical flow is listed as a CS indication. Reduced growth velocity, low fluid or abnormal UA after 34 weeks -> delivery; before 34 weeks give steroids. If MCA/ductus venosus decompensation before 34 weeks, deliver without waiting for CTG.

Large-for-gestational-age appendix in the source

- LGA is EFW >90-95th percentile. Risk factors: gestational/overt DM, prolonged/postdate pregnancy, obesity/excess gain, multiparity and male fetus.
- Complications: operative birth, perineal/pelvic-floor injury with incontinence/prolapse, emergency CS and PPH; fetal shoulder dystocia/asphyxia/birth injury; NICU admission, Erb palsy and hypoglycemia. Source suggests elective CS for EFW >4.5 kg in diabetes or >5 kg without diabetes, or early induction.

11. Fetal Wellbeing and Antepartum Surveillance

Source set: 9- Fetal well being .pdf

STATION SPINE: Reassure through history/examination/obstetric assessment/ultrasound -> select NST, CST, BPP or kick counts -> interpret baseline and variability -> recognize causes of fetal tachycardia or bradycardia.

Four-step reassurance and antepartum tests

- Proper history, physical examination, obstetric examination and ultrasound.
- NST/CTG, contraction stress test, biophysical profile and kick-count method.

Checklist causes

Fetal bradycardia	Fetal tachycardia
Captured maternal heartbeat; medication effect; prolonged deceleration; fetal anomaly; conduction anesthesia; head compression.	Maternal fever; intra-amniotic infection; congenital heart disease; fetal anemia/blood loss; medication effect; uterine rupture.

Physiologic CTG data added by the source

- Normal baseline 110-160 bpm; normal variability 5-25 bpm. Persistently minimal/absent variability is emphasized as an important intrapartum sign of compromise.
- Additional tachycardia causes: pyrexia/chorioamnionitis, fetal anemia/hypoxia, beta-agonists and SVT. Additional bradycardia causes: maternal hypothermia/hypotension/hypoglycemia, beta-blockers, AV block/SSA-Ro disease and fetal metabolic acidosis.

Detailed BPP/NST/CST algorithm

- BPP scoring and thresholds follow the CTG module: 8-10 reassuring; 4-6 concerning with delivery if >=36 weeks or repeat 12-24 h/CST if earlier; 0-2 prompt delivery. Modified BPP is NST + AFI.
- For reduced movement: perform NST; if reactive repeat as required. If nonreactive, use vibroacoustic stimulation then BPP. Source shorthand: BPP 0-2 deliver; 4-6 CST; positive CST deliver; negative CST or BPP 8-10 repeat as needed.
- NST indications listed: old maternal age, diabetes/hypertension, postdates, decreased movements and reduced amniotic fluid.

12. Antepartum Hemorrhage

Source set: 5- APH .pdf

STATION SPINE: Characterize bleeding -> stabilize mother -> assess fetus -> distinguish previa, abruption, vasa previa, rupture and local causes -> image and prepare blood -> deliver according to cause/severity.

Differential, incidence and distinguishing pattern

- Placenta previa, placental abruption, vasa previa/fetal-vessel rupture, uterine dehiscence/rupture, cervical tumor/polyp, lower-tract trauma/infection and bleeding disorder.
- Source incidence: abruption 1/100 deliveries, previa 4/1000 and vasa previa 1/2500. Previa is classically painless; abruption is painful/tense with possible concealed blood loss.

Focused history and examination

- Spontaneous or traumatic onset; amount, duration, color and clots; pain/contractions; fetal movement; shock/oliguria. Pulse is emphasized as more sensitive than BP early in shock.
- General/vital signs, amount of bleeding, abdominal tenderness, fundal height, contractions, presentation/lie/engagement and fetal heart; speculum to exclude local cause.

Risk factors

- Previa: age, multiparity/grand multiparity, smoking, multiple gestation, previous previa, cesarean/surgical delivery, curettage/myomectomy/uterine surgery, threatened abortion and abnormal lie/presentation.
- Abruption: hypertension, abdominal trauma, previous abruption, smoking, multiparity, age, twins, IVF, previous abortions and polyhydramnios.
- Vasa previa: velamentous insertion, vessels between bilobed/succenturiate lobes, ART, low-lying placenta/previa, multiple gestation and prior bleeding/surgical delivery.

Vasa previa

- Classic sequence: membrane rupture -> rapid fetal bleeding/painless vaginal bleeding -> fetal bradycardia, late decelerations or sinusoidal pattern -> severe injury/death.
- Apt test: fetal hemoglobin stays pink while adult hemoglobin becomes yellow-brown. Diagnose antenatally with transvaginal ultrasound and color Doppler.
- Source management: consider third-trimester admission, fetal surveillance, corticosteroids and elective CS at 35-36 weeks (another note says 36 weeks).

Immediate management and cause-specific source plans

- ABC, quantify bleeding, two large-bore IVs, CBC/group/crossmatch/coagulation/KFT/LFT, IV fluids/blood, catheter and urine monitoring, maternal/obstetric exam, ultrasound for viability, placenta, gestational age, liquor, scar and chorionicity.
- Previa: >36 weeks CS; <36 stable expectant care, dexamethasone and tocolytic; <36 unstable emergency CS. Abruption: mild <36 expectant, mild >36 deliver, moderate deliver, severe stabilize and deliver (CS if fetus alive in the source).
- Vasa previa -> CS. Uterine rupture -> repair or hysterectomy if bleeding cannot be controlled. Accreta/increta/percreta -> delivery and hysterectomy.
- Ongoing hemorrhage with large clots and estimated 1.5 L -> cesarean while resuscitating.

Definition, blood-loss classes and placenta previa grading

- APH is vaginal bleeding after 24 weeks and before birth; the teaching slide gives incidence 5%. Classes: spotting; minor <50 mL; major 50-1,000 mL without clinical shock; massive >1,000 mL and/or shock.
- Placenta previa is partial/full implantation in lower segment. Grades shown: low-lying edge does not reach internal os; marginal reaches but does not cover; partial covers os asymmetrically when closed; complete centrally covers os.
- Previa presentation: usually mild/painless around 30 weeks, soft uterus, high presenting part/malpresentation and generally normal FHR unless severe bleeding/abruption. Transabdominal US is stated 95% accurate and transvaginal 100%; digital vaginal examination is contraindicated.
- Previa complications: prematurity, PPROM, repeated-bleeding IUGR, malpresentation, fetal abnormality, maternal death/DIC and placenta accreta spectrum (accreta to myometrium, increta invades myometrium, percreta reaches serosa).

Placental abruption definition, types and complications

- Abruption is separation from implantation site before fetal delivery after 24 weeks; total/partial and concealed/revealed forms are listed.
- Additional risks: low BMI, fetal growth restriction/nonvertex presentation, cocaine/amphetamines, intrauterine infection, PPROM, maternal thrombophilia and folate deficiency.
- Painful bleeding, uterine/back pain and tenderness, high-frequency contractions, reduced movement/fetal distress, maternal hypotension/tachycardia. Diagnosis is clinical; ultrasound mainly confirms viability/growth/liquor/Doppler and excludes previa, and the note says only about 2% are diagnosed by scan.
- Maternal complications: shock, anemia, infection, renal tubular necrosis, prolonged stay, psychological sequelae and Sheehan syndrome. Fetal: hypoxia, SGA/IUGR, prematurity and death.

13. OBGYN Trauma and Road Traffic Accident

Source set: 6-RTA .pdf

STATION SPINE: Maternal ABC first -> mechanism and obstetric red flags -> full trauma and abdominal examination -> fetal assessment/bloods -> recognize concealed abruption -> choose delivery route and anticipate DIC/PPH.

Immediate actions and history

- ABC, call for help and insert two large IV lines.
- Loss of consciousness; mechanism/nature/time of accident; seat belt and front/back seat; direct abdominal trauma; pain at any site; vaginal bleeding and fetal movements.

Examination and investigations

- BP, pulse and respiratory rate; general head/neck, chest, heart and limb examination; abdomen for tension, localized tenderness and contractions.
- Ultrasound for fetal heart, AFI and signs of abruption; NST; crossmatch, blood group, CBC and coagulation profile.

Deterioration, delivery and complications

- Dizziness with no visible bleeding and absent fetal heart on ultrasound -> concealed placental abruption.
- Vaginal delivery if stable, cervix dilated and no obstetric CS indication. CS if unstable, delay expected or another obstetric indication exists.
- Complications: DIC and PPH with possible hysterectomy.

14. Maternal Compromise and Obstetric Collapse

Source set: 21 Maternal compremise.pdf

STATION SPINE: Call for help -> left lateral tilt -> airway/breathing/circulation and defibrillation/drugs -> treat cause -> resuscitative cesarean within 5 minutes if no response.

Definition and causes

- Maternal collapse is an acute cardiorespiratory and/or CNS event with reduced/absent consciousness, possible arrest/death, occurring during pregnancy or up to 6 weeks postpartum.
- Causes listed: hemorrhage/hypovolemia, pulmonary embolism, amniotic-fluid embolism, eclampsia, cardiac disease, intracranial hemorrhage, anaphylaxis and drug toxicity.

Immediate management

- Call for help and apply left lateral tilt.
- Airway/intubation; oxygen and bag-mask ventilation; chest compressions and two large-bore cannulae; send bloods and begin IV fluids.
- Defibrillate if indicated; give drugs such as adrenaline; treat the underlying cause.

Perimortem cesarean

- If CPR is unsuccessful, achieve cesarean section within 5 minutes. The source also calls the procedure resuscitative hysterotomy.

15. Premature Rupture of Membranes

Source set: 16- PROM .pdf

STATION SPINE: Leak history -> sterile speculum and confirmation tests -> exclude urine/discharge -> monitor infection and fetus -> steroids/antibiotics and expectant care -> deliver for infection, labor, compromise, abruption or source timing.

History, examination and confirmation

- Duration, gush/intermittent leakage, amount, clear/watery character, color and odor; morning/standing/cough relation; fever/hotness, chills/rigors, abdominal/labor pain, bleeding and fetal movement.
- Vitals for fever/tachycardia; abdominal tenderness, fundal height less than dates, lie/presentation and fetal heart.
- Aseptic speculum for pooling/fluid through cervix, cough sign, bleeding, stress incontinence and infection. Confirmation: nitrazine, ferning, AmniSure and ultrasound with reduced fluid.

Investigations and surveillance

- CBC/WBC with differential, CRP, urinalysis, swabs where listed, ultrasound/BPP, NST/fetal heart.
- Daily NST, vitals, bleeding, pain and discharge characteristics; weekly or twice-weekly WBC and CRP in the 32-week station.

Differential, fetal complications and treatment

- Urinary/stress incontinence and vaginal discharge/infection.
- Prolonged PROM fetal complications: pulmonary hypoplasia/neonatal death, orthopedic abnormalities and prematurity complications including RDS and intraventricular hemorrhage.
- Hospitalize; dexamethasone/steroid for lung maturity, erythromycin/antibiotics, tocolytic if contracting; ongoing maternal/fetal monitoring.

Delivery

- Deliver for chorioamnionitis, established labor, fetal compromise, moderate-severe abruption or major fetal malformation.
- Stable timing in the sources: 35-36 weeks; another source gives completion of 34-36 weeks. Both are retained.

Definitions, risk factors and full complication list

- PROM: membranes rupture before labor. Preterm PROM: before contractions and before 37 weeks. Prolonged ROM: >18 hours before contractions in term or preterm pregnancy.
- Risk factors: previous PROM (recurrence 20% in the note), uterine overdistension (twins/polyhydramnios/macrosomia), abnormal membrane physiology, cervical incompetence, APH, UTI/infection, smoking/nutritional deficiency (zinc/vitamin C) and trauma/intercourse/iatrogenic causes.
- Complications: oligohydramnios, chorioamnionitis with preterm birth/fetal distress/stillbirth/sepsis, abruption, malpresentation, cord prolapse/injury, pulmonary hypertension/hypoplasia/ARDS/IVH, orthopedic abnormalities, postpartum infection/endometritis.

Expanded diagnostic tests and chorioamnionitis signs

- Amniotic fluid is alkaline: nitrazine turns blue while normal vaginal secretions are acidic/red. Other source tests: fern pattern, IGF-1, placental alpha-microglobulin-1/PAMG-1 (AmniSure), tampon/dye amniocentesis, low AFI, endocervical swab and urine testing.
- Chorioamnionitis signs listed: fever, maternal leukocytosis/tachycardia, fetal tachycardia >160, uterine tenderness, purulent/malodorous fluid and rarely bacteremia.

Gestational-age plan and tocolysis

- Unstable/infection/abruption/cord prolapse/nonreassuring FHR -> prompt delivery and cultures/empiric ampicillin + gentamicin in the teaching slide.
- Stable >=37 weeks: induction or brief expectant management up to 12-24 h. At 34 weeks: expectant management or induction; give a single steroid course if delivery >24 h and <7 days away and no infection. At 24-33 weeks: expectant care, ampicillin plus erythromycin, steroid, short tocolysis up to 48 h and magnesium for fetal neuroprotection if delivery anticipated. <23-24 weeks: poor outcome, expectant approach as described.
- Tocolysis aims to permit lung maturation, reduce prematurity complications and permit NICU transfer. Contraindications: chorioamnionitis, advanced labor >4 cm, nonreassuring fetus, abruption or cord-prolapse risk. Agents listed: beta-agonists (terbutaline/ritodrine, noted as no longer used), magnesium sulfate, indomethacin with premature ductal-closure risk, atosiban and nifedipine.

16. Induction of Labor

STATION SPINE: Reason -> exclude contraindication -> lie/presentation/size -> Bishop score -> fetal well-being -> consent -> method -> monitor labor.

Station snapshots

- **Induction 1:** Mrs. Moza, 37, married 5 years, first pregnancy at 40 weeks, worried about induction and mode of delivery.
- **Induction 2:** 32-year-old G1P0 at 41 weeks with decreased fetal movement, admitted for induction.
- **Induction 3:** student page says a 36-year-old nurse married 7 years; examiner page says 40 years old and married 8 years; first pregnancy at 41 weeks, discussing delivery.

Indications

- Pregnancy beyond 41 weeks.
- Prelabor spontaneous rupture of membranes.
- Maternal diabetes or hypertension.
- Recurrent antepartum hemorrhage.
- Placental abruption.
- Intrauterine growth restriction.
- Oligohydramnios.

Patient-specific reasons in the older primigravida station

- Pregnancy at 41 weeks.
- Old primigravida.
- First pregnancy after 7 or 8 years of marriage, according to the conflicting source pages.
- Placental calcification.
- Avoid post-term syndrome.
- Possibility of reduced liquor.

Mandatory assessment before induction

- Confirm a medical indication.
- Confirm no contraindication to induction or vaginal delivery.
- Confirm fetal lie.
- Confirm fetal presentation.
- In the patient-specific station: longitudinal lie, cephalic occipito-anterior presentation, and average-sized baby.
- Assess cervical condition using the Bishop score.
- Assess fetal well-being with biophysical profile and cardiotocography where listed.
- Counsel and obtain consent.

Methods

- Mechanical cervical dilatation.
- Foley catheter placed in the cervix.
- Membrane sweeping.
- Amniotomy.
- Prostaglandin E2 or prostaglandin suppositories.
- Misoprostol/Cytotec, described as a synthetic prostaglandin E1 analogue.
- Oxytocin infusion.
- Breast and nipple stimulation in Induction 3.

Source-specific 41-week delivery plan

- Wait one more week if there is no labor pain.
- Induce labor the following week.
- Explain that repeat induction may be required.
- If induction fails, proceed to surgical delivery.

First-stage management

- Lateral recumbent position.
- Fluids.
- Maternal monitoring.
- Analgesia.

- Fetal monitoring.
- Uterine-activity monitoring.
- Vaginal examination to assess progress.
- Amniotomy.

Second-stage management

- Comfortable position for effective bearing down.
- Continuous fetal-heart monitoring.
- Record progress every 30 minutes.
- Lithotomy when delivery is imminent.
- Delayed cord clamping for 1-2 minutes.

Signs of placental separation

- Fresh vaginal show of blood.
- Umbilical cord lengthens.
- Fundus rises.
- Uterus becomes firm and globular.

Exam trap

- Preserve the source's unusual 41-week plan rather than silently replacing it with an external protocol.

17. Breech Presentation

Source set: 14- breech .pdf

STATION SPINE: Classify breech -> identify cause and associated conditions -> offer ECV, planned CS or selected vaginal birth -> confirm strict prerequisites -> anticipate head, cord, hypoxia and trauma risks.

Types and frequencies

- Frank/extended breech: buttocks first, hips flexed and knees extended; source frequency 50-75%.
- Footling: one/both feet first; about 20%. Complete/flexed: hips and knees flexed; about 5-10%.

Causes, signs and associated conditions

- Prematurity, fetal anomaly, uterine anomaly, multiple gestation, polyhydramnios, oligohydramnios, abnormal placental location and pelvic tumors.
- Head palpable outside pelvis, fetal heart heard high, and buttock/foot/feet on vaginal examination.
- Associated problems: abruption, PROM, intracranial hemorrhage, IUGR, previa, cord prolapse, aftercoming-head entrapment and high perinatal mortality.

Management and vaginal-birth prerequisites

- Options: elective CS, external cephalic version and vaginal breech delivery.
- Prerequisites: no contraindication to vaginal birth; singleton frank breech; estimated weight 2,500-3,800 g; continuous EFM; normal cervical progress; skilled obstetrician; delivery in/near CS room with anesthesiologist and neonatologist.

Vaginal breech risks

- Head entrapment, cord prolapse, fetal hypoxia/asphyxia, fractures/visceral injury and maternal tears.

Definition, diagnosis and detailed vaginal-birth criteria

- Breech is presentation of fetal buttocks/lower extremities into maternal pelvis and is the commonest malpresentation. Before 28 weeks about 25% are breech in the note; prematurity is the major predisposing factor.
- Leopold: firm head in fundus and softer/smaller breech in lower segment. Vaginal exam may identify buttocks, anus, sacrum and ischial tuberosities; confirm by ultrasound.
- Additional etiologies: hydrocephaly/structural anomaly, bicornuate uterus, contracted pelvis and prior uterine surgery/placenta previa.
- Selected vaginal criteria in the teaching slide: >=36 weeks, frank or complete (not footling), weight 2.5-3.8 kg, flexed head, no CS indication, experienced obstetrician with anesthetist. Use hands-off approach and allow maternal effort/uterine contractions; avoid pulling from below.
- Additional complications: increased CTG abnormalities and brachial-plexus/Erb injury from forceful traction.

18. Labor and Intrapartum Maternal Monitoring

Source set: 17- Labor .pdf

STATION SPINE: Confirm true labor -> assess passenger/passage/powers -> monitor mother, fetus, contractions and progress -> manage three stages -> respond to delay, meconium, bleeding or fetal distress.

Definitions, pelvis and fetal relationships

- Labor: regular, rhythmic painful contractions causing cervical ripening, effacement and dilation and leading to delivery.
- Best pelvis: gynecoid. CPD causes listed: macrosomia and contracted pelvis. Ideal presentation vertex, ideal position occipito-anterior. Lies: longitudinal, transverse and oblique.
- Normal birth in the source: spontaneous onset, low risk throughout, vertex delivery at 37-42 weeks, mother and infant well afterward.

Stages and progress

- Latent first stage: irregular contractions, effacement/dilation to 4 cm; average 8 h nullipara and 5 h multipara; prolonged >20 h nullipara or >12 h multipara.
- Active first stage: regular contractions and progressive dilation beyond 4 cm; source describes 1 cm/h or more and abnormal if <1 cm in 2 h.
- Third stage normally 5-10 min; prolonged after 30 min. Placental separation: cord lengthening, blood gush, fundal rise and firm globular uterus.

Admission and first-stage management

- Complete history/examination, IV line, CBC/Hb, blood group, urinalysis and crossmatch for unbooked labor.
- Monitor maternal vitals, hydration/fluids, analgesia, fetal heart, contractions/uterine activity and progress; vaginal exam every 2-4 h unless urgent; consider amniotomy and augmentation.
- Urgent vaginal exam for fetal distress, ROM, vaginal bleeding, before analgesia and when pushing begins.

Passenger and mechanics

- Variables: size, lie, presentation, attitude, position and station. Cardinal movements: engagement, descent, flexion, internal rotation, extension, external rotation and expulsion.
- Bishop score and cervical-ripening factors named: prostaglandins, estrogens, progesterone/antiprogestogens, relaxin, inflammatory mediators, nitric oxide and apoptosis.

Second/third stage and active management

- Comfortable bearing-down position, continuous FHR, record progress every 30 min, lithotomy when delivery imminent and delayed cord clamping 1-3 minutes.
- Active third stage in notes: oxytocin 10 IU IM at anterior shoulder/immediately after birth, early cord clamp/cut and controlled traction; modified approach favors delayed clamping while maintaining uterotonic and controlled traction.

Meconium/GBS station

- Post-term primigravida with green fluid: diagnose ROM with meconium. Risks: meconium aspiration and neonatal sepsis.
- Admit/augment, continuous CTG and IV antibiotics for GBS-positive status; assess cord prolapse and cervical progress.

Presentation, engagement, station, position, attitude and lie

- Presentation is the fetal part over the pelvic brim. Cephalic landmarks include occiput/vertex; face denominator mentum; breech denominator sacrum; transverse denominator shoulder/olecranon. Slide diameters: biparietal 9.5 cm, breech intertrochanteric 10 cm and shoulder biacromial 11 cm.
- Engagement occurs when the widest diameter of the presenting part enters the inlet. Station is relation to maternal ischial spines.
- Position relates denominator to maternal pelvis; OA is normal/commonest. OP may prolong second stage, cause arrest if rotation/descent fail and increase perineal trauma/episiotomy. Normal attitude is full flexion; lies are longitudinal, transverse and oblique.

Detailed stages and arrest assessment

- First stage source durations: 6-18 h nullipara and 2-10 h multipara (average 10 h). Latent phase to 4 cm is variable/longer in primipara. Active dilation from >4 to 10 cm is quoted as 1 cm/h nullipara and 1.2 cm/h multipara.
- Three Ps: passage (pelvic size/shape), passenger (fetal size/position) and power (uterine contractions; >200 Montevideo units adequate). If dilation is slower/no station change for 2 h, assess uterine dysfunction, malposition and CPD. Caput, extensive molding and overlapping sutures suggest passenger/pelvis problems. Active-phase arrest despite adequate contractions -> CS in the source.
- Second stage is complete dilation to birth: source range 30 min-3 h primigravida and 5-30 min multigravida, with passive and active/maternal-urge phases. Cardinal movement detail: descent, flexion, internal rotation, extension/crowning at about +5, restitution/external rotation and delivery of anterior then posterior shoulder/body.
- Third stage begins after birth; placenta usually 2-10 minutes and should be <30 minutes. After separation signs, controlled cord traction with counterpressure is applied and placenta examined for completeness. Fourth stage lasts 4-6 hours with close vital-sign and bleeding surveillance because serious PPH often occurs then.

19. Operative Vaginal Delivery

Source set: 18 Operative and CS .pdf

STATION SPINE: Confirm indication and prerequisites -> choose forceps/vacuum and classification -> apply by a skilled operator with backup -> abandon after failed pulls/pop-offs -> monitor maternal/neonatal complications and aftercare.

Indications and limitations

- Fetal compromise in second stage, maternal exhaustion, delayed second stage, avoidance of pushing in cerebral aneurysm, proliferative retinopathy, NYHA III/IV cardiac disease or myasthenia, and forceps for aftercoming breech head.
- Vacuum should be avoided below 32 weeks and used cautiously 32-36; do not use for face or breech. Fetal bleeding disorder or fracture predisposition is a relative contraindication.

Prerequisites

- Head <=1/5 palpable abdominally/engaged, vertex, full dilation, ruptured membranes, exact position known, adequate pelvis and no CPD/caput-molding concern.
- Explain and consent; adequate regional/pudendal analgesia where appropriate; empty bladder/remove catheter.
- Skilled operator, correct equipment/lighting, backup CS plan (theatre within <30 min for mid-cavity), anticipate shoulder dystocia/PPH and have neonatal resuscitation staff.

Failure and abandonment

- Failure factors from checklist: wrong cup type, inadequate initial assessment, incorrect cup placement/replacement, wrong traction pathway and poor maternal effort/inadequate Syntocinon.
- Abandon after 2 pop-offs or 3 pulls without progress/imminent delivery, or no progressive descent after 3 contractions with a correctly applied instrument.
- Higher failure rates: BMI >30, estimated weight >4,000 g, occipito-posterior and mid-cavity/1/5 palpable head.

Complications and aftercare

- Maternal: third/fourth-degree tears, vaginal/vulval trauma, fecal/anal sphincter or voiding dysfunction, PPH, cervical laceration, uterine rupture and psychological trauma/tokophobia.
- Fetal: subgaleal/intracranial hemorrhage, skull fracture, facial nerve injury, shoulder dystocia, facial/scalp marks/lacerations, chignon, cephalohematoma, jaundice and retinal hemorrhage.
- Aftercare: paracetamol/diclofenac, antibiotics, thromboprophylaxis when risk factors, bladder care, physiotherapy and psychological support. Source gives ~80% spontaneous vaginal birth in a subsequent pregnancy.

Instrument types, parts and classification

- Forceps categories/examples: classical/direct OA (Wrigley, Simpson/Neville Barnes), rotational/minimal rotation (Kielland) and Piper for the aftercoming breech head. Parts: blades with cephalic and pelvic curves, shanks, lock, shoulders and handles.
- Vacuum/ventouse types shown: Malmstrom metal cup, soft Silastic cup, OP Kiwi and standard Kiwi. Soft cup has higher failure but less neonatal scalp trauma.
- Classification is by station/rotation (outlet, low and mid-cavity in the source diagrams); operative delivery choice depends on head level, position and rotation.

Additional contraindications and risk counseling

- Frequent maternal risks: PPH, vaginal tear/abrasion and anal-sphincter or voiding dysfunction. Serious risks: third/fourth-degree tears and significant vaginal/vulval trauma, higher with forceps.
- Frequent fetal risks: forceps facial marks, chignon/cup mark in nearly all vacuum cases, cephalohematoma, facial/scalp laceration, jaundice/hyperbilirubinemia and retinal hemorrhage; serious subgaleal/intracranial hemorrhage and rare facial palsy.

20. Cesarean Section and Previous-Cesarean Counseling

Source set: 18 Operative and CS .pdf

STATION SPINE: Define indication and incision -> prepare/consent/anesthetize -> anticipate immediate, early and late complications -> review old operative details and current pregnancy -> counsel mode of birth.

Definition, incisions and layers

- CS delivers fetus, placenta and membranes through abdominal and uterine incisions under anesthesia.
- Skin: Pfannenstiel/low transverse (better cosmetic result, less analgesia, stronger wound) versus vertical midline (better access/extension but more dehiscence and later hernia).
- Uterine incisions: lower transverse (commonest), inverted T, low vertical and classical upper vertical. Layers: skin, fat, rectus sheath, rectus muscle, parietal peritoneum, visceral peritoneum and uterine muscle.

Classical CS and indications

- Classical incision is rare; source indications: fibroid occupying lower segment, inaccessible lower segment from dense adhesions, selected previa, transverse lie back-down, conjoined twins/abnormality, constriction ring and cervical cancer.
- General CS indications listed: maternal request, two previous lower-segment CS, one classical CS, prior rupture or cavity-entering myomectomy; labor dystocia, fetal distress/bradycardia/cord prolapse, CPD/macrosomia, multiple pregnancy, malpresentation; previa/major abruption/vasa previa/accreta spectrum, IUGR/placental insufficiency, hypertension/diabetes and selected maternal infection.

Preparation and perioperative points

- Elective CS not before 39 weeks in the source because of neonatal respiratory morbidity. Obtain consent and respect dignity, privacy, views and culture.
- CBC for anemia, antibody screen, group and save/crossmatch. Give prophylactic antibiotic before skin incision. Regional anesthesia is preferred in the source.
- Approximate blood loss 1,000 mL.

Complications

- Immediate: anesthesia complications/death, hemorrhage/transfusion, amniotic-fluid embolism, ileus and injury to bladder, ureter, vessels or bowel.
- Early: secondary hemorrhage, endometritis, atelectasis, UTI, wound infection/dehiscence, hematoma, thrombophlebitis/DVT/PE and breast infection. Source states infectious risk is 5-20-fold higher and endometritis/UTI/wound infection occur in about 8%.
- Late: adhesions/obstruction/infertility, incisional hernia/fistula, placenta previa/accreta spectrum and future uterine rupture.

Repeat-CS and VBAC counseling station

- After four CS: review previous obstetric details, last operative adhesions, placental site, fetal growth/health, lie/presentation; counsel anesthesia, hemorrhage/transfusion, bladder/bowel injury, adherent-placenta hysterectomy, infection and hernia; order CBC, group/Rh and crossmatch 2 units.
- After one CS: ask incision type, indication, extension, infection, gestational age, labor/dilation, presentation/fetal status/outcome and baby weight; current disease, presentation, placenta and estimated weight; perform clinical pelvimetry. Counsel that VBAC carries a small uterine-rupture risk; the source does not provide a final eligibility algorithm.

Timing and additional source labels

- Cesarean is classified as emergency or elective/planned. The source reiterates lower-segment transverse as commonest and classical upper vertical as the rare incision with greatest future rupture implications.

21. Postpartum Hemorrhage

Source set: 7- PPH.pdf

STATION SPINE: Recognize blood loss/shock -> call for help and ABC -> two IV lines and blood -> assess tone, trauma, tissue and coagulation -> uterotonics/transfusion -> surgery if uncontrolled.

Clinical severity table from the source

Blood loss	Systolic BP	Signs	Shock
500-1,000 mL (10-15%)	Normal	Palpitations, dizziness, tachycardia	Compensated
1,000-1,500 mL (10-25%)	Slight decrease	Weakness, sweating, tachycardia	Mild
1,500-2,000 mL (25-35%)	70-80 mmHg	Restlessness, pallor, oliguria	Moderate
2,000-3,000 mL (35-45%)	50-70 mmHg	Collapse, air hunger, anuria	Severe

Predisposing history and immediate checklist

- Parity; twins/polyhydramnios; PET; previous PPH; previous CS; prolonged labor; prolonged third stage; instrumental delivery; augmentation; check whether placenta is complete.
- Call for help, ABC, two large-bore IVs, crossmatch, palpate/massage uterus, vaginal examination, uterotonics, check lacerations, transfuse as needed and escalate to surgery.

Definition, timing, causes and prevention

- Source definition: blood loss >500 mL after vaginal birth or >1,000 mL after CS. Primary/early PPH occurs within 24 hours; secondary/late PPH after 24 hours. Uterine atony is the commonest primary cause; infection is highlighted as the commonest secondary cause.
- Organize primary PPH by the source 4/6 Ts: Tone (atony), Trauma (laceration), Tissue (retained placenta), Thrombin/coagulopathy, Turned-over uterus (inversion) and unexplained hemorrhage.
- Prevention/active third stage: oxytocin after shoulder delivery, uterine massage, controlled cord traction with suprapubic counterpressure after separation signs, and examine placenta/speculum/observe vitals in the first postoperative hour.

Cause-specific recognition and treatment

- Tone/atony: overworked uterus (rapid/prolonged labor or excess oxytocin), overdistension (twins/polyhydramnios/macrosomia), infection or uterine-relaxing drugs (MgSO4, beta-agonists, halothane). Doughy enlarged uterus. Treat massage, oxytocin, ergometrine, carboprost/Hemabate, misoprostol; B-Lynch, Bakri balloon, embolization/ligation or hysterectomy if refractory.
- Trauma: operative/traumatic birth; laceration with contracted uterus. Apply pressure and surgically repair.
- Tissue: accessory lobe/accreta, missing cotyledons with contracted uterus. Manual removal or ultrasound-guided curettage; accreta may require hysterectomy.
- Thrombin/DIC risks: abruption, severe PET, amniotic-fluid embolism, fetal demise/coagulopathy. Generalized oozing/petechiae; CBC/platelets/PT/PTT/INR. Treat cause, ICU and blood products (FFP/cryoprecipitate/platelets).
- Uterine inversion: risk myometrial weakness, previous inversion, fundal/accreta placenta and excessive cord traction/fundal pressure. Beefy bleeding vaginal mass and impalpable uterus. Manual replacement (tocolytic if required), then IV oxytocin.
- Uncontrolled/unexplained hemorrhage: uterine/internal-iliac ligation or embolization if stable; laparotomy/hysterectomy if unstable.

Secondary PPH and complications

- Secondary causes listed: retained products/placental tissue, endometritis, subinvolution, uterine myoma and blood clot.
- Complications: maternal morbidity/mortality, renal failure, chronic anemia and Sheehan syndrome (amenorrhea, failure to lactate, hypothyroidism).
- General work-up/management additions: oxygen as required, Foley to empty bladder, CBC/group/crossmatch/coagulation/KFT/LFT; ergometrine caution with hypertension and carboprost relative contraindication in asthma.

22. Postpartum Care and Ward Round

Source set: 19- postpartum .pdf

STATION SPINE: Handover mother and newborn -> ask maternal recovery symptoms -> examine vitals, uterus and legs -> review feeding, lochia, urine and perineum -> counsel contraception.

Consultant handover

- Booked/unbooked; term/preterm; delivery type (CS/SUD/instrumental); newborn weight, location/fate (N.N or NICV), live status and “ALS”; estimated blood loss and episiotomy/perineal details; maternal group, age, gravidity/parity, delivery time, pre-delivery Hb and general condition.

Morning postpartum review

- General condition, dizziness/fainting, lactation, abdominal pain, lochia, episiotomy/tear pain, passage of urine and lower-limb complaints.
- Vitals, fundal height, uterine contraction and lower-limb examination for DVT.
- Contraception in the checklist: progestogen-only pill if lactating; combined pill if not lactating; IUCD and other methods.

Unexpanded abbreviations retained: SUD, N.N, NICV and ALS are not defined by the source.

23. Puerperal Sepsis and Puerperal Pyrexia

Source set: 20 Puerperal sepis.pdf

STATION SPINE: Recognize postpartum genital-tract infection -> apply the source definition -> identify risk factors and organisms -> culture before antibiotics -> look for pelvic/systemic complications -> begin empirical anaerobic coverage.

Examiner scenario and core checklist

- 32-year-old G4P4, 9 days after forceps delivery for a prolonged second stage, presents with lower-abdominal pain, fever, chills and bad-odor bloody vaginal discharge.
- Most likely diagnosis: puerperal sepsis. Serious examiner-listed complications: septic shock, pelvic thrombophlebitis and pelvic abscess.
- Examiner organism lines: anaerobic organisms (Peptostreptococcus and Peptococcus), mixed infections, and aerobic organisms, mostly Escherichia coli.
- Patient-specific predisposing factors: prolonged rupture of membranes, prolonged labor, frequent vaginal examinations and forceps delivery.
- Before antibiotics: WBC count and differential, blood culture, endocervical/high-vaginal cultures, and urine routine/microscopy/culture. Begin empirical antibiotics as soon as possible and provide anaerobic coverage.

Definition and clinical criteria in the embedded teaching material

- Puerperal sepsis is defined as genital-tract infection occurring at any time from onset of rupture of membranes or labor until the 42nd postpartum day, with two or more of the following.
- Oral temperature 38.5 C/101.3 F or higher on any occasion; abnormal vaginal discharge such as pus; abnormal/foul odor of discharge; and delayed uterine involution, stated as <2 cm/day during the first 8 days.
- An additional box defines primary puerperal sepsis as genital-tract infection after delivery and calls it the commonest cause of puerperal infection.

Expanded causative organisms from the embedded box

- Anaerobic streptococci; group-A hemolytic streptococci; staphylococci causing suppuration/pus; E. coli and non-hemolytic streptococci; and specific organisms such as Clostridium welchii and tetani.

Antepartum and intrapartum predisposing factors

- Antepartum: malnutrition, anemia, preterm labor, early rupture/PROM/PPROM, precipitate delivery, immunocompromise such as AIDS, diabetes, obesity and organisms of normal vaginal flora. Handwritten additions emphasize reduced immunity and early delivery.
- Intrapartum: repeated vaginal examinations, dehydration, ketoacidosis during labor, traumatic vaginal delivery, APH or PPH, retained placental tissue/membranes, prolonged labor, obstructed labor, and cesarean or instrumental delivery. Handwritten additions mention difficult labor and fluid/blood loss during labor.

Local, pelvic and systemic complications/signs

- Pelvic tenderness, pelvic peritonitis, generalized peritonitis, fornix tenderness, endometritis/endomyometritis/endoparametritis, a bulging fluctuant mass in the pouch of Douglas, pelvic abscess, phlebitis/thrombophlebitis, bacteremia, endotoxic or septic shock, and septicemia.

Expanded diagnostic evaluation

- High-vaginal and endocervical swabs for culture in aerobic and anaerobic media plus antibiotic-sensitivity testing.
- Clean-catch midstream urine for urinalysis, culture and sensitivity.
- Blood for total/differential WBC, hemoglobin and platelet count; low platelets may indicate septicemia or DIC. Thick blood film is listed for malaria parasites.
- Blood culture when fever is accompanied by chills and rigor.
- Pelvic ultrasound for retained products, pelvic collection/abscess and ultrasound-guided sampling of pus/fluid for culture; color-flow Doppler is listed to detect venous thrombosis.
- CT or MRI, especially when the diagnosis is uncertain or pelvic-vein thrombosis is suspected; chest X-ray for lung pathology such as collapse/atelectasis after inhalational anesthesia; blood urea/electrolytes for renal failure; laparotomy to explore the abdominal organs when required.

Source boundary: no antibiotic drug names, doses or duration are supplied by the examiner sheet; the guide therefore retains only immediate empirical treatment with anaerobic coverage.

O1. Anemia in Pregnancy - Three Constructed OSCE Stations

Source set: Anemia and Hyperemesis Gravidarum(2).pdf

CONSTRUCTED-STATION SPINE: Pregnancy-specific Hb threshold -> indices and morphology -> confirm cause -> assess consequences -> treat by severity and gestation -> monitor response.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Iron-deficiency anemia at 28 weeks

STUDENTS INFORMATION
A 28-year-old G2P1 at 28 weeks reports fatigue, dizziness, palpitations and exertional dyspnea. Hb is 8.9 g/dL, MCV 72 fL, MCH/MCHC are low and ferritin is 12 micrograms/L. Discuss the diagnosis, assessment and management.

Examiner questions

- Define anemia using pregnancy-specific thresholds and distinguish physiological hemodilution from pathological anemia.
- Interpret the red-cell pattern and confirm iron deficiency.
- Discuss maternal/fetal consequences and the treatment ladder.
- Explain monitoring of response and duration of treatment.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Definition and stage threshold	At 28 weeks, anemia is Hb <10.5 g/dL in the lecture; first-trimester cutoff is 11 g/dL and postpartum cutoff 10 g/dL.	**2**
Physiological comparison	Pregnancy plasma volume expands about 50%, begins in first trimester and plateaus by third; Hb falls by hemodilution while MCV and MCHC remain unchanged. The lecture notes absent/reduced expansion in pre-eclampsia.	**2**
Symptoms and contributors	Fatigue, dizziness, palpitations, irritability, dyspnea and rare pica; assess low intake, impaired absorption, multiple pregnancy/increased demand, infestation/malaria, blood loss and lack of supplements.	**2**
CBC and morphology	Low Hb with reduced MCV, MCH and MCHC; microcytic hypochromic blood film. The lecture calls MCV the first index to become abnormal.	**2**
Iron profile	Ferritin <30 micrograms/L, serum iron <12 micromol/L and saturation <15% as written. Ferritin best assesses stores in absence of inflammation; marrow iron is most reliable but invasive.	**2**
Maternal/fetal effects	Maternal recurrent infection, transfusion need, PPH and postpartum depression; fetal low birth weight, SGA, preterm birth and long-term neurocognitive effects.	**2**
Oral first line	Diet plus oral iron. Morning/empty-stomach dosing with water or vitamin C; meat and vitamin C enhance absorption, while bread/phytate, tea/coffee/chocolate/tannins and calcium inhibit it.	**2**
Tolerance and preparations	GI effects: nausea, epigastric pain and constipation; dose-related. Try alternate-day or lower-iron preparation; avoid slow-release/enteric-coated products. Ferrous fumarate 210 mg or dried sulphate 200 mg each contain 65 mg elemental iron; gluconate 300 mg contains 35 mg.	**2**
Response and duration	Earliest response is reticulocytosis; check Hb after 2-4 weeks. Continue at least 3 months after Hb normalizes and until 6 weeks postpartum.	**2**
Escalation	IV iron for intolerance/nonresponse from second trimester onward and consider after 34 weeks with Hb <100 g/L; example ferric carboxymaltose. Lecture transfusion threshold: Hb <8 g/dL in late pregnancy.	**2**

What you must memorize from this station

- Hb cutoffs: 11 first trimester; 10.5 second/third; 10 postpartum.
- Physiologic anemia lowers Hb but leaves MCV/MCHC unchanged; IDA is microcytic hypochromic.
- Ferritin <30, serum iron <12 and saturation <15% are the lecture confirmation values.
- Management ladder: diet -> oral iron -> IV iron -> transfusion when severe/late.
- Reticulocytes rise first; recheck Hb at 2-4 weeks; continue through 6 weeks postpartum.

EXAM TRAPS: Do not diagnose iron deficiency from fatigue alone. Do not stop iron as soon as Hb normalizes. Keep dietary intake, absorbed iron and physiological requirement as different numbers.

Station 2: Macrocytic anemia and folate counseling

STUDENTS INFORMATION
A 30-year-old woman at 10 weeks has Hb 9.7 g/dL, increased MCV and hypersegmented neutrophils. She has diabetes, BMI 32 and takes an antiepileptic drug. Explain the likely diagnosis, differential, prophylaxis and management principles.

Examiner questions

- Interpret the macrocytic/megaloblastic pattern.
- Explain folate physiology and the timing of neural-tube closure.
- State routine and high-risk folic-acid regimens.
- Compare this with physiological anemia and iron deficiency.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Pattern	Megaloblastic anemia is low Hb with increased MCV; causes listed are folate deficiency and vitamin B12 deficiency.	**2**
Earliest morphology	Hypersegmentation of neutrophils is the lecture's earliest morphological evidence of folate deficiency.	**2**
Frequency	Folate deficiency is the second most common cause of anemia in pregnancy; IDA is first.	**2**
Neural-tube timing	Folic acid is required for neural-tube closure, completed 15-28 days after conception (approximately 4-6 weeks gestation).	**2**
Routine prophylaxis	400 micrograms/day for all women planning pregnancy, starting 12 weeks before pregnancy and through the first trimester.	**2**
High-dose regimen	5 mg/day for high-risk women.	**2**
High-risk groups I	Woman with spina bifida or a previous fetus with neural-tube defect; antiepileptic drugs or sulfasalazine.	**2**
High-risk groups II	Diabetes, BMI >30, hemoglobinopathy, malabsorption such as Crohn disease, or proven folate deficiency.	**2**
Morphologic comparison	Physiological hemodilution: indices unchanged. IDA: low MCV/MCH/MCHC. Megaloblastic: increased MCV with folate/B12 differential.	**2**
Safety reasoning	Prophylaxis must start before conception because closure occurs early; do not wait until the first booking visit.	**2**

What you must memorize from this station

- Macrocytic/megaloblastic pattern = increased MCV; think folate or B12.
- Hypersegmented neutrophils are the earliest lecture clue.
- Routine dose 400 micrograms/day; high-risk dose 5 mg/day.
- High-risk list: prior/personal NTD, antiepileptics/sulfasalazine, diabetes, BMI >30, hemoglobinopathy, malabsorption, proven deficiency.
- Neural tube closes by about 4-6 weeks, explaining preconception therapy.

EXAM TRAPS: The source once calls the pattern “macrocytic hypochromic”; memorize the safe core: increased MCV and folate/B12 differential.

Station 3: Hemoglobinopathy in pregnancy

STUDENTS INFORMATION
A 24-year-old primigravida is found to have HbA2 4.2% on hemoglobin electrophoresis. Her husband is also a carrier. A second part of the station asks how pregnancy is managed in thalassemia and sickle cell disease.

Examiner questions

- Interpret the electrophoresis and inheritance.
- Explain partner testing and genetic counseling.
- Compare thalassemia and sickle cell disease.
- State maternal/fetal complications and pregnancy management.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Inheritance and diagnosis	Hemoglobinopathies are autosomal recessive; carrier versus disease; diagnose with Hb electrophoresis. If both partners are carriers, offer genetic counseling/diagnostic options.	**2**
Normal hemoglobins	HbA >95% = alpha2 beta2; HbA2 = alpha2 delta2 and normally 2-3%; HbF = alpha2 gamma2 and normally 0.8-2%.	**2**
Thalassemia clue	Reduced production of normal hemoglobin; HbA2 >3% is the lecture clue.	**2**
Sickle molecular basis	Valine substitutes for glutamic acid at position 6 of beta chain; Hb aggregates when deoxygenated, causing sickling. HbS/HbC are abnormal and normally absent.	**2**
Sickle crises	Hemolytic and painful crises, usually in the last trimester in the lecture.	**2**
Maternal sickle complications	Anemia, recurrent infection, chronic hyperbilirubinemia, acute chest syndrome, pre-eclampsia, VTE and death.	**2**
Fetal sickle complications	Miscarriage, fetal growth restriction, premature labor and placental abruption.	**2**
Shared management	Multidisciplinary care, serial growth scans from 20 weeks, correct anemia/target Hb “10” as written, folate 5 mg/day, low-dose aspirin and postnatal LMWH.	**2**
Thalassemia-specific	Screen iron overload with liver tests/cardiac echo; iron chelation; desferrioxamine safe from 20 weeks (SC antenatally, IV in labor); cardiac failure is primary cause of death.	**2**
Sickle-specific	Avoid hypoxia, stress, infection and hemorrhage; prevent infection, screen for PET and remain alert for acute chest syndrome.	**2**

What you must memorize from this station

- HbA2 >3% points toward thalassemia; both-carrier couples need genetic counseling/diagnosis.
- Sickle mutation: beta-chain position 6 glutamic acid -> valine; deoxygenation triggers sickling.
- Shared plan: MDT, growth scans from 20 weeks, Hb target 10, folate 5 mg, aspirin, postpartum LMWH.
- Thalassemia danger = iron overload/cardiac failure; sickle dangers = crisis, acute chest, infection, PET and VTE.

EXAM TRAPS: The shared six-point plan applies to both thalassemia and sickle cell disease. Do not give iron automatically to a hemoglobinopathy without proving deficiency.

Three-station master memory sheet

- Stage-specific Hb thresholds: 11 g/dL first trimester, 10.5 g/dL second/third, 10 g/dL postpartum.
- Pregnancy hemodilution: plasma volume +50%; MCV and MCHC unchanged.
- IDA: microcytic hypochromic, ferritin <30 micrograms/L, iron <12 micromol/L, saturation <15%.
- Oral iron first line; check Hb after 2-4 weeks; reticulocytes rise first; continue 3 months after normalization and to 6 weeks postpartum.
- Routine folate 400 micrograms/day; high risk 5 mg/day.
- Hemoglobin electrophoresis localizes thalassemia/sickle disease; partner status determines fetal genetic risk.

Source-specific wording retained: ferritin/iron/saturation thresholds, IV-iron timing tension, transfusion threshold and hemoglobinopathy management are reproduced from the attached lecture-derived guide without external replacement.

O2. Hyperemesis Gravidarum - Three Constructed OSCE Stations

Source set: Anemia and Hyperemesis Gravidarum(2).pdf

CONSTRUCTED-STATION SPINE: Confirm objective systemic illness -> exclude mimics -> investigate dehydration/metabolic effects -> rehydrate and protect with thiamine -> control vomiting -> prevent thrombosis and deficiency complications.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Diagnosing hyperemesis and excluding mimics

STUDENTS INFORMATION
A 26-year-old primigravida at 9 weeks has persistent vomiting, 6% loss of prepregnancy weight, dark urine, oliguria, tachycardia and postural hypotension. Discuss diagnosis, differential diagnosis and investigations.

Examiner questions

- Differentiate ordinary nausea/vomiting of pregnancy from hyperemesis.
- Identify objective severity features.
- List the diagnoses that must be excluded.
- Order and interpret the source investigations.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Normal NVP timing	Occurs in 50-80%; begins around 5 weeks, peaks 8-10 weeks and disappears by 16-18 weeks.	**2**
HG definition	Severe nausea/vomiting with dehydration, >5% prepregnancy weight loss, ketonuria/ketosis and electrolyte abnormalities; frequency 0.3-3%.	**2**
Clinical dehydration	Reduced urine output, dark urine, tachycardia, postural hypotension and muscle wasting.	**2**
Diagnostic principle	Hyperemesis is a diagnosis of exclusion.	**2**
Pregnancy-related mimics	Multiple pregnancy and molar pregnancy.	**2**
Infective mimics	UTI, ear infection and gastrointestinal infection.	**2**
Endocrine/metabolic mimics	Thyrotoxicosis, hyperparathyroidism, DKA and Addison disease.	**2**
Surgical/neurologic/drug mimics	Peptic ulcer, pancreatitis, cholecystitis, raised intracranial pressure, iron supplements and antibiotics.	**2**
Ultrasound	Confirm gestational age and assess multiple or molar pregnancy.	**2**
Laboratory set	CBC/raised hematocrit, electrolytes (low Na/K/Cl and metabolic alkalosis), TFT, glucose, LFT (abnormal in 50%), urine ketones and UTI assessment.	**2**

What you must memorize from this station

- HG = severe vomiting plus objective systemic consequences; >5% weight loss is a defining lecture threshold.
- Diagnosis of exclusion: pregnancy, infection, endocrine/metabolic, GI/surgical, neurological and drug causes.
- Raised hematocrit reflects hemoconcentration.
- Ultrasound must check dates, multiple gestation and molar pregnancy.

EXAM TRAPS: Severe vomiting alone is insufficient. Use dehydration, weight loss, ketosis and electrolyte disturbance.

Station 2: Stepwise management and antiemetics

STUDENTS INFORMATION
The same patient cannot tolerate fluids and has ketonuria, hyponatremia and hypokalemia. Explain immediate management, medication options, prevention and the criteria suggesting response.

Examiner questions

- Give the source sequence for initial resuscitation.
- State the lecture fluid protocol and electrolyte approach.
- List antiemetic regimens and adjuncts.
- Describe response/discharge-type targets.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Remove triggers/NPO	Stop drugs that may cause nausea/vomiting and keep NPO initially.	**2**
Fluid choice	IV normal saline and Ringer lactate; the lecture says dextrose-containing fluids are inappropriate.	**2**
Source fluid schedule	2 L in 2 hours, then 1 L every 4 hours for 3 days, reproduced as the lecture protocol.	**2**
Electrolytes	Correct sodium, potassium and chloride; avoid rapid sodium correction.	**2**
Thiamine	Give thiamine supplementation, especially before carbohydrate/dextrose exposure.	**2**
Prevention/support	Thromboprophylaxis, emotional support and frequent reassurance.	**2**
Antiemetic 1	Vitamin B6 10-25 mg orally every 6-8 hours; promethazine 12.5-25 mg oral/IM/rectal every 4-8 hours.	**2**
Antiemetic 2	Metoclopramide 5-10 mg IV/oral every 6-8 hours; prochlorperazine 5-10 mg oral/IV/IM every 6 hours or 25 mg rectally twice daily PRN.	**2**
Ondansetron/adjuncts	Ondansetron 8 mg oral/IM every 12 hours; source cautions congenital-defect risk before 10 weeks. PPIs are stated safe; corticosteroids may be used in severe cases.	**2**
Response targets	No ketonuria, tolerates oral diet and normal electrolytes; the source response slide ambiguously lists “nausea/vomiting,” interpreted as improvement but not rewritten as an exact criterion.	**2**

What you must memorize from this station

- Initial order: stop offending drugs -> NPO -> IV saline/Ringer -> correct electrolytes -> thiamine -> antiemetics -> thromboprophylaxis/support.
- Memorize the source fluid schedule and exact antiemetic doses.
- Avoid dextrose before thiamine protection.
- Response: ketones clear, oral diet tolerated, electrolytes normalize and vomiting clinically improves.

EXAM TRAPS: The exact fluid schedule is lecture-specific. Do not rapidly correct hyponatremia. Ondansetron has a special source caution before 10 weeks.

Station 3: Neurologic deterioration and serious complications

STUDENTS INFORMATION
After prolonged vomiting, a 12-week pregnant woman receives carbohydrate-containing fluid and develops confusion, blurred vision, nystagmus and gait ataxia. Discuss the diagnosis, recognition, complications and prevention.

Examiner questions

- Identify the immediate diagnosis and its cause.
- List symptoms/signs and confirmation.
- Explain why treatment sequence matters.
- List other serious complications of hyperemesis.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Diagnosis	Wernicke encephalopathy due to thiamine (vitamin B1) deficiency.	**2**
Precipitant	Carbohydrate-containing food/fluids can precipitate it in a thiamine-depleted patient.	**2**
Symptoms	Blurred vision, unsteadiness, confusion and memory problems.	**2**
Signs	Nystagmus, ophthalmoplegia, sixth-nerve palsy, gait ataxia or finger-nose ataxia.	**2**
Confirmation/prognosis	Diagnosis is clinical and may be confirmed by MRI; fatal but reversible; source associates it with 40% fetal loss.	**2**
Safety sequence	Administer thiamine before carbohydrate/dextrose and correct nutritional/volume deficits.	**2**
Hyponatremia	May cause lethargy and seizures.	**2**
Correction complication	Rapid sodium correction risks osmotic demyelination syndrome/central pontine myelinolysis.	**2**
Other physical complications	Mallory-Weiss tears and thrombosis.	**2**
Other deficiency/psychological effects	Vitamin B6 and B12 deficiency plus psychological distress; include thromboprophylaxis and emotional support.	**2**

What you must memorize from this station

- Wernicke triad domain: eye signs + ataxia + confusion/memory change in prolonged vomiting.
- Thiamine deficiency may be precipitated by carbohydrate; thiamine must precede dextrose.
- Hyponatremia causes seizures; rapid correction causes osmotic demyelination.
- Remember Mallory-Weiss tear, thrombosis, B6/B12 deficiency and psychological morbidity.

EXAM TRAPS: Do not wait for MRI to start thinking clinically. The source calls Wernicke fatal but reversible.

Three-station master memory sheet

- Ordinary NVP: 50-80%, starts 5 weeks, peaks 8-10, settles 16-18. HG: 0.3-3%, >5% weight loss plus dehydration/ketosis/electrolyte abnormality.
- HG is a diagnosis of exclusion; ultrasound for dates/multiple/mole and labs for hemoconcentration, electrolytes, thyroid, glucose, liver and urine.
- Use saline/Ringer, electrolyte correction, thiamine, antiemetics, thromboprophylaxis and support.
- Wernicke is the dangerous classic complication; carbohydrate can precipitate it.
- Never correct sodium rapidly.

Source-specific wording retained: the exact fluid schedule, antiemetic doses, ondansetron caution and ambiguous response wording are preserved as lecture-derived statements.

O3. Rh Isoimmunisation - Three Constructed OSCE Stations

Source set: Rh Isoimmunisation(2).pdf

CONSTRUCTED-STATION SPINE: D-negative mother -> antibody status -> fetal D status -> prevention after exposure or fetal-anemia surveillance after sensitization -> dose adequacy -> delivery/transfusion.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Unsensitized Rh-negative antenatal counseling

STUDENTS INFORMATION
A 29-year-old primigravida at 28 weeks is Rh(D)-negative. Her antibody screen is negative and her partner is Rh-positive. Counsel her about the disease mechanism and routine/incident anti-D prophylaxis.

Examiner questions

- Explain sensitization and HDFN.
- Distinguish prevention from established disease.
- State routine prophylaxis schedules.
- List sensitizing events and situations in which prophylaxis is unnecessary.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Mechanism	D-positive fetal RBCs enter a D-negative maternal circulation -> maternal anti-D IgG -> placental transfer in a D-positive pregnancy -> splenic RBC destruction.	**2**
Disease sequence	Hemolysis -> fetal anemia -> hyperdynamic circulation -> high-output heart failure -> hydrops fetalis -> fetal death.	**2**
Screening branch	All D-negative women: antibody screen at first prenatal visit. Negative = prevention pathway; positive = identify antibody and manage alloimmunization.	**2**
Routine regimen 1	300 micrograms (1500 IU) at approximately 28 weeks.	**2**
Routine regimen 2	Alternative lecture school: 100 micrograms (500 IU) at 28 and 34 weeks. Repeat ICT/antibody screen before routine dose.	**2**
Sensitizing events I	Miscarriage/induced abortion, invasive prenatal procedure, blunt abdominal trauma and external cephalic version.	**2**
Sensitizing events II	Ectopic pregnancy, fetal death in second/third trimester, APH in second/third trimester and hydatidiform-mole nuance.	**2**
Fetal-death nuance	The demise itself, not delivery, is the sensitizing event because it may result from massive FMH/occult abruption.	**2**
Molar nuance	Complete mole has no fetal RBCs; partial mole has fetal RBCs; early diagnostic uncertainty prevents a simplistic rule.	**2**
No-prophylaxis situations	Biologic father certainly D-negative or fetus confirmed D-negative by cfDNA/fetal testing. Invasive testing itself is not an exemption unless it establishes fetal D negativity.	**2**

What you must memorize from this station

- Anti-D prophylaxis prevents maternal immune memory; it does not treat an already sensitized pregnancy.
- Memorize both routine schedules: 300 micrograms at 28 weeks OR 100 micrograms at 28 and 34 weeks.
- Repeat antibody screening before routine dosing.
- Sensitizing events: loss/abortion, procedures, trauma, ECV, ectopic, fetal death, APH and partial/uncertain mole.

EXAM TRAPS: An Rh-negative mother does not automatically get the same answer: first ask whether anti-D is already present and whether the fetus can be D-positive.

Station 2: Postpartum fetomaternal hemorrhage and dose adequacy

STUDENTS INFORMATION
An unsensitized Rh-negative woman delivers an Rh-positive infant after a complicated delivery with suspected large fetomaternal hemorrhage. Explain testing, dose calculation and interpretation after anti-D.

Examiner questions

- Select screening and quantitative tests for fetomaternal hemorrhage.
- State dose coverage and conversion facts.
- Explain pharmacokinetics/passive antibody findings.
- Describe the prevention impact figures in the lecture.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Need to test	D-negative women should be assessed at delivery for excessive FMH so the dose covers the actual exposure.	**2**
Rosette test	Qualitative, sensitive screening test; a negative test leads to the standard anti-D dose.	**2**
Quantification	Kleihauer-Betke test or flow cytometry quantifies FMH.	**2**
FMH frequency facts	>20-30 mL in about 1/200-300 deliveries; >80 mL in 1/1000; >150 mL in 1/5000.	**2**
300-microgram dose	1 microgram = 5 IU; 300 micrograms = 1500 IU and covers 15 mL D-positive RBCs or 30 mL fetal whole blood.	**2**
50-microgram dose	50 micrograms = 250 IU and covers 2.5 mL D-positive RBCs or 5 mL fetal whole blood.	**2**
Volume distinction	Do not confuse packed fetal red-cell volume with fetal whole-blood volume; whole-blood figure is double.	**2**
Pharmacokinetics	Peak serum level faster IV than IM; half-life approximately 24 days.	**2**
Passive antibody	Low maternal titer <=4 may persist for weeks; newborn DAT may be positive without adverse clinical effects.	**2**
Efficacy sequence	Without complete prophylaxis 16%; postpartum prophylaxis 1-2%; postpartum plus routine antenatal prophylaxis 0.1-0.3%.	**2**

What you must memorize from this station

- Rosette screens; Kleihauer-Betke/flow cytometry quantify.
- 300 micrograms covers 15 mL RBCs/30 mL whole blood; 50 micrograms covers 2.5/5 mL.
- Anti-D half-life about 24 days; passive low titer and positive neonatal DAT may be harmless.
- Prevention reduces sensitization 16% -> 1-2% -> 0.1-0.3%.

EXAM TRAPS: Do not swap the RBC and whole-blood volumes. A positive DAT after prophylaxis does not automatically equal clinically significant neonatal hemolysis.

Station 3: Established alloimmunization and fetal anemia

STUDENTS INFORMATION
A 26-week Rh-negative woman has a positive indirect Coombs test with anti-D identified and a maternal anti-D level of 18 IU/mL. Discuss assessment and management of the fetus.

Examiner questions

- Confirm the diagnosis and determine whether the fetus is at risk.
- Explain maternal and fetal surveillance.
- Define hydrops and treatment options.
- Discuss delivery and neonatal issues.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Diagnosis	Maternal antibody screen/ICT positive followed by antibody identification/typing; remember anti-c and other antibodies are possible despite previous anti-D.	**2**
Fetal antigen status	Determine fetal Rh(D) type; confirmed D-negative fetus is not a target for anti-D HDFN.	**2**
Critical maternal value	Lecture critical anti-D value is 15 IU/mL; 18 IU/mL crosses the stated risk threshold.	**2**
Primary fetal surveillance	MCA peak systolic velocity by ultrasound, interpreted against gestational-age reference values.	**2**
Tests not preferred	Amniotic-fluid bilirubin is obsolete; fetal blood sampling is not routine.	**2**
Hydrops definition	At least two of skin edema, ascites, pericardial effusion and pleural effusion.	**2**
Pathophysiology	IgG-opsonized fetal RBCs are phagocytosed by fetal splenic macrophages, causing progressive anemia and cardiac decompensation.	**2**
Treatment near term	Deliver for neonatal treatment when severe fetal anemia is present and gestation is sufficiently advanced.	**2**
Treatment earlier	Intrauterine transfusion when delivery is not appropriate; lecture also lists serial plasmapheresis and investigational IVIG.	**2**
Neonatal planning	Coordinate neonatal assessment/treatment; distinguish maternal ICT from newborn DAT and remember passive prophylactic anti-D can affect DAT.	**2**

What you must memorize from this station

- Established disease: identify antibody -> fetal D type -> maternal level/titer -> MCA-PSV -> deliver or transfuse.
- Critical lecture value = 15 IU/mL.
- Hydrops requires at least two fluid/edema compartments.
- MCA-PSV is current noninvasive surveillance; amniotic bilirubin obsolete and FBS not routine.

EXAM TRAPS: Do not give prophylaxis as the main treatment once alloimmunization is established. Do not confuse 15 IU/mL maternal critical value with the 1500 IU contained in a 300-microgram dose.

Three-station master memory sheet

- Prevention and treatment are separate pathways.
- Routine schedules: 300 micrograms at 28 weeks or 100 micrograms at 28 and 34 weeks; screen first.
- After delivery/large FMH: rosette screen, KB/flow quantify and adjust dose.
- Critical established-disease value: maternal anti-D 15 IU/mL; monitor fetal MCA-PSV.
- Hydrops = two or more of skin edema, ascites, pericardial effusion and pleural effusion.

Source-specific wording retained: both prophylaxis schools, dose-volume conversions, critical 15 IU/mL value, molar nuance and invasive-testing ambiguity are preserved.

O4. Thyroid Disorders in Pregnancy - Three Constructed OSCE Stations

Source set: Thyroid Disorders in Obstetrics(2).pdf

CONSTRUCTED-STATION SPINE: Gestational age -> pregnancy-specific biochemistry -> hypothyroid replacement or Graves balance -> determine what crosses placenta -> predict fetal/neonatal effect -> manage storm in the correct sequence.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Thyroid physiology, test interpretation and hypothyroidism

STUDENTS INFORMATION
A woman with known hypothyroidism presents at 9 weeks. TSH is elevated and fT4 is low. She has severe nausea, recently started iron/calcium and admits inconsistent thyroxine use. Explain interpretation and management.

Examiner questions

- Explain normal thyroid physiology and laboratory changes in pregnancy.
- Classify the biochemical pattern.
- Identify reasons for under-replacement.
- State the lecture management/monitoring principles.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Early fetal physiology	Before 12 weeks maternal T4, not fT3, crosses placenta and is converted to T3 in fetal brain; after 12 weeks fetal thyroid becomes largely independent if iodine adequate. Term study transfer 0.008%.	**2**
TBG effect	Estrogen-driven glycosylation increases TBG half-life from about 15 minutes to 3 days and triples TBG by 20 weeks; total T4/T3 rise.	**2**
Test choice	Lecture regards total T4/T3 as unreliable and prefers fT4/fT3 with pregnancy/trimester-specific ranges.	**2**
hCG link	First-trimester hCG can stimulate TSH receptor, especially in multiple pregnancy, trophoblastic disease and hyperemesis.	**2**
Four patterns	Overt: low fT4/high TSH; known controlled: normal; subclinical: normal fT4/high TSH; hypothyroxinaemia: low T4/normal TSH.	**2**
Current diagnosis	Low fT4/high TSH = inadequately treated/overt hypothyroidism; initiate or optimize thyroxine urgently, especially first trimester.	**2**
Why symptoms insufficient	Hypothyroid, hyperthyroid and normal pregnancy symptoms overlap; target biochemical euthyroidism.	**2**
Absorption/adherence causes	Vomiting, iron/calcium binding, malabsorption, suboptimal preconception control and changed adherence.	**2**
Dose/monitoring	Source mentions 25- or 50-microgram (~25%) adjustments; test pre-pregnancy, early first trimester and later second/third trimester, using trimester ranges.	**2**
Source tensions	Lecture conclusion says established disease needs increased thyroxine, but earlier slide says not routinely; first-trimester control emphasized. Preserve both without inventing reconciliation.	**2**

What you must memorize from this station

- Before 12 weeks maternal T4 is crucial for fetal brain; maternal fT3 does not cross in the lecture.
- TBG triples by 20 weeks; total hormones rise, so use free hormones and pregnancy-specific ranges.
- Four patterns must be distinguished, especially subclinical vs hypothyroxinaemia.
- Check vomiting, iron/calcium and adherence before attributing abnormal TSH solely to pregnancy.

EXAM TRAPS: A low TSH in early pregnancy can be an hCG effect. Do not treat or interpret values with non-pregnant reference ranges.

Station 2: Graves disease, antithyroid therapy and fetal/neonatal risk

STUDENTS INFORMATION
A 22-week woman with Graves disease has persistent tachycardia and is taking carbimazole. Ultrasound later shows fetal tachycardia, growth restriction and a fetal goitre. Discuss maternal treatment and fetal/neonatal surveillance.

Examiner questions

- Identify clues and complications of Graves disease.
- Compare PTU and carbimazole.
- Explain fetal Graves and treatment.
- Plan delivery and neonatal assessment.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Main goal/complications	Achieve euthyroidism early; maternal PET, heart failure and thyroid storm; fetal IUGR, prematurity and stillbirth.	**2**
Clinical clues	Failure to gain weight despite appetite, tachycardia >100 not slowing with Valsalva and onycholysis. Eye signs/pretibial myxoedema do not reflect current activity.	**2**
Symptomatic control	Propranolol may control tachycardia/tremor/anxiety; source says benefits outweigh fetal-growth concern when required.	**2**
Antithyroid drugs	Carbimazole or PTU block synthesis; both cross placenta in similar amounts and can suppress fetal thyroid. Titrate to serial biochemistry and use lowest dose allowing control.	**2**
Pregnancy course	TSH-receptor antibody titre rises first trimester/puerperium, falls second/third; monthly TFT if stable, more frequently new/relapse; many reduce/stop drug then restart/increase postpartum.	**2**
Agranulocytosis	Both drugs can cause unpredictable agranulocytosis; sore throat must be reported immediately.	**2**
Fetal-risk population	Measure stimulating antibodies in active Graves and past Graves even after surgery or radioactive iodine.	**2**
Fetal Graves clues	After ~20 weeks: fetal tachycardia, excessive movements, FGR, oligohydramnios and goitre; can cause craniosynostosis, hydrops, IU death, polyhydramnios and obstructed labor from neck extension.	**2**
Fetal treatment	If advanced gestation deliver; otherwise high maternal PTU/carbimazole titrated to fetal heart rate. Fetal blood sampling not routine unless doubt.	**2**
Neonatal plan	Cord-blood TFT. Drug-related neonatal hypothyroidism usually transient; antibody-driven hyperthyroidism often appears 7-10 days after birth with poor feeding/weight loss.	**2**

What you must memorize from this station

- Graves antibodies and antithyroid drugs both cross placenta, but push fetal thyroid in opposite directions.
- Fetal thyroid can respond after about 20 weeks.
- Sore throat on PTU/carbimazole = agranulocytosis warning.
- Neonatal hyperthyroidism may be delayed 7-10 days because maternal drug clears before antibody.
- Radioactive iodine is totally contraindicated in pregnancy; surgery, if required, usually second trimester.

EXAM TRAPS: A mother treated by thyroidectomy or radioiodine can still have circulating stimulating antibodies. Do not use eye signs as a current disease-activity marker.

Station 3: Hyperemesis-related thyrotoxicosis, iodine deficiency and thyroid storm

STUDENTS INFORMATION
At 10 weeks, a woman with severe hyperemesis has suppressed TSH and very high fT4, no goitre or eye signs and tachycardia that improves with IV fluids. The examiner then asks how to manage thyroid storm and explain iodine deficiency.

Examiner questions

- Differentiate hCG-mediated biochemical hyperthyroidism from Graves disease.
- State whether antithyroid therapy is required.
- Explain iodine physiology and fetal consequence.
- Give the emergency sequence for thyroid storm.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
hCG mechanism/context	hCG stimulates TSH receptor; common with severe hyperemesis, multiple gestation and trophoblastic disease; source reports biochemical hyperthyroidism in >60% of severe HG.	**2**
Pattern/course	Suppressed TSH with high/very high fT4; symptoms start with pregnancy, normalize as HG resolves; cited series normalized by 19 weeks.	**2**
Distinguishing clues	No goitre/eye signs, tired/washed-out rather than hyperadrenergic, tachycardia improves with IV rehydration and thyroid autoantibodies are absent.	**2**
Treatment decision	Usually no antithyroid medication; treat hyperemesis and confirm biochemical normalization.	**2**
Iodine loss	Pregnancy increases GFR/iodine loss, thyroid uptake and transplacental transfer, depleting maternal stores.	**2**
Iodine consequence	Severe deficiency may prioritize maternal iodine trapping over fetal needs and cause cretinism in the lecture.	**2**
Storm first step	PTU 300-600 mg loading, then 150-300 mg every 4 hours to block synthesis.	**2**
Storm second step	Several hours later give iodide to inhibit hormone release; PTU must precede iodide.	**2**
Storm adjuncts	Propranolol, glucocorticoids, fluids, cooling, nutrition, electrolyte correction and oxygen.	**2**
Monitoring/delivery	Continuous maternal and fetal monitoring; avoid delivery during storm unless fetal condition requires it.	**2**

What you must memorize from this station

- Low TSH + HG + no goitre/eye signs + tachycardia improves with fluids = hCG effect, not Graves.
- Transient HG-related hyperthyroidism usually does not need antithyroid drugs.
- Iodine deficiency deprives mother/fetus of substrate and can cause severe neurodevelopmental injury.
- Storm order: PTU first -> iodide hours later -> beta blockade + steroids + intensive supportive care.

EXAM TRAPS: Never give iodide before PTU in the lecture sequence. Do not mistake an hCG-heavy first-trimester state for autoimmune Graves.

Three-station master memory sheet

- Use gestational age and pregnancy-specific ranges before labeling disease.
- Before 12 weeks maternal T4 supports fetal brain; TBG triples and total hormones rise.
- Hypothyroidism is a replacement/biochemical-monitoring problem; Graves is a maternal-control versus fetal-drug-effect balance.
- Graves antibodies can stimulate fetal thyroid after 20 weeks; drugs can suppress it.
- Radioactive iodine is contraindicated. Thyroid storm: PTU then iodide, plus propranolol, steroids and supportive care.

Source-specific wording retained: internal tensions regarding thyroxine dose adjustment, outcomes and placental transfer are flagged rather than silently reconciled.

PART II - GYNECOLOGY

Checklist-derived chapters plus Puberty and Vulvar/Vaginal constructed three-station topics.

G1. Puberty - Three Constructed OSCE Stations

Source set: Puberty(2).pdf

CONSTRUCTED-STATION SPINE: Timing -> sequence/milestone -> Tanner stage -> central versus peripheral or hypogonadotropic versus hypergonadotropic localization -> bone age/imaging -> cause-specific treatment.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Normal female puberty and Tanner staging

STUDENTS INFORMATION
A mother brings her 12-year-old daughter for reassurance about breast development, pubic hair and irregular early menstruation. Explain normal puberty, sequence and Tanner staging.

Examiner questions

- Define puberty and key milestone terms.
- Explain the HPO/adrenal sequence.
- Describe Tanner stages I-V.
- Counsel about menarche and early cycles.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Definition/terms	Puberty is reproductive and sexual development/maturation changing a child into an adult. Thelarche = breast; adrenarche = pubic/axillary hair; menarche = first menstruation.	**2**
Axis	GnRH -> LH/FSH -> ovary -> estradiol/progesterone feedback; ovarian estrogen initiates major female pubertal changes.	**2**
Developmental timeline	After childhood suppression, DHEA/DHEAS rise at 6-8 years and pulsatile gonadotropin secretion begins around 8-9 years.	**2**
Stage I	Prepubertal; growth 5-6 cm/year; papilla only; vellus/no terminal pubic hair.	**2**
Stage II	Growth 7-8 cm/year; palpable breast bud and enlarged areola; minimal coarse pigmented straight hair mainly along labia. Mean breast age 11.2 (9-13.4).	**2**
Stage III	Peak growth 8 cm/year at age 12.5; breast contour and areola enlarge; dark coarse curly hair over mons; axillary hair and acne. Mean 12.2 (10-14.3).	**2**
Stage IV	Growth 7 cm/year; areola/papilla form secondary mound; adult-quality hair without medial-thigh spread. Mean 13.1 (10.8-15.4).	**2**
Stage V	Adult breast contour with areola recessed; abundant adult pubic hair extends to medial thighs but not linea alba; source says no further height increase after 16. Mean 15.3 (11.9-18.8).	**2**
Menarche	Mean age 12.8 years; early cycles may be anovulatory, irregular and unpredictable.	**2**
Modifiers	Race, heredity, body weight and exercise are listed as factors influencing onset; preserve as lecture-listed modifiers.	**2**

What you must memorize from this station

- Breast bud = Tanner II; secondary areolar mound = IV; areola returns to general contour = V.
- Stage III is peak growth and adds axillary hair/acne.
- Menarche mean 12.8; early cycles may be irregular/anovulatory.
- Thelarche, adrenarche and menarche are different milestones.

EXAM TRAPS: Stage breast and pubic hair separately. Do not use the milestone terms as synonyms. Race-related Stage-II timing is lecture-specific, not a universal rule.

Station 2: Precocious puberty

STUDENTS INFORMATION
A 7-year-old girl has progressive breast development, pubic hair and accelerated growth. Discuss classification, causes, investigations and management.

Examiner questions

- Define precocious puberty.
- Distinguish central from peripheral forms.
- Plan investigations.
- State treatment logic.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Definition	Onset of puberty before 8 years in girls (and before 9 years in boys, as the lecture states).	**2**
Central type	Gonadotropin-dependent activation of central HPO axis.	**2**
Central causes	Unknown etiology 75%; CNS malformations and brain tumors 25%, as slide-specific proportions.	**2**
Peripheral type	Estrogen-dependent sex-steroid effect from peripheral source rather than normal central activation.	**2**
Peripheral causes	Hormone-secreting tumors and exogenous hormone ingestion/exposure.	**2**
Hormone profile	Determine hormonal pattern and whether process behaves as central or peripheral.	**2**
Bone age	Hand/wrist X-ray assesses skeletal maturation and advancement.	**2**
Imaging	Brain imaging for CNS causes; pelvic ultrasound for ovarian/uterine maturation or lesion.	**2**
Tumor assessment	Tumor markers when a hormone-secreting tumor is suspected.	**2**
Treatment	GnRH analogue for central activation plus treatment of cause; peripheral disease requires treatment/removal of hormone source.	**2**

What you must memorize from this station

- Cutoff: before 8 years in girls.
- Central = gonadotropin-dependent; peripheral = estrogen-dependent.
- Core tests: hormone profile, bone age, brain imaging, pelvic ultrasound and tumor markers.
- GnRH analogue targets central disease; every cause must also be treated.

EXAM TRAPS: Do not stop after saying “GnRH analogue.” The lecture separately requires treatment of the underlying cause.

Station 3: Delayed puberty

STUDENTS INFORMATION
A 15-year-old girl has no breast development or other secondary sexual characteristics. Discuss definition, localization, causes, investigations and management.

Examiner questions

- Define delayed puberty using the lecture cutoff.
- Use gonadotropins to classify the disorder.
- List causes in each branch.
- Plan investigations and treatment.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Definition	No signs of secondary sexual characteristics by age 14 years in the female-focused lecture.	**2**
Localization logic	Low FSH/LH = hypogonadotropic central/functional suppression; high FSH/LH = hypergonadotropic gonadal failure.	**2**
Hypogonadotropic causes I	Constitutional delay, anorexia nervosa and excessive exercise.	**2**
Hypogonadotropic causes II	Chronic illness, pituitary tumors and Kallmann syndrome.	**2**
Hypergonadotropic causes I	Idiopathic premature ovarian failure and autoimmune ovarian failure.	**2**
Hypergonadotropic causes II	Chemotherapy/radiotherapy, Turner syndrome and XX gonadal dysgenesis.	**2**
FSH/LH	First localization test to select the branch.	**2**
Additional tests	Karyotype for chromosomal/gonadal dysgenesis; pelvic ultrasound for uterus/ovaries; hand/wrist X-ray for bone age.	**2**
Treatment principles	Treat underlying nutritional, exercise-related, chronic, pituitary, ovarian or iatrogenic cause; watchful waiting when appropriate.	**2**
Replacement/growth	Gonadal hormone replacement when indicated; growth hormone is listed but not routine for every patient.	**2**

What you must memorize from this station

- Delayed puberty cutoff in this lecture = no secondary sexual characteristics by 14.
- Low gonadotropins: constitutional/functional/CNS. High gonadotropins: ovarian failure/dysgenesis.
- Start with FSH/LH, then karyotype, pelvic ultrasound and bone age.
- Treatment is cause-specific; hormone replacement and selected watchful waiting/growth hormone are listed.

EXAM TRAPS: Turner syndrome and XX gonadal dysgenesis are hypergonadotropic. Growth hormone is an option, not a universal treatment.

Three-station master memory sheet

- Normal sequence is a process: adrenal and ovarian clocks, Tanner I-V, then menarche.
- Precocious cutoff <8 years in girls; central vs peripheral directs investigation/treatment.
- Delayed cutoff no secondary sexual characteristics by 14; FSH/LH localizes central vs gonadal cause.
- Bone age is central to both early and delayed puberty assessment.

Source-specific wording retained: age cutoffs, Tanner age ranges, race-related timing note and the broad “no further growth after 16” statement are treated as lecture-specific.

24. Gynecological Examination and Pap Smear

Source set: 3 Gyne and pap smear exam.pdf

STATION SPINE: Prepare and consent → general/abdominal assessment → external vulval inspection → bimanual examination → speculum examination and sampling → inspect on withdrawal → complete safely.

Complete examination framework

- General examination, vital signs and weight; thyroid examination; breast examination.
- Abdomen: inspect for scars and ascites; palpate for tenderness and masses; palpate the groins for inguinal lymphadenopathy.
- Gynecological examination: external vulval examination, bimanual examination and speculum examination.

Preparation and prerequisites

- Introduce yourself; wash hands; explain what the examination involves and why; reassure that it should not be painful and that you will stop if it becomes too uncomfortable; obtain verbal consent.
- Request a chaperone; ensure privacy; use a comfortable bed with good lighting.
- Ask the patient to empty her bladder, remove clothing from the waist down and remove sanitary protection; cover with a sheet.
- Prepare gloves, lubricant, antiseptic where listed, an appropriate speculum, cytobrush, spatula and glass slide, plus swabs or Pipelle biopsy equipment when indicated.

External vulval examination

- Inspect pubic-hair distribution and secondary sexual characteristics; note clitoromegaly.
- Inspect vulva/labia and skin for erythema, color change, ulcers, warts, lesions, masses, tumors and cysts.
- Look for discharge, its color/consistency, and bleeding; inspect the perineal body and anus.
- Ask the patient to cough or bear down to assess urinary leakage and cystocele/rectocele/prolapse; palpate the labia majora for swellings.

Bimanual examination

- Insert two lubricated fingers properly while the abdominal hand palpates.
- Assess uterine size, direction/position, regularity/surface, mobility and tenderness.
- Assess both adnexa for masses and tenderness.

Cusco speculum sequence

- Warm and lubricate the speculum where explicitly scored. Separate the labia with the left hand; insert with blades vertical/sideways, rotate about 90° so the blades become horizontal, then open slowly under good light.
- Inspect the vaginal walls and cervix/external os for discharge, bleeding, ectropion/erosion, polyps, masses, inflammation or atrophy.
- Take a Pap smear with brush/cytobrush and spatula, HVS/endocervical swabs, biopsy or Pipelle sample as indicated.
- During withdrawal, close slightly and inspect the anterior and posterior vaginal walls; rotate back and remove gently.

Pap smear and vaginal-sample preparation

- The source recommends that the patient is not menstruating and avoids intercourse, vaginal medications, douches, spermicidal foam, cream or jelly for 48 hours beforehand.
- Label and send samples with the request form. Allow privacy to dress, thank the patient, discard equipment safely, wash hands, summarize findings and suggest further investigations.

Speculum types, uses and limitations

Instrument	Source-listed uses/advantages	Limitations
Cusco bivalve	Self-retaining; inspection of vagina/cervix; HVS, endocervical swab, Pap, Pipelle; IUD insertion/removal; IUI, egg collection, embryo transfer; PROM pooling. Easy and adjustable.	May hide anterior/posterior walls—inspect during gradual withdrawal. Source notes incomplete protection during cauterization.
Sims univalve (single/double)	More vaginal-wall exposure; D&C; evacuation of incomplete/missed/molar pregnancy; polypectomy/cervical biopsy; cerclage; hysteroscopy/laparoscopy; vaginal repair and vaginal hysterectomy.	Needs an assistant and is not self-retaining; cervix may be difficult to expose with a large cystocele.
Other named types	Pediatric, Huffman, Pederson, Graves and anal specula.	Named without further detail.

Other instruments named in the source

- Kidney dish; female catheter for intermittent catheterization under anesthesia.
- Uterine sound: determines uterine size/direction before D&C;, IUD insertion or biopsy; perforation is a risk.
- Hegar dilators: inserted sequentially to the internal os for evacuation, biopsy or molar pregnancy.
- Vulsellum/tenaculum: grasps and stabilizes the cervix; ovum forceps remove products or cervical polyps; sponge forceps hold gauze, clear the field or assist with laceration assessment in PPH.
- Sharp or blunt curette: biopsy/curettage/evacuation; aggressive curettage may cause Asherman syndrome.
- Green-Armytage forceps: catches uterine angles at cesarean section.
- Vacuum cup note: source indications include prolonged second stage and fetal distress; prerequisites include full dilation, ruptured membranes, engaged vertex, empty bladder and consent.

Checklist-specific scoring order

- Do not begin at the speculum: the source awards separate marks for prerequisites, external inspection, lateral walls, cervix, Pap sampling, withdrawal inspection and bimanual examination.
- Use the examiner terms: vulva/labia, skin lesions, external os, brush and spatula, uterine size/direction/regularity and adnexa.

25. Infertility and Subfertility

Source set: 4 Infertility.pdf

STATION SPINE: Define the couple's problem → investigate both partners simultaneously → start with semen analysis and ovulation assessment → assess tubes/uterus → choose timed intercourse, IUI, IVF/ICSI or corrective surgery.

Definitions and probability of conception

- Infertility: failure to achieve pregnancy after 12 months of regular unprotected intercourse when the woman is under 35; investigate after 6 months when she is over 35.
- Primary infertility: no previous conception (source proportion 70%). Secondary infertility: failure after a previous conception.
- Subfertility means reduced reproductive potential; sterility means complete inability to conceive.
- Source probabilities for a newly married couple: about 50% by 3 months, 75% by 6 months, 85% by 1 year and 95% by 2 years; monthly fecundability is about 20%.

Distribution of causes

Category	Source proportion/examples
Female	40%: ovulatory, tubal, uterine, cervical and endometriosis-related causes.
Male	30%: semen abnormality, sexual dysfunction, structural, environmental or genetic factors.
Combined	10%.
Unexplained	20% after standard assessment.

Female causes

- Ovulatory: PCOS (WHO II and most common in the source), premature ovarian failure/WHO III, hypogonadotropic hypogonadism/WHO I, hyperprolactinemia, thyroid disease, obesity and other endocrine dysfunction.
- Tubal: PID/STIs, previous pelvic surgery or ligation, adhesions, endometriosis and apparent HSG obstruction from mucus plug or spasm.
- Uterine: Müllerian anomalies, submucosal fibroid, endometrial polyp and Asherman syndrome.
- Cervical: stenosis, chronic inflammation and Müllerian-duct abnormality.
- Source-listed controversial/other factors: immune "killer T cells," thrombophilia, luteal-phase defect/inadequate progesterone and ovarian cyst.

Male causes

- Environmental/lifestyle: smoking, alcohol, heat, tight underwear and stress.
- Sexual dysfunction: erectile or ejaculatory dysfunction.
- Structural: varicocele, torsion and vasectomy; abnormal semen after mumps or antisperm antibodies.
- Genetic: cystic fibrosis, Klinefelter syndrome and immotile-cilia disorders.

Focused couple history

- Both partners' ages; duration of infertility; previous conception; cohabitation; frequency, timing and last intercourse; knowledge of ovulation; previous investigations/treatment.
- Woman: menstrual pattern, dysmenorrhea, midcycle pain/spotting, pregnancy/obstetric history, contraception/IUD, PID, endometriosis, fibroid, thyroid symptoms, galactorrhea, hirsutism, pelvic surgery, family history and medications.
- Man: childhood mumps, testicular trauma/torsion/cancer, heat exposure, medications, chemotherapy/radiation, smoking, alcohol, diet and sexual function.

Semen-fluid analysis (SFA)

Parameter	Source values/notes
Volume	Hypospermia <1.5 mL; source normal 1.5–5 mL, while another station uses >2 mL.
Concentration/total	Normal >15 million/mL and total >39 million; handwritten older note also shows >80 million total.
pH	Basic, 7.2–8.
Motility	A (rapid progressive) >25%, or A+B >50%; B is sluggish progression.
Morphology	A station note records >50%; retain as source wording.
Abnormal result	Repeat after 4–6 weeks; then assess for IUI or ICSI as indicated.

Source conflict retained: semen reference values vary across the uploaded notes; all listed values are preserved.

Female investigations

- Pregnancy test; pelvic examination and transvaginal ultrasound including antral-follicle count.
- Hormones: FSH, LH, AMH, estradiol, TSH/T4, prolactin, mid-luteal progesterone and other source-listed endocrine testing.
- Tubal/uterine assessment: HSG; if abnormal, laparoscopy with dye test; hysteroscopy; D&C; ultrasound and other structural assessment.
- Karyotype when indicated. Correct thyroid or prolactin abnormality before proceeding.

Male investigations

- General and genital physical examination, testicular ultrasound, SFA, hormonal profile and karyotype where indicated.

Diagnostic laparoscopy/hysteroscopy/D&C; station

- Arrange admission, consent, procedure in the second half of the cycle, pregnancy testing and general anesthesia.
- The source describes two abdominal ports, combined laparoscopy/hysteroscopy, blue-dye tubal-patency assessment and diagnostic D&C.;
- Benefits: direct assessment of uterine, tubal and ovarian abnormalities, endometriosis, infection/adhesions and tubal patency.
- Complications listed: vascular laceration, uterine perforation, cardiorespiratory effects of pneumoperitoneum, bowel injury and bladder injury.

Management by cause

- Ovulation induction with timed intercourse; source drugs include clomiphene in PCOS and later notes also discuss other ovulation-induction options.
- IUI for selected cervical/male/unexplained factors; IVF for irreparable tubal disease, oligospermia or unexplained infertility; ICSI for severe male-factor disease.
- Tubal options: adhesiolysis, reanastomosis/tuboplasty, or salpingectomy followed by IVF in severe disease. Cervical stenosis may be dilated or bypassed with IUI.
- Asherman syndrome after PPH/curettage: amenorrhea/hypomenorrhea, infertility or recurrent loss; assess with US/SIS/HSG/hysteroscopy; treat with hysteroscopic adhesiolysis and estrogen.

26. Menopause

Source set: 5 Menopause (HAVOCS).pdf

STATION SPINE: Define and date menopause → recognize HAVOCS and urogenital symptoms → assess cardiovascular/bone/cancer risks → investigate appropriately → individualize nonhormonal or hormonal treatment.

Definitions and terminology

- Menopause: permanent cessation of menstruation from cessation of ovarian function; diagnosed retrospectively after 12 months of amenorrhea. Average age is about 48–52 years; source mean is 51.
- Perimenopause: from first climacteric symptoms until 1 year after the final menstrual period, averaging about 4 years. Premenopause: immediately before menopause. Climacteric: transition from reproductive to nonreproductive life. Postmenopause: after the final menstrual period.
- Premature menopause is before 40 and may be induced by surgery, chemotherapy or radiation. The source separately describes premature ovarian failure before 30 with X-chromosome, Fragile-X, galactosemia or autoimmune/endocrine associations.

Physiology and hormone pattern

- Progressive oocyte atresia after roughly 400 ovulations leads to ovarian resistance to gonadotropins and falling estrogen, progesterone and androgen production.
- Inhibin and estradiol fall; estrone becomes relatively prominent; progesterone is low; FSH and LH rise. The source notes FSH >40 in established ovarian failure.

Symptoms: HAVOCS and time course

Period	Source features
Early, approximately 0–5 years	Amenorrhea; hot flushes/night sweats/heat intolerance (up to 85%); mood disturbance, depression, anxiety, insomnia, irritability, poor concentration/memory; aches; dry/itchy skin and collagen loss; coarse hair; reduced libido.
Intermediate, 5–10 years	Vaginal dryness and atrophy, dyspareunia, sexual dysfunction, sensory urgency, recurrent UTI, urinary incontinence and urogenital prolapse.
Long term, >10 years	Osteoporosis (spine/femoral neck), cardiovascular disease with LDL rise/HDL fall, and dementia in the source.
HAVOCS mnemonic	Hot flushes; Atrophy of vagina; Osteoporosis; Coronary artery disease; Sleep impairment.

Risk modifiers and examination

- Earlier menopause: genetic tendency, smoking (source says about 3 years earlier), chemotherapy/radiotherapy, steroids, fair/thin body habitus, sedentary lifestyle and low calcium intake. The source states number of pregnancies and OCP use do not accelerate menopause.
- Examine general state, BP and cardiovascular risk; breast size/texture; thyroid; abdomen/pelvis; vaginal, urethral and cervical atrophy; prolapse and urinary leakage.

Investigations

- FSH: source uses >30 and recommends two measurements 2 weeks to 3 months apart when confirmation is needed.
- Exclude pregnancy and other endocrine causes with hCG, TSH and prolactin; assess lipids/cardiovascular risk.
- Bone assessment: calcium, bone-mineral density/DEXA. Cancer-health maintenance: breast examination/mammogram and cervical smear.
- The station checklist specifically awards Pap smear, mammogram and lipid profile.

Management

- Lifestyle and bone protection: calcium/vitamin D, stop smoking and excess alcohol, weight-bearing exercise. Alendronate/bisphosphonate is listed as first-line osteoporosis treatment; estrogen is not first-line for osteoporosis in the notes.
- Nonhormonal options for vasomotor symptoms: antidepressant, beta-blocker and alpha-agonist.
- HRT: estrogen plus progestin if the uterus is present; estrogen alone after hysterectomy; testosterone may be added for selected symptoms. Use the minimum effective dose for the shortest duration (source average 2–3 years), mainly for vasomotor or urogenital symptoms.
- Source HRT risks: breast cancer, venous thromboembolism and endometrial cancer.

HRT contraindications listed in the source

- Estrogen/progesterone receptor-positive breast cancer; estrogen-dependent endometrial cancer; coagulopathy; DVT/PE; stroke/TIA; coronary disease; active liver disease; unexplained vaginal bleeding. Handwritten notes also add uncontrolled hypertension and pregnancy.

Station answer map

- 52-year-old with no bleeding for 2 years: define menopause; state age 48–52; give any three symptoms (hot flushes, vaginal dryness, mood disturbance, sexual dysfunction, sleep disturbance, urinary incontinence); two risks (cardiac, osteoporosis, genital prolapse); and three investigations (Pap smear, mammogram, lipid profile).

27. Amenorrhea

Source set: 6 Amenorrhea.pdf

STATION SPINE: First exclude pregnancy → separate primary from secondary amenorrhea → use secondary sexual characteristics and uterine presence to guide FSH/LH, karyotype and imaging → treat the specific compartment disorder.

Definitions and when to investigate

- Primary amenorrhea: no menses by age 15 with normal secondary sexual characteristics, or by age 13 without them. A source flowchart also uses age 14 without secondary characteristics and age 16 with them.
- Secondary amenorrhea: no menses for 6 consecutive months after previously regular cycles, or 12 months after prior oligomenorrhea.
- Always exclude pregnancy first. The source identifies pregnancy as the most common cause of secondary amenorrhea.

Source threshold conflict retained: the uploaded algorithms use both 13/15 and 14/16 age thresholds for primary amenorrhea.

Primary amenorrhea: absent secondary sexual characteristics

- Perform pelvic ultrasound for ovaries/uterus, karyotype, FSH/LH and assess height.
- Low FSH/LH → hypothalamic/pituitary causes: intracranial lesion, hydrocephalus, skull trauma, craniopharyngioma, empty sella, Kallmann syndrome, Laurence–Moon–Biedl, Prader–Willi, constitutional delay, weight loss/anorexia, excessive exercise, chronic illness or stress.
- High FSH/LH → gonadal failure: premature ovarian failure after chemotherapy/pelvic irradiation; Turner syndrome 45XO with short stature/streak ovaries; 46XX or 46XY gonadal agenesis/dysgenesis.

Primary amenorrhea: secondary sexual characteristics present

- Use ultrasound and karyotype to determine whether a uterus is present.
- Absent uterus: 46XX Müllerian agenesis/MRKH or 46XY complete androgen-insensitivity syndrome.
- Present uterus: outflow obstruction (imperforate hymen, transverse vaginal septum, absent vagina) or endocrine causes such as hypo/hyperthyroidism, hyperprolactinemia, Cushing syndrome or PCOS.
- Source androgen interpretation: total testosterone ≥5 suggests late-onset CAH, Cushing syndrome, androgen tumor or 5-alpha-reductase deficiency; 2.5–5 is associated with PCOS.

Focused history and examination

- Sexual activity and pregnancy possibility; cyclic pain/hematocolpos; growth and pubertal development; stress, depression, weight loss, diet, exercise and chronic illness.
- Headache, visual symptoms or galactorrhea; family history of delayed menarche or premature menopause; autoimmune disease; chemotherapy/radiation; antipsychotics, opioids and other drugs.
- Examine height, weight/BMI, BP, Tanner stage, breasts and hair; external genitalia/clitoromegaly; vaginal rugosity/atrophy; hirsutism, virilization, acne, galactorrhea, thyroid/Cushing signs and chromosomal features; abdomen/pelvic mass and visual fields.

Core investigations

- Urine/serum hCG; FSH, LH, prolactin, TSH and testosterone/SHBG; other adrenal enzymes when indicated.
- Transabdominal pelvic ultrasound; karyotype; examination under anesthesia/vaginoscopy if appropriate; bone-density assessment; CT/MRI of the head for central disease.

Key primary-amenorrhea syndromes

Condition	Source pattern and management
MRKH	46XX, normal ovaries/hormones and secondary characteristics, normal external genitalia, absent uterus/upper vagina; renal anomalies 15–40%, skeletal 10–20%. Assess karyotype, renal US/MRI/laparoscopy. Create/maintain a functional vagina; oocyte retrieval with surrogacy or uterine transplant is listed.
Complete AIS	46XY; testes make testosterone and AMH but androgen-receptor defect; female external phenotype, absent Müllerian structures, short blind vagina, breast development, absent pubic/axillary hair. Functional vagina; delay gonadectomy until after puberty; give HRT.
Swyer syndrome	46XY gonadal dysgenesis; no AMH/androgens, therefore uterus/cervix/tubes develop but no secondary characteristics. Pubic hair argues against complete AIS. Remove gonads after diagnosis because source malignancy risk is 20–30%.
Turner syndrome	45XO; short stature, webbed neck, cubitus valgus, widely spaced nipples, cardiac/renal/autoimmune-thyroid disease and streak ovaries. Mosaic cases may menstruate but develop POF. Low-dose estrogen followed by cyclic estrogen/progesterone; egg donation.
Kallmann	Congenital GnRH deficiency with anosmia/hyposmia; KAL mutation at Xp22.3 and migration defect in the source.
CAH	Usually 21-hydroxylase deficiency; androgen excess, ambiguous genitalia at birth and possible salt wasting.

Outflow obstruction and hypothalamic causes

- Imperforate hymen: normal secondary characteristics with cyclic pain; source treatment is cruciate incision. Transverse septum: septoplasty.
- Anorexia: weight 10–12% below ideal, delayed secondary characteristics, hypothermia/cold intolerance, bradycardia, hypotension and lanugo; low LH/FSH/estradiol. Manage nutrition, psychological illness and selected estrogen support.
- Athletic amenorrhea: stress/energy deficit suppresses GnRH and may cause osteoporosis/stress fracture (female-athlete triad). Improve diet and reduce exercise; estrogen/progesterone or COC if lifestyle measures fail.

Secondary amenorrhea: cause distribution and evaluation

- Source proportions: ovarian 40%, hypothalamic 35% and uterine 5%.
- History: pregnancy/contraception, previous cycles, heavy bleeding/PPH/curettage, weight/exercise/stress, medication, cranial radiation/head injury, headache/vision/galactorrhea, acne/hirsutism, thyroid symptoms, hot flushes/vaginal dryness, chemotherapy, diabetes/autoimmune disease and family menopause before 40.
- Examine BMI, acanthosis, galactorrhea, hirsutism/virilization, thyroid/Cushing signs and visual fields; order hCG, FSH/LH, prolactin, TSH, testosterone/SHBG and pelvic ultrasound.

Asherman and Sheehan syndromes

- Asherman syndrome after uterine instrumentation/curettage: amenorrhea or hypomenorrhea, infertility and recurrent loss; US/SIS/HSG/hysteroscopy; hysteroscopic adhesiolysis plus estrogen.
- Sheehan syndrome: pituitary infarction after PPH/hypovolemia. Failure to lactate is the earliest source feature, followed by amenorrhea, low libido, fatigue and weight loss; GH/prolactin/gonadotropins and sometimes ACTH/TSH are deficient.

Drugs associated with hyperprolactinemia in the notes

- Dopamine antagonists; tricyclics, antipsychotics, antiemetics, SSRIs and MAOIs; H2 blockers; reserpine; methyldopa; opiates; benzodiazepines; barbiturates; estrogens and antiandrogens.

28. Polycystic Ovary Syndrome

Source set: 7 Pcos (retrodram critiria).pdf

STATION SPINE: Recognize oligo/anovulation plus hyperandrogenism → exclude mimics → apply at least two Rotterdam criteria → screen metabolic/endometrial risk → treat according to cycle, hair, metabolic or fertility goal.

Core concept and Rotterdam criteria

- PCOS is chronic anovulation associated with irregular or absent menses, subfertility, obesity and hyperandrogenic features. It may begin before menarche; FSH/LH may remain in the normal range.
- After excluding other causes, diagnose with at least two of: oligo/anovulation; clinical or biochemical hyperandrogenism; polycystic ovaries on ultrasound.
- Source ultrasound description: 10–12 small follicles in at least one ovary, usually bilateral, mostly <10 mm and/or ovarian volume >10 mL (“string of pearls”). The source cautions against using ultrasound within 8 years of menarche.
- Obesity and insulin resistance are common associations but not required diagnostic criteria.

Phenotypes and risk factors

- Phenotype A: hyperandrogenism + ovulatory dysfunction + polycystic ovaries. B: hyperandrogenism + ovulatory dysfunction. C: hyperandrogenism + polycystic ovaries. D: ovulatory dysfunction + polycystic ovaries.
- Risk/association list: oligo-ovulation, obesity, insulin resistance/type 1 or type 2 diabetes, premature adrenarche, first-degree family history and antiepileptic drugs, especially valproate.

Presentation and station scenario

- Menstrual irregularity, oligomenorrhea or amenorrhea; infertility; hirsutism, acne and coarse dark facial/lower-abdominal hair; obesity, insulin resistance and acanthosis nigricans.
- Psychological associations: anxiety, depression, psychosexual difficulty and eating disorder; source also lists metabolic syndrome and sleep apnea.
- Melanie station: 24 years, no period for 6 months despite repeated negative pregnancy tests, BMI 29 kg/m², coarse dark hair on face and lower abdomen, long-standing irregular cycles, weight gain after marriage and desire for pregnancy.

Focused history/examination and exclusions

- Cycle pattern, infertility and pregnancy tests; hirsutism/acne; galactorrhea and thyroid symptoms; weight/lifestyle; drug/social/family history.
- Examine BMI, BP, hair/acne, acanthosis and signs of virilization or alternative endocrine disease.
- Exclude pregnancy, thyroid disease, hyperprolactinemia, congenital adrenal hyperplasia, androgen-secreting tumor, Cushing syndrome and other hyperandrogenic conditions.

Investigations

- β-hCG, TSH, prolactin, FSH/LH, androgen profile including free testosterone, DHEAS, SHBG and 17-hydroxyprogesterone when indicated; source also lists IGF-1.
- Glucose screening/OGTT (source triggers it when BMI >28), fasting lipids and pelvic ultrasound.
- Continue BP, weight, smoking, glucose and lipid surveillance; one source suggests lipids every 2 years and BP yearly.

Complications

- Subfertility/infertility, menstrual irregularity and pregnancy loss; endometrial hyperplasia/cancer from chronic anovulation; type 2 diabetes and cardiovascular/metabolic disease; pregnancy-related complications; psychological burden.

Management by patient goal

Goal	Source-listed options
All patients	Lifestyle change, diet, exercise and weight reduction first.
Cycle/endometrial protection	Combined oral contraceptive or cyclic progestogen; source notes OCP regulates cycles and reduces endometrial-hyperplasia risk.
Hirsutism/acne	OCP, antiandrogen such as spironolactone, laser/electrolysis; ensure appropriate contraception with antiandrogen therapy.
Metabolic	Metformin and ongoing glucose/lipid/BP monitoring.
Pregnancy	Ovulation induction and specialist referral. Source sequence: letrozole first, gonadotropins second, IVF third; other station material names clomiphene.
Resistant fertility disease	Laparoscopic ovarian drilling/diathermy by an expert, once in a lifetime in the source; temporary effect and possible reduction of ovarian reserve.

Clomiphene adverse effects listed

- Abnormal bleeding, breast tenderness, headache, nausea/vomiting, diarrhea, flushing and visual disturbance.

Source wording retained: the file title spells “retrodram criteria”; the diagnostic criteria shown are the Rotterdam criteria.

29. Vaginal Discharge and Lower Genital Tract Infections

Source set: 9 Vaginal discharge (amsel criteria) .pdf

STATION SPINE: Characterize color/odor/consistency and inflammation → perform speculum, pH and wet-mount testing → apply Amsel criteria for BV → distinguish Candida and Trichomonas → treat the source-defined organism.

Discharge color guide in the source

- Clear: normal, pregnancy, ovulation or hormonal. White: physiologic or yeast. Grey: bacterial vaginosis. Yellow-green: sexually transmitted infection. Pink: cervical bleeding/irritation or implantation. Red: menstruation, cervical infection/polyp or endometrial/cervical cancer.

Bacterial vaginosis (Gardnerella/anaerobic overgrowth)

- Source prevalence 22–50% and described as the most common cause. It is an imbalance with reduced lactobacilli rather than a true inflammatory infection.
- Risk associations: sexual activity (source says not a true STI), IUCD, douching, pregnancy and smoking.
- Often asymptomatic; thin homogeneous grey/white or yellow adherent discharge with fishy odor. Pruritus and pain are uncommon.

Amsel criteria: diagnose when 3 of 4 are present

- Clue cells on saline wet mount.
- Vaginal pH >4.5.
- Positive amine/whiff test after KOH.
- Thin, homogeneous grey-white/yellow adherent discharge.
- Source treatment: symptomatic BV with metronidazole or clindamycin for 7 days. Listed associations/complications: preterm birth, spontaneous abortion, postpartum endometritis and reinfection; retest at 3 months in the notes.

Candidal (monilial) vaginitis

- Station pattern: 37-year-old G6P6 with regular cycles and recurrent odorless, heavy, plaque-like white-to-yellow discharge despite local/systemic therapy.
- Risk factors: antibiotics, stress, pregnancy, diabetes, depressed immunity/HIV/steroids, moist environment and topical contraceptives.
- Symptoms/signs: intense vulvar itching and burning, dysuria, dyspareunia, excoriations, erythema and white curdy/cheese-like sticky odorless discharge. Vaginal pH is 4–4.5; KOH wet preparation shows pseudohyphae.
- Source treatment notes list fluconazole or topical azole. One uploaded teaching page labels Candida as an STI; this is retained as a source statement rather than harmonized.

Trichomonas vaginalis

- Source describes a pear-shaped protozoan/STI that may reside in male semen.
- Foul, frothy yellow-green discharge, strawberry cervix, vulvar burning/pruritus, dyspareunia and dysuria; pH >4.5; detect on wet mount.
- Treat both partners with metronidazole. Source pregnancy associations include preterm birth and IUGR.

Work-up and differential diagnosis

- General/physical and external examination; speculum examination; vaginal swab for culture/sensitivity; saline wet smear; 10% KOH wet smear; vaginal pH and whiff test.
- Additional source tests: WBC, CRP, ESR, Pap smear and pelvic ultrasound when the presentation requires broader assessment.
- Differentials listed by the station: bacterial vaginitis, bacterial vaginosis, Trichomonas, contact vulvitis, atrophic vulvitis and pinworms; expanded notes add local allergy, urinary causes, medication/immunosuppression, malignancy and PID.

Exam trap: the checklist scores “bacterial vaginitis” and “bacterial vaginosis” as separate entries; reproduce both if answering that exact station.

30. Pelvic Inflammatory Disease and Cervicitis

Source set: 8 PID (postcoital bleeding)p.pdf

STATION SPINE: Recognize an ascending reproductive-tract infection → take sexual/bleeding history → examine cervix and pelvis → obtain Chlamydia/gonorrhea testing and inflammatory assessment → treat patient and partner → admit when severe.

Definition, organisms and risk factors

- PID is inflammation/infection of the upper genital tract ascending from the vagina/cervix.
- Source organisms: Chlamydia (most common), gonorrhea, E. coli and Streptococcus.
- Risk factors: age under 35/teenage, multiple sexual partners, unprotected intercourse, IUCD, nulliparity and previous STI.

Acute PID presentation

- Lower abdominal/pelvic pain and tenderness, pain with walking or intercourse, abnormal discharge, fever/chills, cervical-motion tenderness, adnexal tenderness, rebound or right-upper-quadrant pain.
- Diagnosis is mainly clinical in the source. Classic triad: pelvic pain, cervical-motion tenderness and adnexal tenderness, often with mucopurulent discharge.

Postcoital-bleeding/cervicitis station

- Layla: 36–38 years, recently married; intermenstrual and postcoital bleeding, dyspareunia, no dysmenorrhea, Microgynon use, heavy smoker (2 packs/day for 4 years), no previous cervical smear, previous partner with multiple sexual partners.
- Findings: temperature 37.2°C, mild right-iliac-fossa tenderness, red cervix, yellow discharge, 1×2 cm raw area that bleeds on touch; Pap smear negative for neoplasia; cervical biopsy shows cervicitis.
- Acknowledge risk of both PID and cervical cancer. A normal Pap smear does not end the evaluation of an abnormal cervix.

Investigations

- Gynecological pelvic examination and speculum examination.
- HVS and endocervical swabs/cultures for Chlamydia and gonorrhea; CBC/WBC, ESR and CRP.
- Pregnancy/urine testing when appropriate; transvaginal ultrasound; laparoscopy when diagnosis remains uncertain or complications are suspected.
- For an abnormal cervix/postcoital bleeding: Pap smear, cervical biopsy and colposcopy referral.

Treatment goals and source regimen

- Eradicate infection/inflammation in the woman and sexual partner, improve symptoms and examination findings, and prevent long-term sequelae.
- One source regimen: single-dose intramuscular ceftriaxone plus doxycycline twice daily for 14 days unless admission is required.
- Treat the husband/partner once PID is confirmed; the notes separately state doxycycline for a recent contact interval written as “last 6 days.”

Admission criteria

- Surgical emergency such as appendicitis cannot be excluded; failure of oral treatment; severe illness with persistent/recurrent high fever or nausea/vomiting; tubo-ovarian abscess; pregnancy in the extended notes.

Tubo-ovarian abscess and chronic PID

- TOA is described as end-stage acute PID: severe bilateral pain, sepsis/high fever/tachycardia/hypotension, peritoneal signs and adnexal mass; CT may show bilateral complex masses. Admit for IV clindamycin plus gentamicin in the source; about 75% respond, otherwise drain.
- Chronic PID: chronic pain, infertility, dyspareunia, ectopic pregnancy, AUB and adhesions, often without fever/discharge or raised WBC/ESR. Ultrasound may show hydrosalpinx; laparoscopy shows adhesions; analgesia/adhesiolysis is listed.
- Fitz-Hugh–Curtis syndrome: right-upper-quadrant pain from perihepatic adhesions.

Long-term sequelae

- Chronic pelvic pain, recurrent infection, dyspareunia/sexual problems, pelvic adhesions, infertility, ectopic pregnancy, abnormal bleeding and recurrent abortion in the source lists.

31. Abnormal Uterine Bleeding (PALM–COEIN)

Source set: 10 Abnormal uerine bleeding (palm coein).pdf

STATION SPINE: Confirm stability and exclude pregnancy → define the bleeding pattern → use PALM-COEIN → examine for anemia/structural disease → investigate endometrium and hormones → control acute bleeding or treat chronic cause.

Normal-cycle reference and terminology

- Source normal cycle: 24–38 days, cycle-to-cycle variation 7–9 days, duration up to 8 days and blood loss under 80 mL.
- AUB is symptomatic variation in frequency, regularity, duration or volume from menarche to menopause, including intermenstrual bleeding.
- Polymenorrhea <21 days; oligomenorrhea >35 days; menorrhagia >80 mL or >7 days with regular cycles; metrorrhagia irregular or between normal periods; menometrorrhagia heavy irregular bleeding.
- Dysfunctional uterine bleeding (DUB): hormonal/ovulatory dysfunction without organic or pregnancy cause, common around puberty and perimenopause.

PALM–COEIN classification

Structural (PALM)	Nonstructural (COEIN)
P – Polyp	C – Coagulopathy, including von Willebrand disease and drugs such as aspirin/anticoagulants.
A – Adenomyosis	O – Ovulatory dysfunction.
L – Leiomyoma	E – Endometrial cause, including endometritis with heavy/intermenstrual bleeding.
M – Malignancy and hyperplasia	I – Iatrogenic, including gonadal steroids and cesarean-scar defect.
	N – Not otherwise classified, e.g., arteriovenous malformation.

Focused history

- Duration of problem; length of each period; heaviness, flooding, pad saturation, clots and previous normal cycle pattern.
- Dysmenorrhea/abdominal pain; intermenstrual and postcoital bleeding; last Pap result; contraception; pregnancy possibility.
- Symptoms of anemia (fatigue, dizziness, headache, postural symptoms), hypothyroidism and systemic/coagulation disease; prior investigations/treatment and medications.

Examination

- Assess stability, pallor, pulse and postural BP; general condition and thyroid.
- Abdominal examination for masses; speculum examination and bimanual pelvic examination.

Investigations and procedures

- Pregnancy test; CBC/hemoglobin, iron/ferritin, coagulation profile, TSH, prolactin, estrogen/progesterone when indicated, LFT, creatinine/BUN.
- Transvaginal ultrasound, saline-infusion sonography, Pap smear, hysteroscopy, D&C;/endometrial biopsy; laparoscopy for selected pathology.
- Menorrhagia station endpoint: no organic cause except iron-deficiency anemia and D&C; histology showing hormonal imbalance → DUB.

Acute AUB management in the source

- Assess hemodynamic stability; admit/transfuse for severe anemia or instability. The source gives Hb <7 g/dL as a transfusion/admission trigger.
- Control bleeding before delayed imaging; perform endometrial biopsy after stabilization if cancer risk is present.
- Medical first-line for a stable, minimally symptomatic nonpregnant patient: high-dose progestin-only therapy. Escalation options include D&C;, intrauterine balloon tamponade and selective embolization, plus treatment of the cause.

Chronic medical management

- NSAIDs reduce blood loss by 20–30% in the source; tranexamic/antifibrinolytic therapy up to about 40%. One note says not to combine antifibrinolytic with estrogen.
- Combined hormonal methods or progestogens/norethisterone coordinate endometrial shedding; LNG-IUS/Mirena suppresses endometrium; endometrial ablation is listed.

- Checklist medical list: tranexamic acid, NSAIDs, combined oral contraception, progestogens/norethisterone with Mirena, and GnRH agonist.

> Exam trap: do not describe “heavy bleeding” vaguely. The checklist separately scores duration, pad saturation/flooding, clots, intermenstrual bleeding and postcoital bleeding.

32. Urinary Incontinence

Source set: 11 Urine incontinance.pdf

STATION SPINE: Identify when leakage occurs → distinguish stress, urge, overflow, bypass and mixed patterns → perform the source test/urodynamics → begin conservative treatment and escalate according to type.

Definition and shared risk factors

- Urinary incontinence is involuntary urine leakage causing social or hygienic difficulty.
- Risk factors shared with prolapse: family/personal history, multiple vaginal births (strongest source factor), macrosomia, forceps, menopause/connective-tissue weakness, age/HRT, obesity, chronic cough/smoking, constipation/straining, heavy lifting, hysterectomy, pelvic surgery and previous vaginal repair.

Types and distinguishing features

Type	Source pattern	Assessment/management
Stress urinary incontinence	Most common in women; leakage with exertion, cough or sneeze from increased intra-abdominal pressure; often associated with cystocele. Urethral hypermobility 85–90%; intrinsic sphincter deficiency 10–15%, older/estrogen-deficient, more severe/larger leakage.	Cough stress test with a full bladder in lithotomy. Mild–moderate: pelvic-floor/Kegel exercise and weight loss. Surgical option: tension-free vaginal tape (TVT) synthetic mesh.
Urge/overactive bladder	Urgency immediately precedes leakage; source says most common in men and second in women. About 90% idiopathic, 10% neurologic.	Urodynamics may show uninhibited detrusor contractions, low capacity and strong flow. Fluid/caffeine/evening modification, bladder training, Kegel and weight loss; antimuscarinic then beta-3 agonist.
Overflow	Bladder overfilling with nocturia, hypotonic bladder or outlet obstruction. Causes include diabetes/neurologic disease, male BPH, large prolapse/cystocele, severe stenosis or after surgery.	Treat cause; intermittent self-catheterization or suprapubic catheter is listed.
Bypass/fistula	Continuous leakage bypassing normal outlet; uncommon.	Source states surgical treatment.
Mixed	Combination of stress and urge features.	Address both components.

Medication adverse effects

- Antimuscarinic adverse effects listed: dry mouth, constipation, blurred vision and somnolence.

OSCE-focused questions

- Ask frequency, nocturia, urgency, urge leakage, stress leakage, hesitancy, initiation difficulty, recurrent UTI, slow stream, incomplete emptying and need to reduce a prolapse during voiding.
- Clarify volume, triggers, warning, nighttime symptoms, neurological/diabetic history, obstetric injury and pelvic surgery.

33. Pelvic Organ Prolapse

Source set: 12 Pelvic prolapse.pdf

STATION SPINE: Bulge → bladder → bowel → sexual impact → risk factors → grade relative to hymen → conservative support or compartment-specific repair.

Definition, frequency and support levels

- Prolapse is descent/protrusion of pelvic organs into or beyond the vaginal opening; diagnosis is by vaginal examination. The source states that it affects about 50% of women.
- Level 1 apical support: cardinal/uterosacral ligaments → uterine prolapse; after hysterectomy → vault prolapse.
- Level 2: anterior pubocervical fascia → cystocele (most common); posterior support failure → rectocele or enterocele.
- Level 3: perineal membrane/body → deficient perineum.

Focused history: complete examiner inventory

Domain	Questions
Bulge/general	Size and consistency; always outside or moves in/out; increases through day; itching, bleeding, low-back pain, fullness/heaviness and effect of standing.
Bladder	Frequency, nocturia, urgency, urge and stress incontinence, hesitancy, initiation difficulty, recurrent UTI, slow stream, incomplete emptying and need to push/reduce prolapse during voiding.
Bowel	Constipation, obstructed defecation/splinting, incomplete emptying, fecal incontinence/soiling.
Sexual	Reduced sensation/satisfaction, embarrassment, dyspareunia and coital incontinence.
Risk factors	Menopause, high parity, forceps, large baby, heavy lifting, pelvic surgery/previous repair, hysterectomy, obesity, smoking, constipation/straining, chronic cough and family/connective-tissue weakness.

Compartment clues

- Cystocele: stress incontinence, urgency/frequency and recurrent UTI. Rectocele: obstructed defecation and need to splint the posterior vaginal wall. Deficient perineum: widened introitus and unsatisfactory sexual function.

Source grading relative to the hymen

- Grade 1: leading edge more than 1 cm above hymen. Grade 2: from 1 cm above to 1 cm below hymen. Grade 3: more than 1 cm below but less than total vaginal length minus 2 cm. Grade 4: complete eversion/procidentia.

Management

- Asymptomatic: no treatment. Conservative: pelvic-floor/Kegel exercise, topical estrogen in appropriate menopausal tissue and pessary.
- Source note describes cesarean delivery as preventive only for the first three births; retain this as a source statement.
- Surgery named by compartment: anterior colporrhaphy, posterior colpoperineorrhaphy, vaginal hysterectomy and sacrocolpopexy.

34. Uterine Fibroids

Source set: 13 Fibroid.pdf

STATION SPINE: Identify type and dominant symptom → distinguish from adenomyosis → image the uterus → observe, control bleeding/shrink, embolize or remove according to symptoms and fertility → anticipate pregnancy complications.

Definition, behavior and risk factors

- Fibroid is a benign smooth-muscle tumor of the uterus with a pseudocapsule. The source states 50–60% are asymptomatic/multiple and malignant transformation is rare (about 0.1%).
- It is estrogen-responsive and may enlarge in pregnancy.
- Risk factors: nulliparity, older age at first pregnancy, perimenopause, menarche before 10, age 25–45, African ancestry, obesity and family history.

Types, common symptom and surgical treatment

Type	Source feature/symptom	Surgical treatment
Submucosal	Beneath endometrium; intermenstrual bleeding/menorrhagia, infertility or “fibroid abortion” when pedunculated.	Hysteroscopic resection.
Intramural	Within myometrium; common symptom menorrhagia, although many are asymptomatic.	Myomectomy.
Subserosal/serosal	Outer surface; pressure or pelvic pain; may be pedunculated.	Laparoscopic myomectomy.
Other named sites	Parasitic fibroid; cervical or intraligamentary/broad-ligament fibroid; lower-segment fibroid may cause malpresentation, cesarean delivery and PPH.	Treatment individualized.

Examination and distinction from adenomyosis

- Pallor/tachycardia from iron-deficiency anemia; fundal height; localized, firm, irregular, usually nontender mass that moves with the cervix. Degeneration may make it tender.
- Source comparison: fibroid is firm, localized, asymmetrical and nontender; adenomyosis is soft/diffuse, symmetrical and tender.

Investigations

- Transvaginal ultrasound is the common first test; SIS detects small intracavitary lesions; hysteroscopy for submucosal lesions; MRI differentiates fibroid from adenomyosis.
- The source also lists biopsy/D&C;/hysteroscopy as definitive endometrial assessment when bleeding requires tissue diagnosis.

Treatment options

- Asymptomatic: observation.
- Medical/source adjuncts: tranexamic acid; GnRH analogue such as Decapeptyl for 3–6 months, with source shrinkage around 70% before surgery.
- Uterine-artery embolization preserves the uterus but not necessarily fertility and is described for a limited number of lesions.
- Myomectomy preserves fertility but about one third recur in the source; hysterectomy is definitive.

Degeneration and pregnancy complications

- Degeneration types: hyaline; red/hemorrhagic in pregnancy; cystic; fatty/calcific (“eggshell”). Red degeneration in pregnancy: bed rest and analgesia; source says no surgery during the acute episode.
- Pregnancy complications: pain/red degeneration, miscarriage/recurrent abortion, infertility, preterm labor, placenta previa, abruption, malpresentation, classical cesarean section and postpartum hemorrhage.

35. Adenomyosis

Source set: 14 Adenomyosis.pdf

STATION SPINE: Heavy regular bleeding with a symmetrically enlarged uterus → differentiate from a large/fundal fibroid using TVUS and MRI → identify risk factors/associations → treat symptoms or perform definitive hysterectomy.

Definition and case

- Adenomyosis is benign endometrial glands and stroma within the myometrium, more than 2.5 mm below the basal layer in the source notes; peak age is over 40.
- Mrs. Yusra: 43, G2P2, two surgical deliveries, heavy regular excessive bleeding for 1.5 years, obese and pale; pelvic mass 2 cm above the symphysis; symmetrically enlarged 10-week uterus with no adnexal enlargement/tenderness.

Differential and imaging

- Examiner differential: uterine adenomyosis versus a single fundal/large fibroid.
- Transvaginal ultrasound, then MRI, which the source describes as more accurate. Extended notes list biopsy/histology as definitive.

Risk factors and associations

- Mid-40s, having children/multiparity, two previous surgical deliveries; extended notes add D&C;/uterine surgery, endometriosis, fibroids and excess estrogen exposure.
- Associated gynecological problems: infertility and endometriosis.

Symptoms and signs

- Heavy menstrual bleeding with anemia; prolonged menstrual cramps/dysmenorrhea; intermenstrual spotting; chronic pelvic pain; dyspareunia; bowel/urinary pressure symptoms; lower-abdominal tenderness.
- Typical source examination: globular, diffusely and symmetrically enlarged, soft/tender uterus, especially before/during menses.

Treatment in the extended notes

- NSAIDs, combined oral contraceptive or progestogen/LNG-IUD; GnRH therapy for selected symptoms. Hysterectomy is definitive.

36. Endometriosis

Source set: 15- Endometritis and endometrial cancer .pdf

STATION SPINE: Cyclical pain/dyspareunia/dyschezia ± infertility → focused pelvic examination → ultrasound and laparoscopy/histology → individualize expectant, medical, fertility or surgical treatment.

Definition, epidemiology and sites

- Endometriosis is benign estrogen-responsive endometrial tissue outside the uterus, often in women in their 30s, nulliparous or infertile; the source says about one third have chronic pain.
- Common sites: ovary (most common; endometrioma/chocolate cyst), broad ligament/peritoneum, cul-de-sac/pouch of Douglas, uterosacral ligaments, posterior cervix and rectosigmoid.
- Ovarian endometrioma note: >4 cm → surgery; <4 cm → OCP/symptom management in the source.

Theories and risk factors

- Retrograde menstruation is most common; Müllerian/coelomic metaplasia; lymphatic spread (Halban); genetic and immunologic theories.
- Family history, white ethnicity, autoimmune disease, high social status/delayed marriage, congenital genital anomalies and short menstrual cycles are listed.

Clinical picture and history

- Dysmenorrhea, deep dyspareunia and dyschezia; chronic/cyclical pelvic pain that begins before menses, peaks during or late in menses and gradually resolves; premenstrual/postmenstrual spotting.
- Infertility/subfertility; ask cycle timing/amount, pain relation, contraception, pregnancy history, bowel/urinary symptoms and past/family/social history.

Examination, imaging and diagnosis

- Abdominal and bimanual examination: tender fixed adnexal mass, retroverted/fixed uterus, uterosacral or rectovaginal nodularity and tender nodules in the pouch of Douglas; ovarian scarring/adhesions and “kissing ovaries” may be seen.
- Ultrasound: complex adnexal mass/ground-glass endometrioma. CA125 may be raised and is listed for recurrence monitoring.
- Laparoscopy is the diagnostic gold standard in the source, with histology. Histology may show any two of glandular epithelium, endometrial glands, stroma and hemosiderin-laden macrophages; a negative biopsy does not fully exclude disease in the notes.

Differential diagnosis

- PID/chronic PID/recurrent salpingitis, ovarian cyst/tumor, uterine myoma, hemorrhagic corpus luteum, torsion of ovarian cyst and red degeneration of fibroid.

Infertility mechanisms and source frequencies

- Source notes: 40–60% of affected women are infertile and about 15% of infertile women have endometriosis.
- Mechanisms listed: adhesions/tubal distortion, dyspareunia reducing intercourse, progesterone effects on tube/corpus luteum, macrophage activity and hyperprolactinemia.

Treatment and planning factors

- Choose according to symptoms, age, disease extent, fertility wishes and threat to GI/urinary tract.
- Expectant/analgesic: observation in limited disease and NSAIDs.
- Medical aim is amenorrhea: low-dose combined OCP, progestins, danazol, GnRH agonist for 3–6 months and aromatase inhibitor in the extended notes.
- Surgery: laparoscopic excision/ablation and adhesiolysis; definitive total abdominal hysterectomy with bilateral salpingo-oophorectomy, especially after 40/completed family.
- Fertility: IUI or IVF depending disease and tubal/ovarian status.

37. Endometrial Polyps, Hyperplasia and Endometrial Cancer

Source set: 15- Endometritis and endometrial cancer .pdf

STATION SPINE: Abnormal/postmenopausal bleeding → identify unopposed-estrogen risks → speculum/bimanual and TVUS → obtain endometrial tissue → define stage/pathology → surgery ± adjuvant treatment → structured follow-up.

Cancer overview and risk profile

- The source describes endometrial cancer as the most common gynecologic cancer worldwide. Type I is linked to unopposed estrogen.
- Risk factors: obesity, hypertension, diabetes, endometrial hyperplasia (gland:stroma >50 in the notes), early menarche, late menopause/continued menstruation, nulliparity, PCOS, prolonged estrogen replacement, tamoxifen (2–3-fold source risk) and Lynch syndrome.
- Source protective factors: smoking, oral contraception and parity.

Types and prognosis

Type	Source description
Type I – endometrioid	More common, average age about 63, estrogen-related, better prognosis; source 5-year survival about 85%.
Type II – serous/clear cell	Older average age about 67, poorer prognosis; papillary serous is called the “worst” in the notes; clear cell may resemble secretory/gestational endometrium.
Recurrence	About 75% within 2 years; vaginal vault is a common recurrence site.

Bleeding presentations and differential

- Postmenopausal bleeding is the most common source symptom. Ask timing of menopause, amount/spotting, postcoital or intermenstrual bleeding, HRT/tamoxifen, Pap history, breast cancer/family history, bowel/urinary symptoms and medical/obstetric history.
- Causes of postmenopausal bleeding: endometrial/vaginal atrophy, exogenous estrogen, endometrial cancer, endometrial/cervical polyps and hyperplasia.
- Intermenstrual bleeding differential: hormonal breakthrough, pregnancy, cervical or endometrial polyp, malignancy, perimenopausal DUB, bleeding disorder/anticoagulant, atrophic vaginitis and infection.

Examination and diagnostic sequence

- General condition/BMI; abdomen for pelviabdominal mass; speculum; bimanual/vaginal examination.
- TVUS for endometrial thickness; source teaching note uses >5 mm as a biopsy/D&C; trigger in postmenopausal bleeding and another page mentions a 9-mm screening value. Preserve these as source-specific values.
- Endometrial sampling/Pipelle, hysteroscopy and D&C; CBC, kidney/liver function, CA125 and other preoperative testing when listed.
- Pap smear is not a substitute for endometrial sampling.

Source conflict retained: the slides contain both >5-mm diagnostic and >9-mm screening-style thickness statements.

Endometrial polyp station

- A 3-cm endometrial polyp with intermenstrual bleeding: hysteroscopy, D&C; and polypectomy; aims are symptom relief and histopathology.

Pathology/staging information after unexpected malignancy

- Clarify whether preoperative D&C; was performed; whether ovaries/tubes were removed; peritoneal cytology; hysterectomy type/route; pelvic/paraaortic lymph nodes.
- Pathology data: tumor type, grade/differentiation, size and location, depth of myometrial invasion, cervical involvement, CA125, lymph-node status and stage.

Spread, staging and treatment

- Spread routes: direct extension (most common), transtubal, lymphatic and hematogenous (worst in the source). Endometrial cancer is surgically staged.
- Stage IA: total hysterectomy and bilateral salpingo-oophorectomy, no adjuvant radiotherapy in the source. Stage IB/type II with negative nodes: hysterectomy/BSO plus radiotherapy.
- Stage II: modified radical or radical type III hysterectomy with BSO and pelvic/paraaortic lymph-node assessment, with radiotherapy as listed. Advanced disease may receive radiotherapy or chemotherapy.

Prognostic factors and follow-up

- Stage, histologic type, grade/differentiation, depth of myometrial invasion, nodes, tumor size >4 cm, hormone-receptor status, age and nondiploid tumor biology.
- Follow-up: history and physical examination; vault smear every 3 months for 2 years, every 6 months for the next 3 years, then annually. CA125/HE4, CBC, chest X-ray or CT are listed when clinically indicated or previously elevated.

38. Cervical Cancer

Source set: 16 cervical cancer.pdf

STATION SPINE: Prevent and screen → recognize postcoital/abnormal bleeding → inspect and biopsy the cervix → stage clinically with rectovaginal/cystoscopic assessment → treat stage IIA with radical surgery and nodal assessment in the source station.

Overview, histology and development

- Mean age in the source is about 52, with peaks at 35–39 and 60–64. Squamous-cell cancer is most common (about 90%) and arises from the transformation zone; adenocarcinoma is associated with HPV and is increasing; small-cell disease is described as the worst/rare; large-cell has better prognosis than small-cell.
- Source sequence: oncogenic HPV infection of metaplastic transformation-zone epithelium → viral persistence → clonal progression to carcinoma in situ → invasion through basement membrane.
- HPV 16 and 18 cause most cervical cancers; vaccination is emphasized.

Risk factors

- Young age at first coitus/early marriage (station gives 16 years), multiple sexual partners, promiscuous male partner, STI history, smoking, prolonged oral-contraceptive use, immunodeficiency, high parity and lower socioeconomic status.
- Source counseling also warns never to rely on a previous normal Pap smear when symptoms or an abnormal cervix are present.

Prevention and screening listed

- Primary prevention: HPV vaccination and safer sexual practices.
- Secondary prevention: cervical screening/Pap smear, speculum/vaginal examination and prompt assessment of postcoital bleeding. One station says annual Pap smear and annual pelvic ultrasound; retain as checklist wording.
- Pap smear is a screening test, not a diagnostic test. Colposcopy guides biopsy but is not itself the final diagnostic tool. Biopsy is the source “only diagnostic tool.”

Symptoms and examination

- Postcoital bleeding is the classic first symptom; also abnormal uterine/intermenstrual/postmenopausal bleeding, abnormal vaginal discharge, dysuria and loin pain.
- Late features: obstructive uropathy, edema/peritoneal or metastatic signs, weight loss and reduced appetite.
- Speculum may show an ulcerative, friable, highly vascular cervix that bleeds on touch. Bimanual and rectovaginal examination assess primary size, parametria and uterosacral extension; local invasion may involve bladder or rectum.

Investigations and clinical staging

- Speculum and colposcopy with directed cervical biopsy; fractional D&C; routine blood tests.
- Cystoscopy, urine cytology and proctosigmoidoscopy assess adjacent-organ involvement; chest X-ray and CT/MRI/PET assess spread and surgical suitability. CA125 is listed but noted to be nonspecific.
- The source describes cervical cancer as clinically staged: parametrial involvement on rectovaginal exam → stage IIB; pelvic mass/sidewall involvement on bimanual exam → stage IIIB; bladder involvement on cystoscopy → stage IVA; positive peritoneal cytology/distant disease → stage IVB.

Stage IIA station and management

- Mrs. Sandra: 26, G2P2, married twice, long postcoital bleeding, biopsy-proven well-differentiated squamous-cell carcinoma stage IIA, normal radiology.
- Diagnostic/staging steps accepted: speculum/colposcopy, cervical biopsies, fractional D&C;, cystoscopy/urine cytology and rectovaginal examination.
- Ideal surgical step in the station: radical hysterectomy, pelvic lymphadenectomy and bilateral ovarian translocation.

Smoking effects requested by the examiner

- Smoking damages cervical DNA; smoking products block or reduce antigen-presenting Langerhans cells and local immunity; smoking is stated to encourage harmful sexual habits among tobacco users.

Surgical complications in the teaching map

- Urinary dysfunction is shown as a common complication of radical hysterectomy; rectovaginal fistula is listed as a serious complication.

39. General Genital Cancer and Vulvar Cancer

Source set: 17 Genital cancer.pdf

STATION SPINE: Do not rely on a prior smear → take complete history and examination → image and inspect cervix/vulva → biopsy abnormal tissue → stage with endometrial, bladder and rectovaginal assessment where indicated.

Gynecologic cancer overview

- The source names five main gynecologic cancers: cervical, ovarian, uterine, vaginal and vulvar; fallopian-tube cancer is a rare sixth type.
- Only cervical cancer has an established screening test in the uploaded material.

General genital-cancer station

- 35-year-old nulligravida, married twice over 20 years, two failed IVF trials and abnormal vaginal bleeding.
- Risk factors listed: early age of marriage, multiple sexual partners, nulliparity, smoking and relying on a previous Pap-smear result.
- Next clinic steps: proper history, proper examination, ultrasound, speculum examination and colposcopy.
- Diagnostic plan: fractional D&C;, hysteroscopy, cystoscopy with urine cytology, biopsy of abnormal cervical areas and manual rectovaginal examination under general anesthesia.

Vulvar cancer: definition and presentation

- Cancer of the external female genitalia, including labia majora, labia minora and clitoris. It commonly begins as a lump or persistent sore; most vaginal cancers begin without obvious signs in the source handout.
- Symptoms: persistent itching/burning, pain/tenderness, bleeding not related to menstruation, color/skin changes, and a persistent lump, wart-like lesion or open sore.

Vulvar cancer risk factors

- Increasing age, smoking, immunodeficiency, HPV exposure, cervical cancer, inflammatory vulvar disease, vulvar intraepithelial neoplasia (VIN), genital warts or other chronic vulvar skin disease, family history of melanoma, multiple sexual partners, young age at first intercourse and abnormal Pap history.

Types, diagnosis and treatment listed

- Most common type: vulvar squamous-cell carcinoma; two pathways are shown—HPV-related in younger women and chronic inflammation-related in older women.
- Other named types: melanoma, extramammary Paget disease, Bartholin-gland adenocarcinoma, verrucous carcinoma, basal-cell carcinoma and sarcoma.
- Diagnosis: history/physical and pelvic examination, Pap test, HPV test, biopsy, colposcopy, MRI, CT and PET when indicated.
- Treatment: primary surgery for squamous cancer, with radiation and/or chemotherapy depending on stage/field; melanoma treated with wide local excision; Paget disease may require repeated excision and other oncologic therapy.

Prevention messages

- Reduce sexually transmitted infection risk, use condoms, receive HPV vaccine and perform regular self-examination in the source handout.

G2. Benign and Malignant Lesions of the Vulva and Vagina - Three Constructed OSCE Stations

Source set: Benign and Malignant Lesions of the Vulva and Vagina(2).pdf

CONSTRUCTED-STATION SPINE: Look at morphology -> locate precisely -> match age/pathway -> biopsy when required -> stage invasion/nodes -> choose site/stage-based management.

STATUS: These are original OSCE stations constructed from the attached lecture-derived study guide because no official checklist was supplied. The medical facts and lecture-specific thresholds are source-grounded; the patient scenarios and mark allocation are newly constructed for revision.

Station 1: Benign and inflammatory vulvar lesions

STUDENTS INFORMATION
A 48-year-old woman presents with chronic vulvar itching. During the station, the examiner describes several lesions: a thick leathery plaque, thin white scarred skin, a red erosion with white striae, a cyst at 8 o'clock and a paraurethral cyst. Differentiate and manage them.

Examiner questions

- Compare the three lichenoid disorders.
- Identify common cysts by site.
- Discuss epidermal cysts and genital warts.
- State when biopsy/treatment is required.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Lichen simplex	Itch-driven epithelial hyperplasia; pain/itch, white or dark-red plaques with thick leathery raised surface; intermediate-potency topical corticosteroid.	**2**
Lichen sclerosus	Midlife anogenital disease; intense pruritus/dyspareunia/burning/fissures; pearly white thin inelastic tissue-paper skin, loss of labia minora, buried clitoris, vestibular/perianal scarring.	**2**
Lichen sclerosus diagnosis/treatment	Biopsy: loss of rete ridges, atrophic epithelium and inflammation at basement membrane; high-potency topical steroid.	**2**
Lichen planus	Autoimmune; burning/irritation/dyspareunia; red erosions with white striae; may affect vulva, vagina and mouth; topical/systemic steroids.	**2**
Epidermal cyst	Non-tender, mobile, slow-growing, obstructed hair follicle containing sebum/epithelium; may observe or express/deflate.	**2**
Bartholin cyst	Below hymenal ring at 4 or 8 o'clock; discomfort; marsupialization or incision/drainage if symptomatic/changed.	**2**
Skene cyst	Paraurethral near distal urethra; dyspareunia or urinary obstruction; same procedural options.	**2**
Genital warts	HPV 6/11; posterior fourchette/lateral vulval walls; papillary/exophytic; lecture lists excision and "antiviral therapy."	**2**
Other listed differentials	Hidradenitis/apocrine inflammation, urethral caruncle, varicosities/angiomas/hematomas; psoriasis, Behcet, Crohn, acanthosis nigricans and eczema are named differentials only.	**2**
Pattern method	Describe morphology + exact location + age/pathway; biopsy suspicious/changing or histology-dependent lesions, while not inventing details absent from source.	**2**

What you must memorize from this station

- Thick/leathery = lichen simplex; thin white scarred tissue-paper = lichen sclerosus; red erosions/white striae with vaginal/oral involvement = lichen planus.
- Bartholin 4/8 o'clock; Skene paraurethral.
- HPV 6/11 = genital warts.
- Lichen sclerosus connects to the older-woman vulvar-cancer pathway.

EXAM TRAPS: Lichen sclerosus does not affect the vagina in the lecture; lichen planus can and may cause stenosis. A warty lesion is not always a benign wart.

Station 2: VIN, vulvar cancer and melanoma

STUDENTS INFORMATION
An older postmenopausal woman with years of lichen sclerosus presents with a raised ulcerated labial lesion. The examiner also asks you to distinguish Bowen disease, Paget disease and vulvar melanoma.

Examiner questions

- Explain VIN/in-situ lesions and the two SCC pathways.
- Stage vulvar cancer using the lecture scheme.
- Give stage/site-based management and prognosis.
- Recognize melanoma.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Bowen disease	Squamous carcinoma in situ/VIN 3; mean age 45, 50% asymptomatic, itching most common, elevated multicolored lesion, 20% warty; superficial excision with 5-mm margin or skinning vulvectomy if extensive.	**2**
Paget disease	Adenocarcinoma in situ; elderly white postmenopausal; itchy/tender well-demarcated eczematous white plaque; 20% associated adenocarcinoma; large pale Paget cells in epidermis/adnexal structures.	**2**
SCC pathways	More common older/long-standing lichen sclerosus pathway; less common younger HPV-associated pathway with concurrent VIN.	**2**
SCC presentation/site	90% of vulvar cancers; lump/ulcer and long-standing pruritus; raised, ulcerated, pigmented or warty; usually labia majora/minora, less often clitoris/perineum; 5% multifocal.	**2**
Stages I-II	I <=2 cm node-negative; IA invasion <1 mm, IB >1 mm. II >2 cm node-negative.	**2**
Stages III-IV	III any size with adjacent urethra/vagina/anus and/or unilateral regional nodes; IVA upper urethra/bladder/rectal mucosa/pelvic bone or bilateral nodes; IVB distant including pelvic nodes in lecture scheme.	**2**
Early management	IA radical local excision with 1-cm margin, no groin dissection. IB/II lateral lesion: local excision + ipsilateral inguinofemoral nodes; midline: bilateral groin dissection.	**2**
Advanced management	Stage III radical vulvectomy + bilateral groin dissection; bulky/advanced: preoperative radiation/chemoradiation then conservative excision; postoperative radiation for source-listed nodal findings/extranodal spread.	**2**
Prognosis	Lymph-node status is strongest anchor: 5-year survival 90% with negative nodes versus 50% positive.	**2**
Melanoma	Second most common vulvar cancer; postmenopausal white woman, labia minora/clitoris, small but early metastasis; excisional biopsy, depth predicts prognosis, 5-year survival 30%; 1-cm excision if superficial, add ipsilateral nodes if depth >=1 mm.	**2**

What you must memorize from this station

- Bowen = squamous in situ, age ~45, multicolored/elevated; Paget = elderly eczematous plaque with pale Paget cells.
- SCC is 90%; chronic itch + labial ulcer/lump is classic.
- Node status drives survival: 90% negative vs 50% positive.
- Lateral lesion -> ipsilateral groin; midline -> bilateral.
- Melanoma prognosis follows dermal depth, not surface size.

EXAM TRAPS: The guide preserves the lecture’s legacy FIGO staging. Do not call every warty lesion a wart; Bowen and SCC can be warty.

Station 3: Vaginal disease, VAIN and vaginal cancer

STUDENTS INFORMATION
A postmenopausal woman has vaginal bleeding and discharge. Examination identifies an upper vaginal lesion; another patient has painful erosive vaginal inflammation with narrowing. Discuss differential diagnosis, biopsy, staging and management.

Examiner questions

- Review infectious and inflammatory vaginal disease.
- Explain VAIN.
- Describe vaginal cancer presentation and investigation.
- State FIGO staging, treatment and complications.

Examiner checklist - constructed 20-mark station

Examiner checklist domain	Full-mark content	Marks
Infectious causes	Bacterial vaginosis, Candida albicans and Trichomonas vaginalis; inflammation/discharge confirmed with microbiological swabs.	**2**
Erosive lichen planus	Autoimmune vaginal pain/inflammation; may cause stenosis if untreated; vaginal trainers plus intravaginal steroids.	**2**
Not vaginal	Lecture states lichen sclerosus and eczema do not affect the vagina.	**2**
VAIN origin/presentation	Usually extension of CIN from cervix; often asymptomatic.	**2**
VAIN treatment	Cauterization, excision, radiotherapy or expectant management according to patient, grade and size; source gives no numerical cancer risk.	**2**
Vaginal cancer presentation	Rare, cause unknown, cervical-like risk factors; early disease often asymptomatic and advanced disease causes bleeding/discharge; pain indicates spread to pelvic nerves.	**2**
Diagnosis/workup	Vaginal biopsy; investigations same as cervical cancer.	**2**
Treatment/progression	Surgery rarely possible because often advanced; radiotherapy and chemotherapy usually first-line; progression usually local.	**2**
Staging	I mucosa only; II subvaginal infiltration not pelvic wall; III pelvic wall; IVA bladder/rectal mucosa; IVB beyond pelvis.	**2**
Complications	Advanced rectovaginal and vesicovaginal fistulae.	**2**

What you must memorize from this station

- Lichen planus can narrow the vagina; treat with trainers and intravaginal steroids.
- VAIN is usually cervical CIN extension and often asymptomatic; source gives no numeric progression risk.
- Vaginal cancer stages anatomically: mucosa -> subvaginal tissue -> pelvic wall -> bladder/rectum -> beyond pelvis.
- Biopsy confirms; radiotherapy/chemotherapy are usually first line in advanced presentation.

EXAM TRAPS: Do not invent the VAIN cancer-risk percentage. Pain in vaginal cancer suggests spread beyond the vagina to pelvic nerves.

Three-station master memory sheet

- Solve vulvar lesions by morphology + exact site + age/pathway.
- Lichen patterns and cyst locations are high-yield.
- VIN/cancer requires biopsy and node-aware staging; node status dominates prognosis.
- Vaginal disease includes infection, erosive lichen planus, VAIN and cancer; staging is anatomical.

Source-specific wording retained: older VIN terminology, legacy FIGO staging, “antiviral therapy” wording and unanswered VAIN risk percentage remain clearly identified as source-derived.

40. Benign Ovarian Cysts and Tumors

Source set: 18 Ovarian cyst.pdf

STATION SPINE: Rule out malignancy and prevent rupture/torsion/hemorrhage → classify functional, epithelial, sex-cord stromal and germ-cell lesions → use bimanual examination and TVUS → observe simple lesions or operate on complex/symptomatic masses.

General features and benign-mass profile

- Most ovarian cysts are asymptomatic, discovered incidentally and may resolve without treatment. Management aims to rule out malignancy and prevent rupture, torsion and hemorrhage.
- Source benign profile: mobile, soft, smooth, unilateral, <8 cm, no septations and no Doppler flow; usually cystic rather than solid.

Functional/physiologic cysts

Lesion	Source features
Follicular cyst	Failure of follicle rupture; may persist several cycles and reach 10 cm; resolves in 2–4 months; common in reproductive age; usually painless, though notes add pain/irregular bleeding.
Corpus-luteal cyst	Corpus luteum grows to about 3 cm and fails to regress after 14 days; thin-walled fluid lesion without septation/calcification, smaller but firmer than follicular; may cause pain/peritoneal irritation.
Hemorrhagic cyst	Bleeding into a corpus-luteal cyst; rupture may cause peritonitis.
Theca-lutein cysts	High hCG with hydatidiform mole/choriocarcinoma or clomiphene ovulation induction; usually bilateral.

Benign epithelial tumors (most common neoplastic group)

- Derived from surface mesothelium; source emphasizes age >40.
- Serous cystadenoma: most common, resembles fallopian-tube lining, bilateral in about 10%, 70% benign; small, unilocular; may form psammoma bodies.
- Mucinous cystadenoma: resembles endocervical epithelium, bilateral <10%, about 85% benign; very large, multilocular, thick mucinous fluid; source notes pseudomyxoma peritonei.
- Endometrioid cystadenoma: difficult to distinguish from endometriosis; pelvic pain/deep dyspareunia from adhesions.

Sex-cord stromal tumors

- May occur at any age, more commonly postmenopausal, secrete hormones and cause bleeding.
- Granulosa-cell tumor: locally malignant but slow with good prognosis; solid, recurrence common; secretes estrogen/inhibin, may cause precocious puberty and predispose to endometrial cancer.
- Theca-cell tumor: benign, solid, unilateral, usually postmenopausal; secretes estrogen and causes bleeding.
- Fibroma: rare, elderly, hard/mobile; ascites plus pleural effusion = Meigs syndrome.
- Sertoli–Leydig tumor: low-grade malignant, around age 30, very rare/small/unilateral; produces androgens and virilization.

Germ-cell tumors

- Most common ovarian neoplasm in reproductive age; source says 2–3% malignant; derived from all three germ layers.
- Benign cystic teratoma/dermoid: ectodermal tissue (sweat/sebaceous glands, hair, teeth) plus mesodermal tissue; about 60% asymptomatic; most common ovarian tumor overall in the notes and prone to torsion/peritonitis.
- Monodermal teratoma: struma ovarii contains mature thyroid tissue. Mature solid teratoma is rare; distinguish from malignant immature teratoma.

Diagnosis and management

- Bimanual examination and pelvic/transvaginal ultrasound.
- Reproductive age: asymptomatic/simple <5 cm → conservative, recheck after next menses and repeat US/CA125 at 3 months. Complex/septated/solid, simple >7 cm or symptomatic → laparoscopy with cystectomy versus oophorectomy while preserving fertility.
- Postmenopausal: simple <1 cm → no action; simple 1–5 cm with no malignant features and normal CA125/asymptomatic → repeat US and CA125 every 4 months for 1 year. Solid/complex/fixed >5 cm, symptomatic or painful → laparoscopy and bilateral salpingo-oophorectomy ± hysterectomy.

Source-specific size rules retained: the slides use <5 cm and >7 cm in reproductive age and 1–5 cm/>5 cm in postmenopause; no external harmonization has been applied.

41. Ovarian Cancer and Adnexal Mass in Pregnancy

Source set: 19 Ovarian cancer (worset cancer).pdf

STATION SPINE: Characterize the adnexal mass → assess malignancy with ultrasound, markers and staging imaging → perform surgical staging/cytoreduction → preserve fertility only in the source-selected young case → structured surveillance.

Overview, age and risk/protective factors

- The source calls ovarian cancer the “worst” gynecologic cancer because early disease is often asymptomatic, diagnosis is commonly advanced (stage III), and it is the leading gynecologic-cancer cause of death. Mean age is 50–60 years.
- Risk factors: early menarche, late menopause, late first pregnancy, BRCA1/BRCA2, family history, previous ovarian cancer, nulliparity, infertility and estrogen-replacement therapy.
- Protective factors reduce lifetime ovulation: OCPs, pregnancy/parity, tubal ligation and the source also lists PCOS.

Symptoms and examination

- Nonspecific early symptoms: vague abdominal pain/bloating and GI complaints; pressure symptoms such as urinary frequency/urgency, constipation or dyspareunia; menstrual irregularity; late abdominal swelling from ascites.
- Vaginal/rectal examination may reveal a solid, irregular, fixed pelvic mass; upper-abdominal mass, combined ascites or pleural effusion may occur.

Tumor groups and markers

Origin	Source types/markers
Epithelial (~80%, postmenopausal)	Serous (most common, source 75%; resembles tubal/glandular epithelium), endometrioid, mucinous and clear-cell/Brenner-related descriptions. High-grade serous disease may arise from fimbrial tube. CA125.
Germ cell (~15%, young)	Dysgerminoma (ovarian counterpart of seminoma, radiosensitive, good prognosis) → LDH; yolk-sac/endodermal-sinus tumor → AFP; immature teratoma; embryonal carcinoma; choriocarcinoma.
Stromal (~5%)	Granulosa cell → estrogen/inhibin; Sertoli–Leydig → androgen.
Metastatic	Krukenberg tumor: bilateral uniform enlargement from metastatic signet-ring carcinoma.
Other marker notes	CEA and CA19-9 for suspected GI/mucinous disease; CA15-3 is listed for breast metastasis context; HE4 and risk-of-malignancy index appear in the expanded work-up.

Ultrasound characterization and benign/malignant clues

- Size; unilateral/bilateral; solid/cystic; wall/septal thickness; papillary projections/excrescences; vascular flow; ascites; and upper-abdominal findings.
- Pregnancy station: 5-cm left solid mass, no septation/papillary projection/ascites, negative vascular flow and normal upper abdomen → source diagnosis benign cystic teratoma.

Differential diagnosis of adnexal/pelvic mass

- Ovarian non-neoplastic: functional/simple corpus-luteum cyst, luteoma of pregnancy, endometrioma and hyperreactio luteinalis.
- Benign neoplastic: dermoid, epithelial and paraovarian. Malignant: epithelial, dysgerminoma and sex-cord stromal; expanded notes add Krukenberg.
- Extraovarian: pedunculated fibroid, pelvic kidney, uterine anomaly/retroverted uterus and colorectal carcinoma. General pelvic-mass list also includes full bladder, gravid uterus, ectopic pregnancy, appendicitis and PID.

Malignancy work-up

- Transvaginal ultrasound with or without Doppler; CT abdomen/pelvis; CA125; CBC, KFT, LFT and crossmatch; chest X-ray.
- Expanded source adds HE4, CEA/CA19-9 as appropriate, bone scan for bone metastasis and risk-of-malignancy index.
- Source note: diagnosis/staging requires exploratory laparotomy; it states there is no role for diagnostic/treatment laparoscopy because rupture may disseminate tumor.

Pathology and prognostic factors

- Stage is the strongest prognostic factor; histologic type (clear cell is noted as worse), grade and cytogenetic/ploidy features matter. Diploid tumors are described as better than aneuploid tumors.
- Clinical factors: patient age and volume of ascites.

Treatment principles

- Initial surgical exploration of abdomen/pelvis. Early stage: TAH/BSO in the general map; grade 1–2 tumor confined to one/both ovaries may require no further treatment after complete staging in the source.
- Poorly differentiated/grade 3 disease receives systemic chemotherapy. Advanced disease: cytoreductive/debulking surgery followed by chemotherapy.

Fertility-preserving suspicious-mass station

- Young woman seeking pregnancy with multilocular right ovarian mass and finger-like projections.
- Intraoperative steps: palpate abdomen/pelvis; peritoneal washings for cytology; biopsy peritoneal surfaces and any visible nodule; ipsilateral salpingo-oophorectomy without hysterectomy if appropriate; ipsilateral pelvic lymphadenectomy; omental biopsy; contralateral ovarian biopsy/frozen section.
- Follow-up/fertility plan: encourage pregnancy soon, use fertility drugs to ensure ovulation, annual TVUS and annual CA125; after family completion, hysterectomy with contralateral salpingo-oophorectomy.

General follow-up

- Every 3 months for 1 year, then every 6 months yearly in the map; history, physical examination and CA125. If recurrence is suspected clinically or by CA125, obtain imaging.

Source boundary: the fertility-preserving plan and the statement excluding laparoscopy are reproduced from the uploaded notes and are not replaced with outside guideline criteria.